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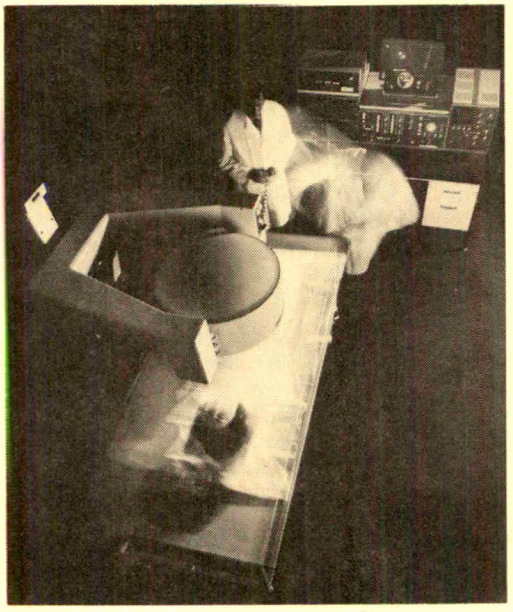
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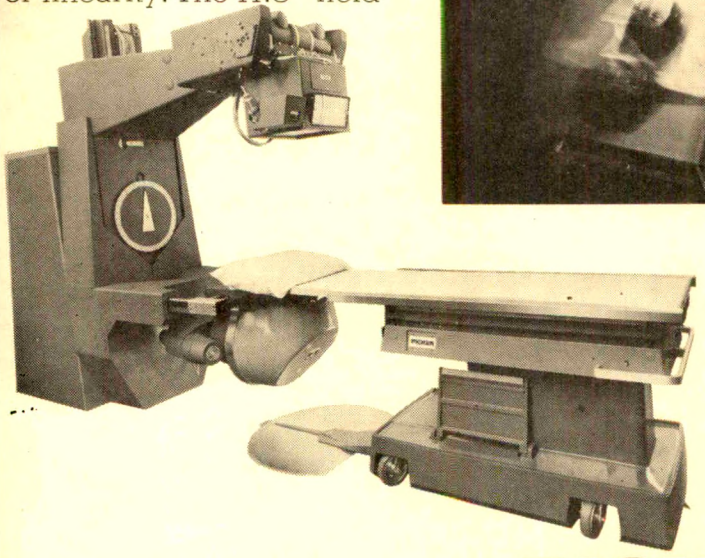


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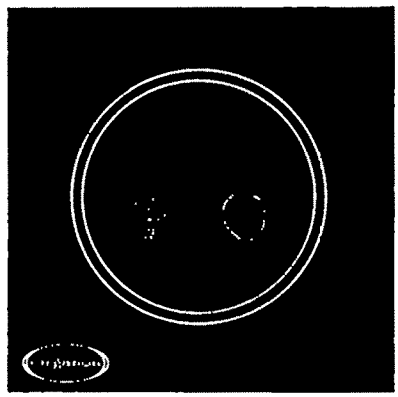
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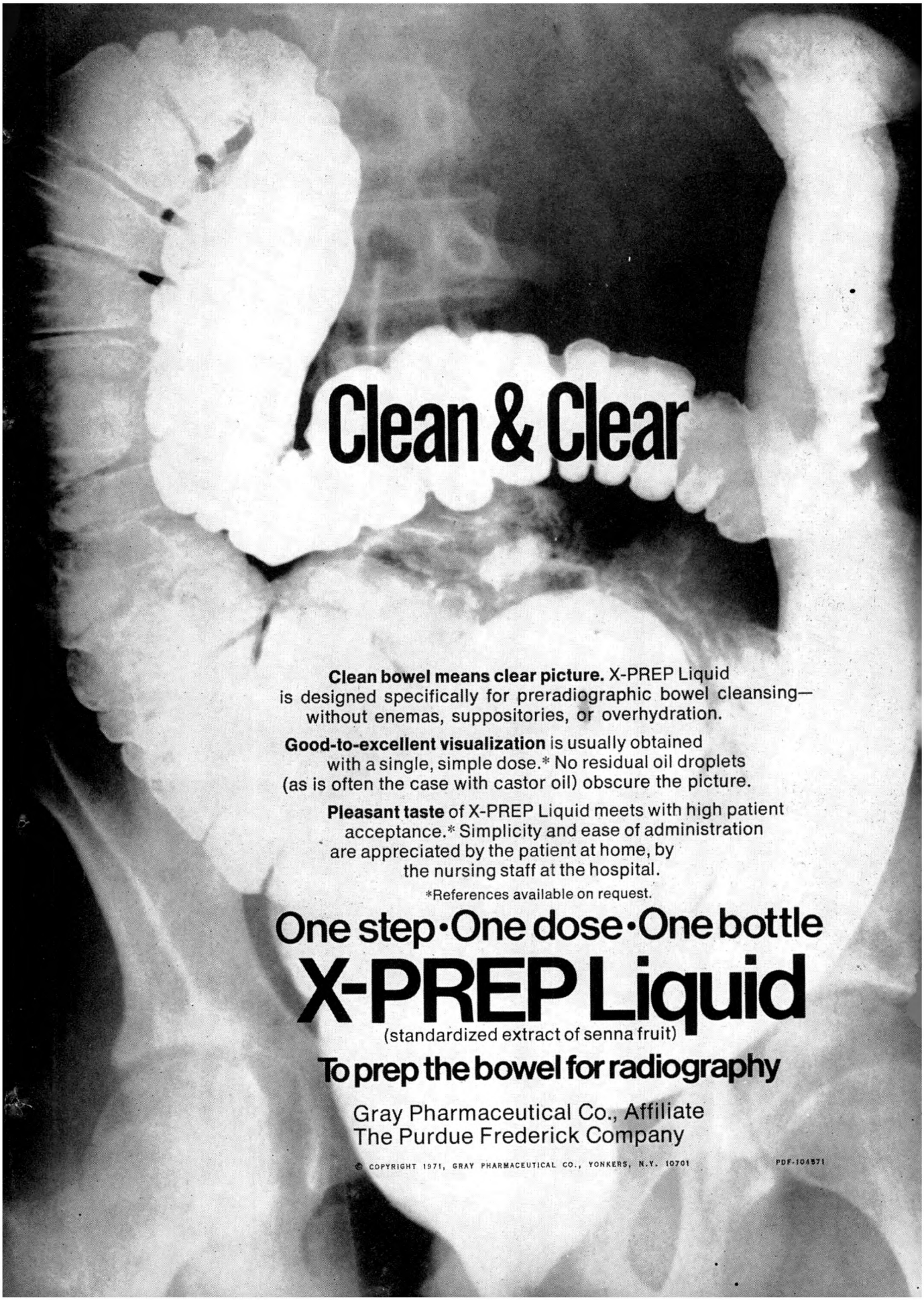
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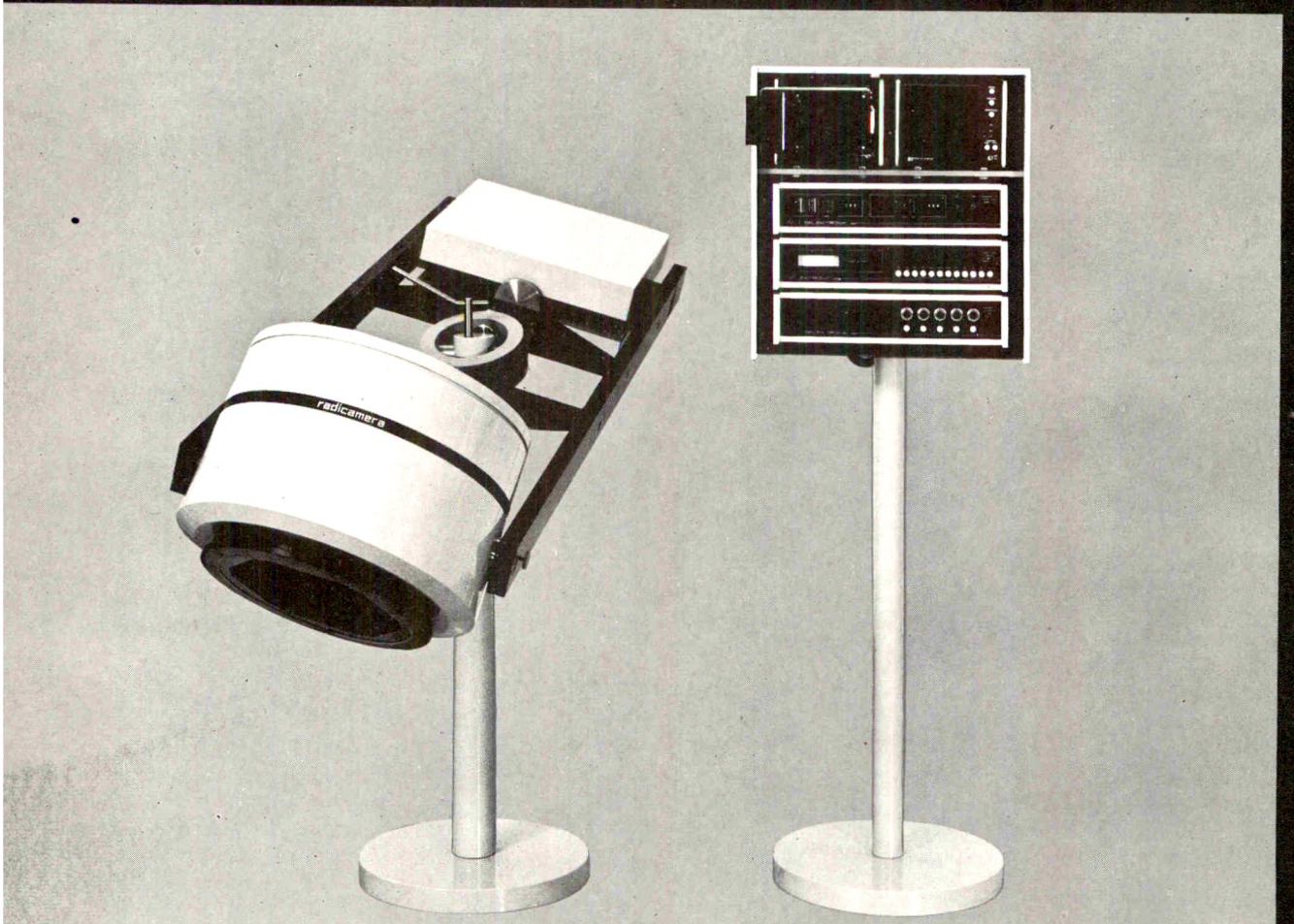
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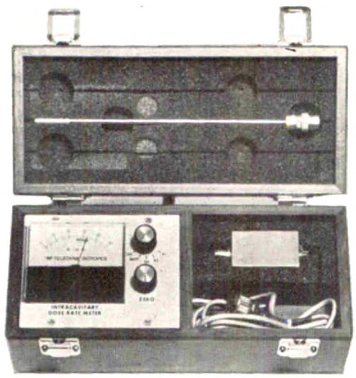
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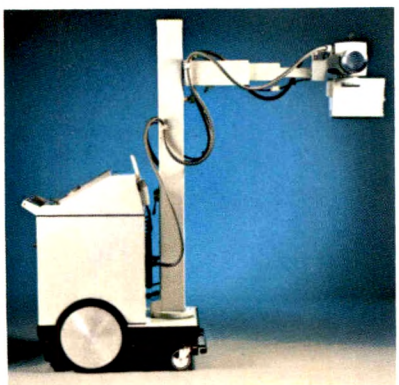
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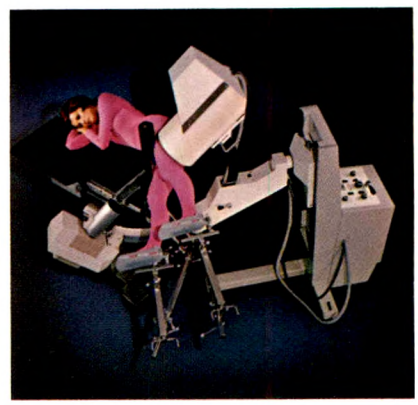
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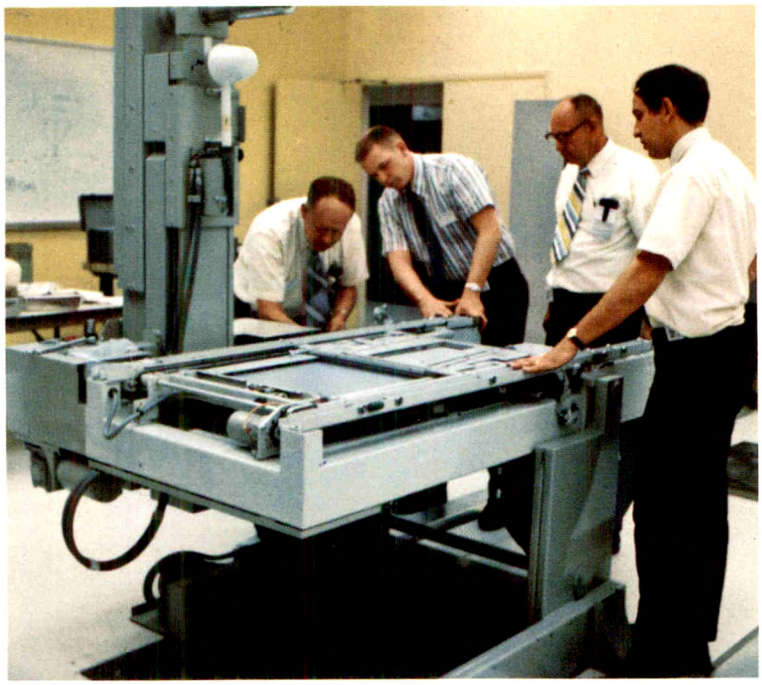
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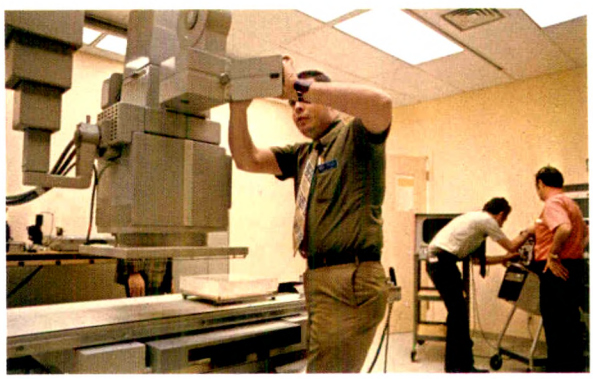
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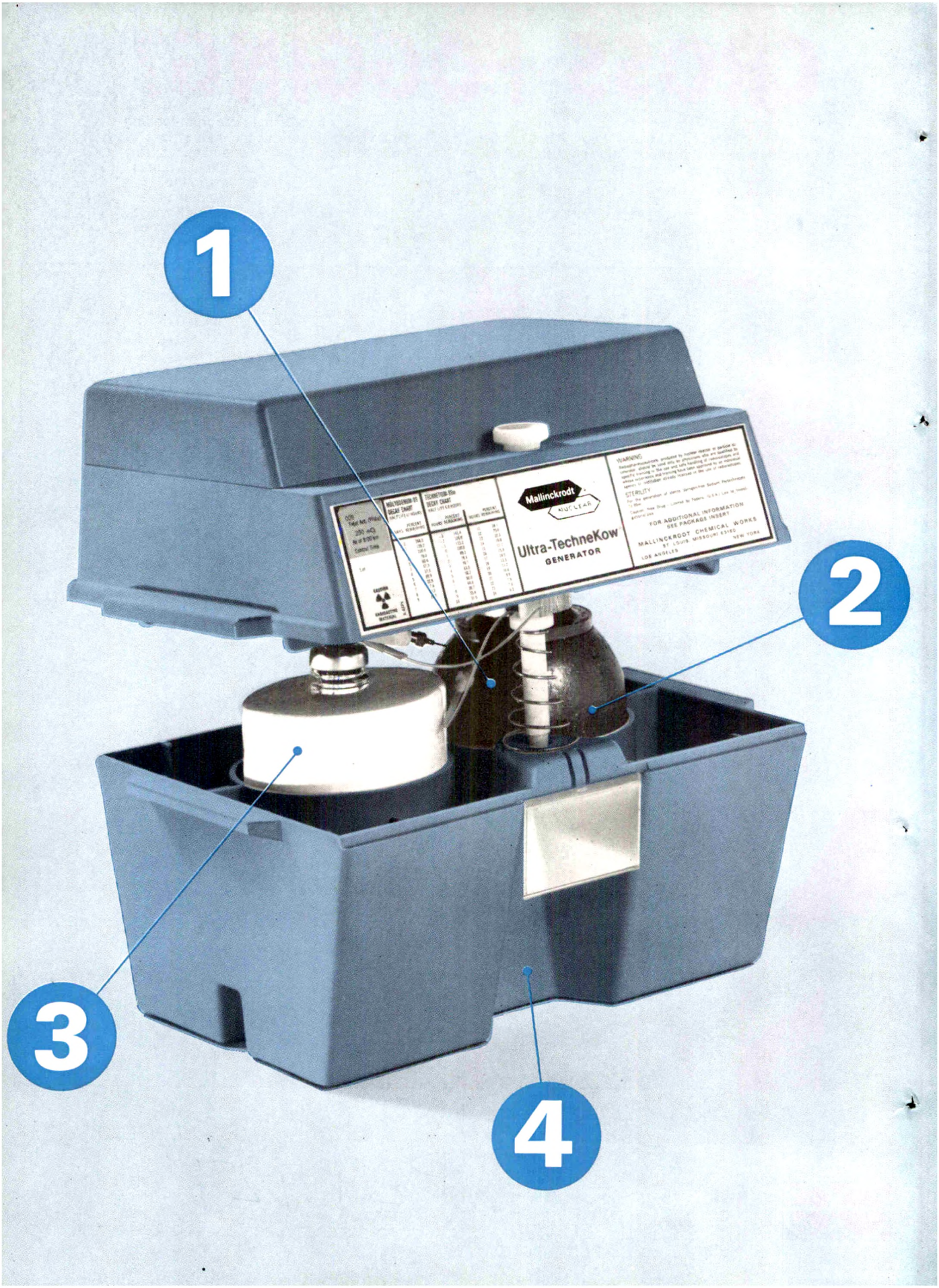
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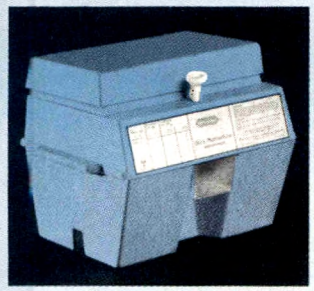
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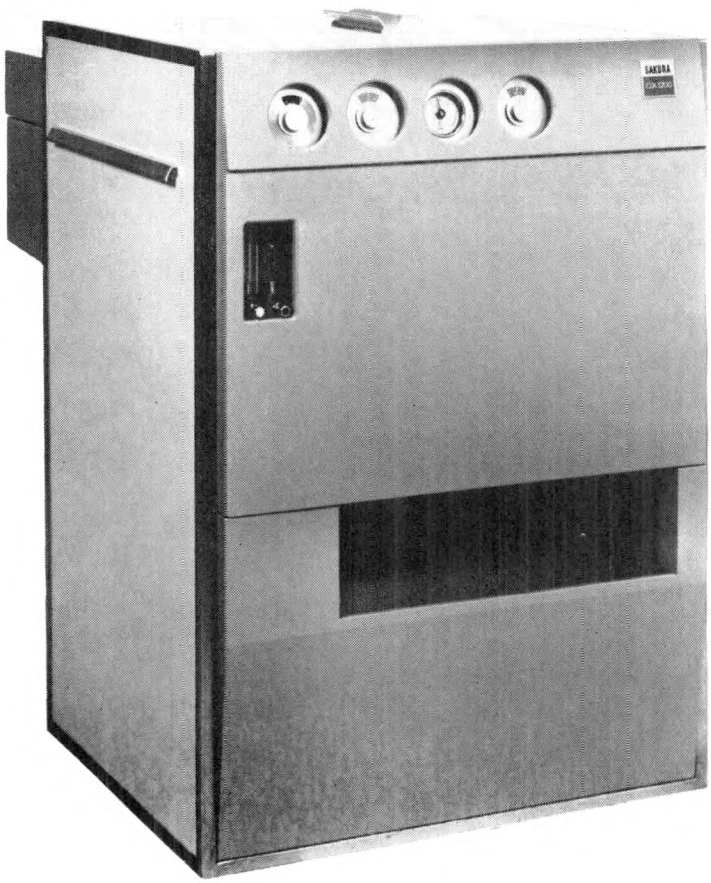
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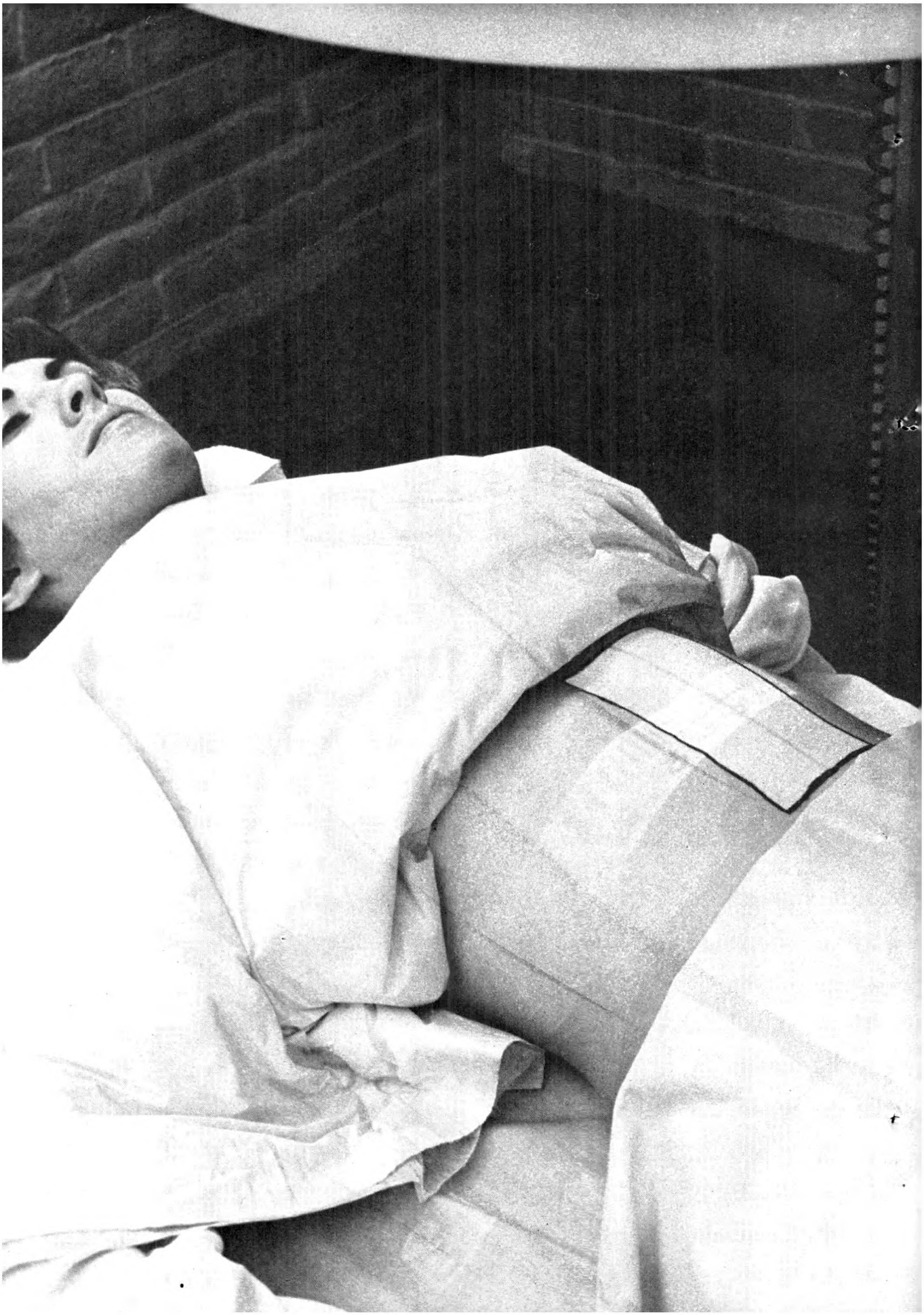
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The SHM plotter uses solid-state computer circuitry, and an ultra-sensitive semiconductor diode detector that moves smoothly through the water phantom in precise ¼ mm steps. Each point is automatically plotted with positional accuracy better than ± ½ mm. A precision-machined drive assembly assures continuous, uninterrupted operation.

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If you have a computer, good! You'll save even more time. And your computer will use real data—not assumed information—because the SHM plotter interfaces directly with any medium-sized computer.

In addition, the SHM plotter makes it easy to experiment with field sizes and shapes, to further improve treatment precision. This also makes it an effective teaching tool.

On top of all this, the SHM plotter is compact enough for you to pack in your car.

Our Radiation Field Scanner is another way we've improved radiotherapy reliability by improving treatment accuracy. It measures the geometry of fields up to 44 x 44 cm in one, fast operation. You get immediate readout on a meter or an optional X-Y plotter: for Co60, Betatron and linear accelerator units. Accuracy is ±1 mm.

Instruments like these are just part of the reason why SHM Nuclear is the fastest growing company in radiotherapy.

For example, we make the Therapi 4, a linear accelerator so reliable it's becoming standard equipment in independent clinics across the country.

We're also providing complete service, and replacement parts that out-perform the originals, for radiotherapy departments which use other people's equipment.

What's more, in our four years as a company, we've successfully introduced more new products and services to improve radiotherapy reliability than any other manufacturer. And more are on the way.

Find out how SHM can help you improve the reliability of your radiotherapy operation. Call or write: SHM Nuclear Corporation, 568 San Xavier, Sunnyvale, CA 94086. Phone: (408) 245-3136.



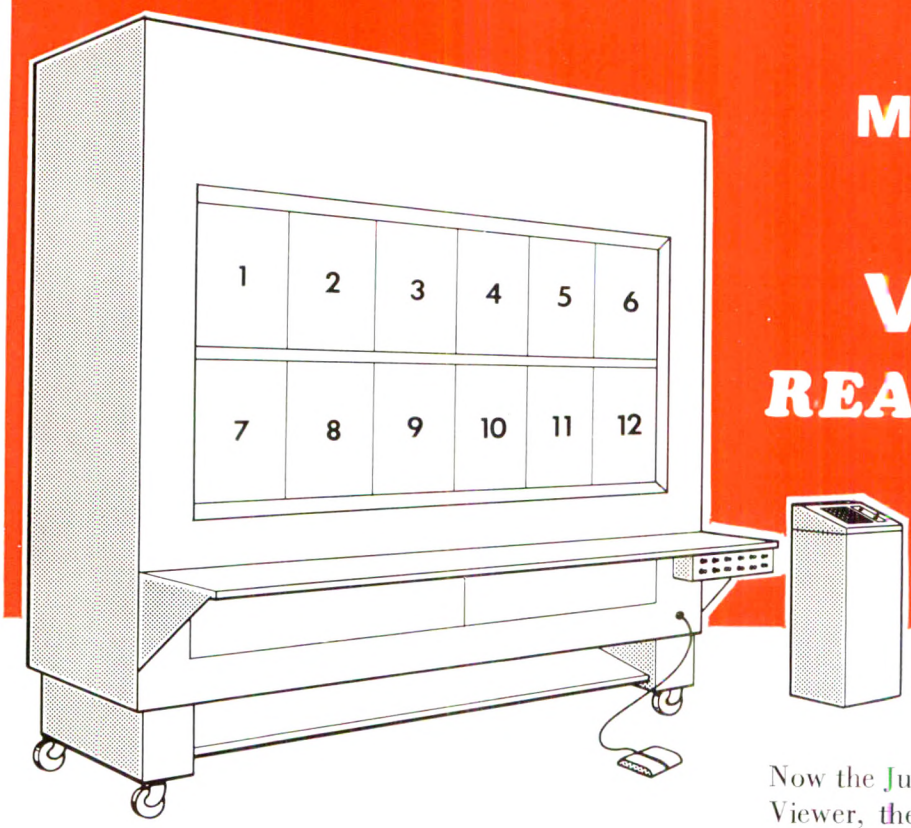
SHM Nuclear Corporation

Radiotherapy reliability—we're doing something about it, from facilities planning to the delivered dose.

Announcing the MV300A

300 FILM MOTORIZED X-RAY VIEWER

READ 12 FILMS Six Over Six



Only 99½" Wide, 93 9/16" High, 28 9/16" Deep, 15" Wide Formica shelf.
300 Film capacity, 50–6 bank panels.

Built and designed same as the MV200A 200 film S & S Motorized Viewer with new modular construction for easy delivery in sections and quick installation.

Now the Jumbo 6 over 6 300 Film Motorized Viewer, the only viewer made to read 12 — 14 x 17" films or 84" wide panels of smaller film sizes. Individual control of light (bright, normal or off) for each of the 12 films. 50-6 Bank wide plexiglas viewing panels.

Fast action–60 Second maximum access time to any film. The 50 button–MV200C Selector is available for automatic case retrieval.

Hundreds of MV200A are in use. Radiologists agree it is the best and now the MV300A offers still greater value; easier reading, larger capacity, faster action, minimum floor space and lowest cost per film!

MANUFACTURED BY:
S. & S. X-RAY PRODUCTS INC.
87-93 JAY STREET, BROOKLYN, N.Y. 11201
Telephone: 212-624-7035

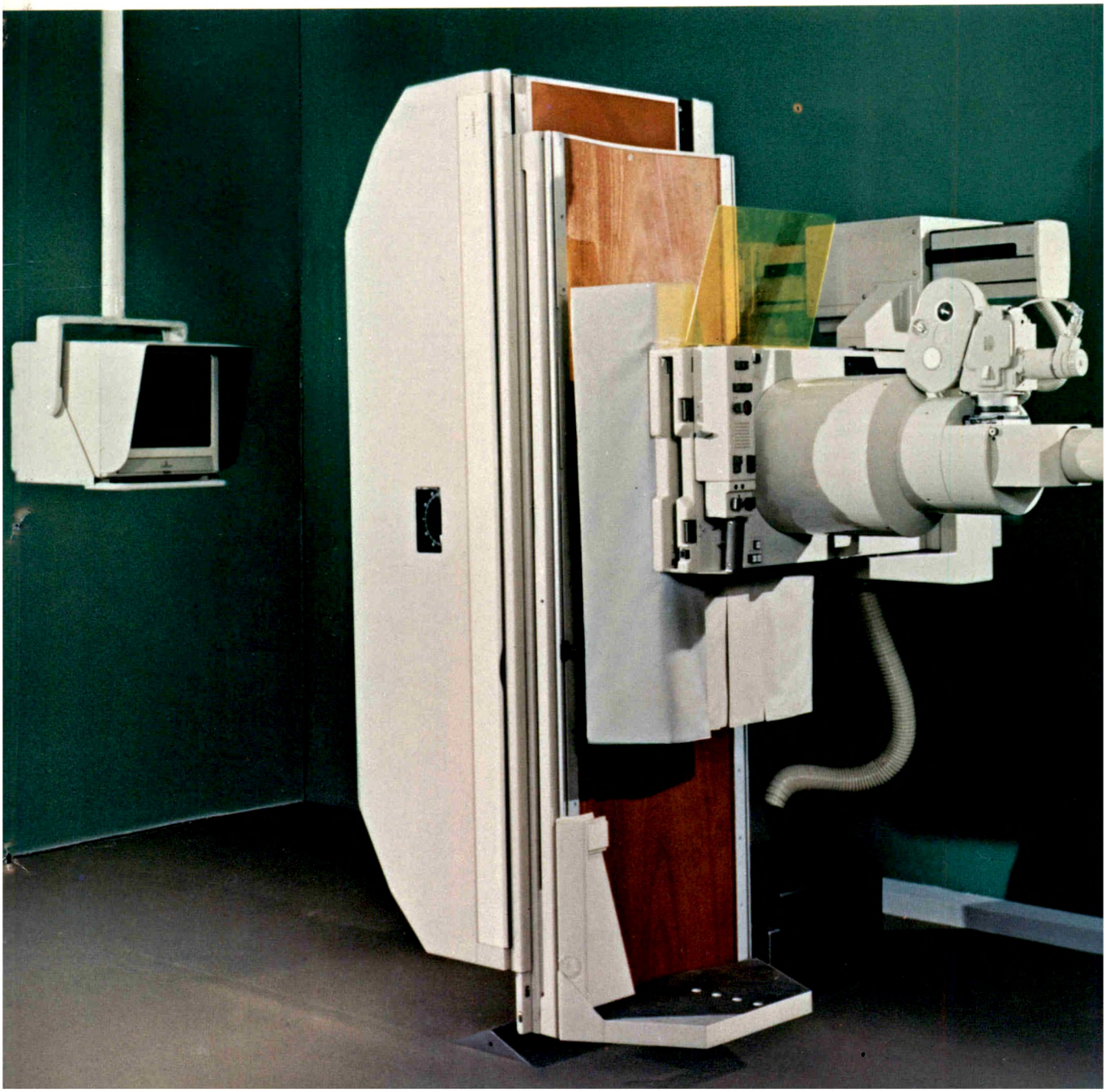
MV300A 300 Film Viewer	$ 9995.00
MV200A 200 Film Viewer	7630.00
MV200C Selector	1395.00

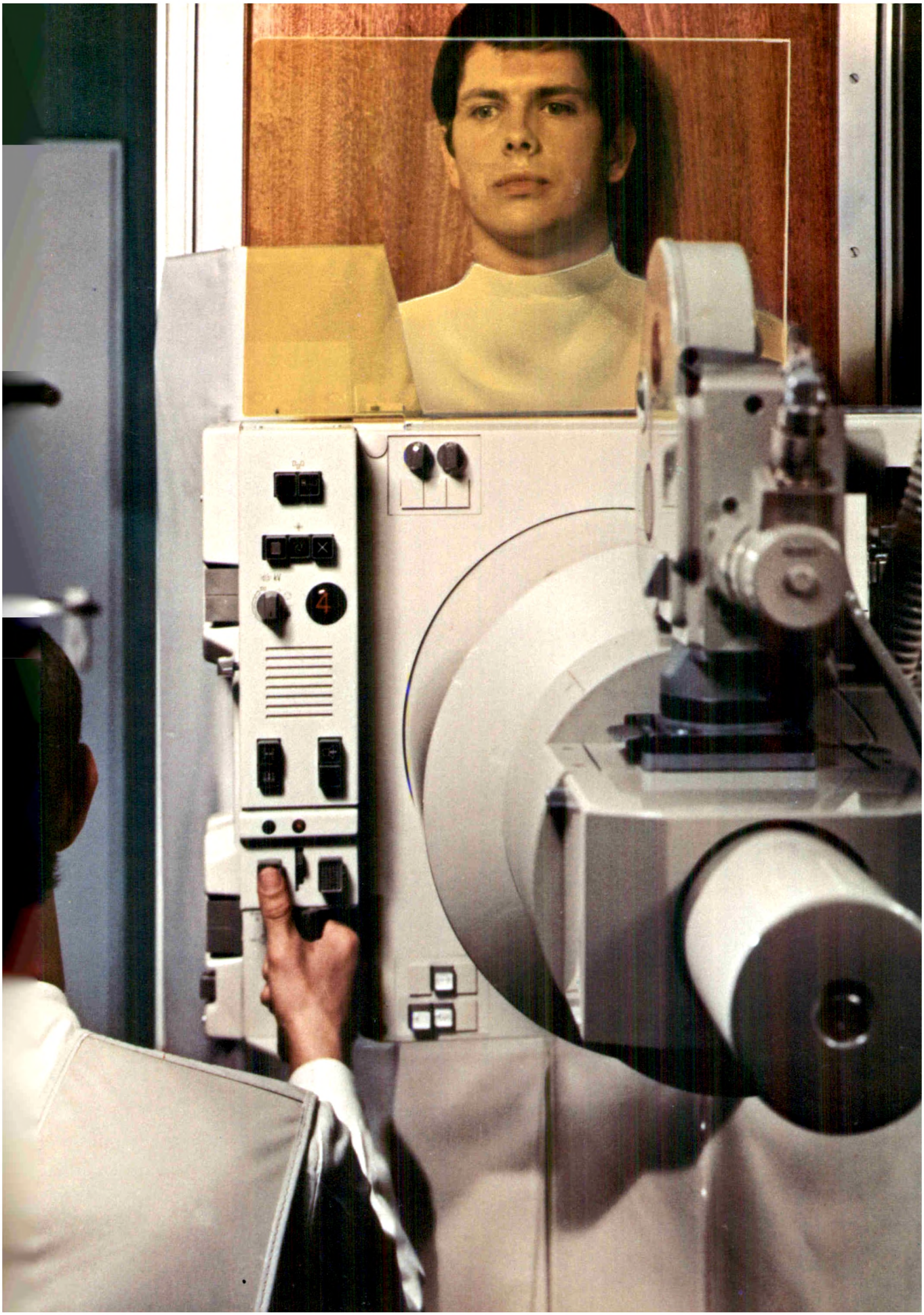
FOB Brooklyn, N.Y.
Brochures, Dimensions & Users List on request.

ONE OF THE MANY S. & S. ACCESSORIES SOLD BY YOUR LOCAL X-RAY DEALER

Siemens

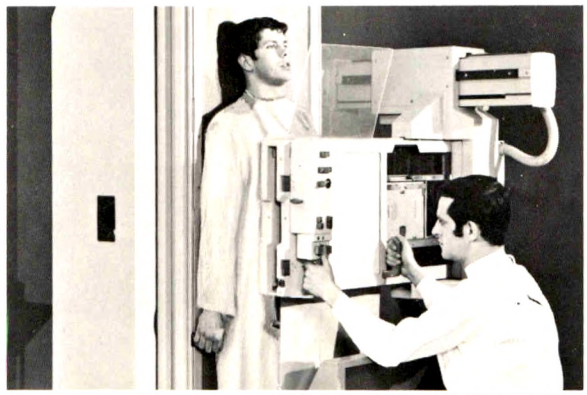
Introducing a fully coordinated
x-ray examination system for the most demanding
requirements of hospitals and private practice.

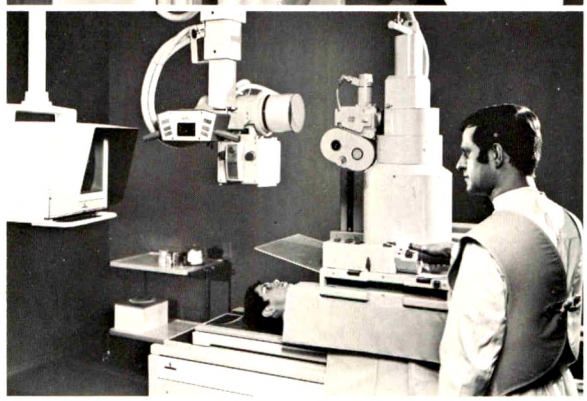




The Sireskop 3 with Explorator 35 spotfilm device brings new operating simplicity to extensive x-ray examination procedures.

The Sireskop 3 is a fully motorized examination table capable of any desired examination position, from vertical to horizontal and to 90° Trendelenburg. The table top moves, starts and stops smoothly without jittering at either of two speeds to match different examination techniques. The Sireskop table top "floats" so that it can be shifted by more than 19 inches headwards and footwards, and about 5-1/2 inches transverse.





The Explorator 35 spotfilm device with its comprehensive cassette program is a completely new development. With its use in conjunction with the Sireskop 3 table, the radiologist can concentrate on the examination procedure with his patient rather than computing, re-computing, setting, re-setting, positioning and re-positioning.

The spotfilm device is suspended unilaterally from a supporting arm. This permits patients to step behind the device from either right or left. And it means the Sireskop 3 can be selected independently of the room arrangement. The Explorator 35 has a control arm that can be operated with either the right or left hand. The controls for fluoroscopy, radiography, collimator actuation and table-top movement are all within reach of the fingers of one hand.

The Explorator 35 accepts standard cassette sizes up to 14 in. x 14 in. All cassettes can be inserted without frames from the front or rear.

When fitted with the Sirecon image intensifier television unit, the Sireskop 3-Explorator 35 combination offers many advantages.

Other special arrangements are possible for cinefluorography, 70 or 100 spotfilming and video recording. Remote control of the equipment is also available.

The Sireskop 3 will accommodate spotfilm devices, Explorator 25 (9-1/2" x 9-1/2"), and Explorator 30 (10" x 12") in addition to the Explorator 35.

Siemens

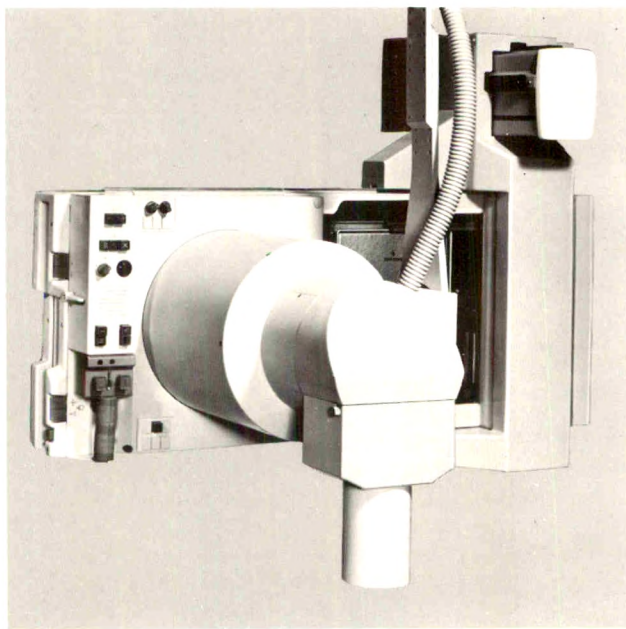
Explorator 35 Spotfilm device leaves nothing to chance.

The Explorator 35 spotfilm device is designed for convenience, ease of operation and, of course, versatility. It provides for electrical control of all the switching procedures. Its light weight and compact size make for ease and flexibility of movement. This is true even with the large-format image intensifier because an auxiliary motor drives the device up or down at the touch of a finger.

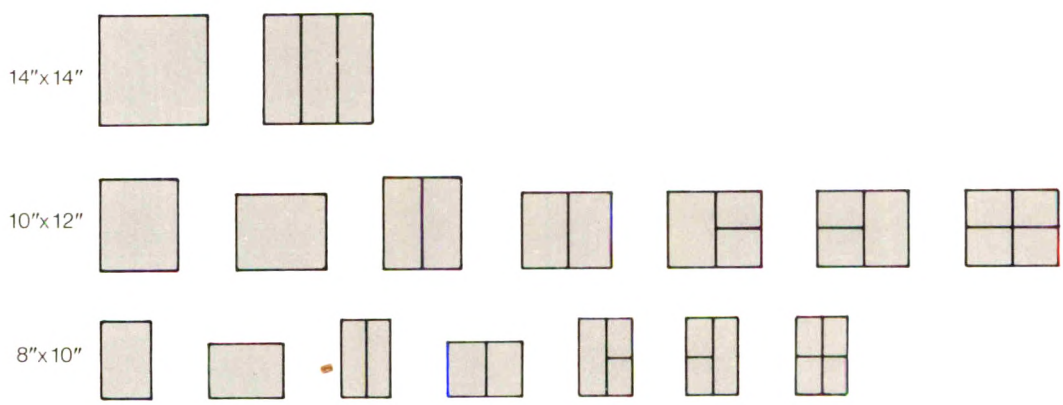
For details, contact your Siemens representative or Robert MacKinnon, Siemens Corporation, 186 Wood Avenue South, Iselin, New Jersey 08830. Phone: (201) 494-1000.

SIEMENS

Siemens. A three billion dollar name in quality products.



Explorator 35 cassette program.



Here are four ways to handle your in-vitro and in-vivo testing requirements.

IN-VITRO

LOGIC™ scintillation well counter

LOGIC™ is a simplified integrated spectrometer and well counter that's easy to operate. Most important is the LOGIC™ unique service commitment. When problems arise, a unique service program goes into action and your unit is back in operation fast. Every LOGIC is built with solid state and integrated circuitry to give greater reliability and less downtime.

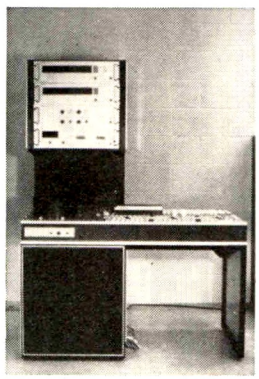
The LOGIC™ symptom describing manual allows you to pinpoint most service problems in minutes. A call to our technical representative confirms or corrects your diagnosis immediately. The correct plug-in circuit board or a replacement LOGIC™ is air shipped to you the same day. You're back in operation within 24 hours. In short, if you have trouble with a LOGIC™, we'll repair or replace it with a service loaner in 24 hours or less.



LKB/Wallac automatic gamma sample changer

The LKB-Wallac 80000 automatic sample changer handles a large capacity of samples to free your skilled staff for other duties. It allows long uninterrupted automatic runs with either uniform or intermixed samples.

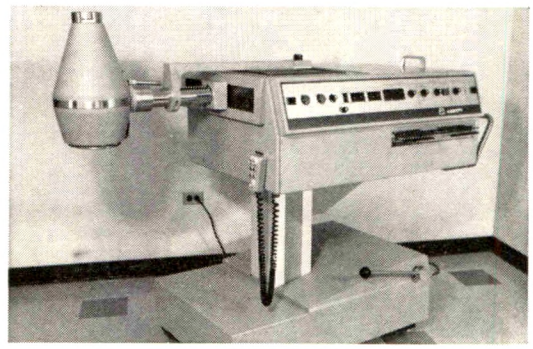
The sample conveyer operates as an endless belt giving you fast, safe and secure pneumatic handling of samples. There are two methods for positive sample identification before measurement, its position on the conveyer belt, and a binary coded cap. And, samples are changed in only 10 short seconds. Data readout is supplied in printed form or on punched tape. The Wallac automatic sample changers simple foolproof controls allow you to handle your needs efficiently and accurately.



IN-VIVO

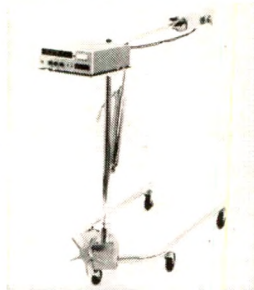
GRAPHIC™ Rectilinear scanner

GRAPHIC™ operation is simple. The control panel is designed for a logical left to right set-up procedure. Start at the left with Power On and work your way in a logical sequence to the right of the panel to Scan On. GRAPHIC™ two-position film cassette allows you to scan 14″ x 17″ in either direction, across the chest or lengthwise along the body. GRAPHIC™ will accomodate a variety of large scan field requirements with uniform ease. And, GRAPHIC™ is built to last requiring a minimum of service attention. It's so rugged that we warranty it for mobile operation. You have to be tough to work under these conditions.



LOGIC™ with uptake module

By simply adding a medical stand and probe with collimator to either of two LOGIC™ models, the 111 or 121, you're ready for thyroid uptake studies. The LOGIC™ modular concept provides add-on capabilities for you. Buy what you need when you need it. And, you still get the unique service commitment provided only by LOGIC™ . . . when problems arise, we'll repair or replace your instrument with a service loaner in 24 hours or less.



And, these instruments come from a full-line supplier who assures you of a continuing service commitment to train and assist your personnel in all their needs. It's our privilege to keep your Abbott instruments operational.

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How strong the rest of the chain is doesn't matter.



It's as true in a fluoroscopic image chain as it is in any other chain. *Every* link has to perform.

But equipment purchasing today has become a numbers game. And that's wrong. If each component of a system is not interfaced to work as a *matched* part of that system, all you've bought is simply more noise.

Profexray is dedicated to that systems concept. We take our pioneering break-throughs like large-format spot-film cameras and the world's first commercially available *x-ray* TV camera and integrate them into an image chain that provides maximum capability—not just the latest technology. We're constantly looking for ways to up-grade each link of that chain. Rest assured that as we do, we'll link up the new technology to weld a stronger chain. To give you a *totally* up-graded system.

Call your Profexray representative today. Let him show you how to link progress with performance.

Litton **PROFEXRAY**
Des Plaines, Illinois 60018



Clinical Radiotherapy Sources

Amersham/Searle has a range of sources designed for maximum integrity, convenience in handling and safety for both operator and patient.

Cesium-137 for Interstitial and Intracavitary Use.
Needles and tubes of welded platinum-iridium alloy containing Cesium-137 in insoluble form. All activities stated in terms of milligrams radium equivalent.

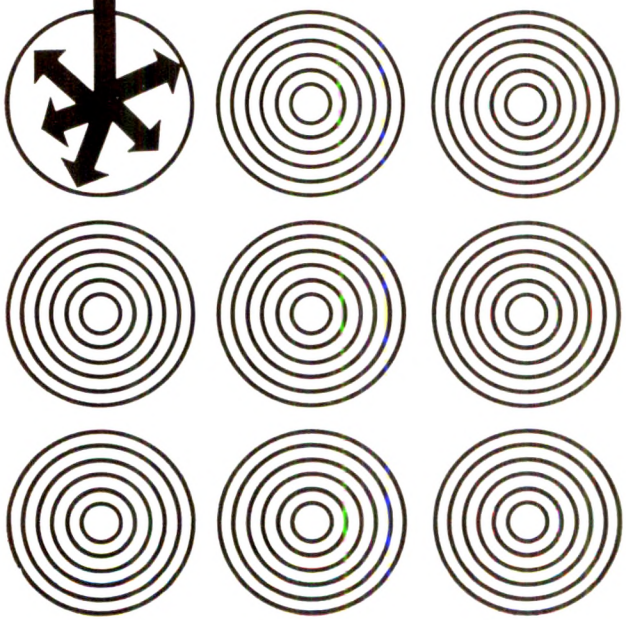
Clinical Beta Sources.
Ophthalmic Applicators
Surface Applicators
Teletherapy Sources
Extracorporeal Blood Irradiators

Please write or call our home office—details of specifications and prices will be sent on request.

OUR SPECIFIC ACTIVITY IS SERVICE.

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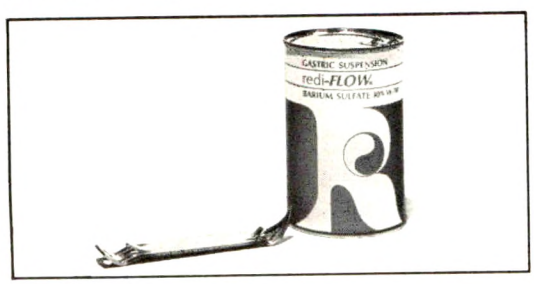
Does your barium suspension contain a secret ingredient?

Such as contamination, perhaps?

Recent published material shows that "Bacterial contamination* was found on three occasions in barium sulfate suspensions prepared for use in examination of the gastro-intestinal tract."[1]

redi-*FLOW*® takes that suspense out of barium sulfate suspension with terminally sterilized individual doses—"the ultimate step in prophylaxis."[1]

Three recent isolated instances show that the possibility of contamination in barium sulfate preparations is a cause for concern. Circumstances indicate that patient-personnel contact is the most likely source.



redi-*FLOW*, the instant barium suspension in individual dosage cans . . . reduces the danger of such contamination to the minimum.

What's more, redi-*FLOW* is a valuable time-saver with: Uniform viscosity for consistent, reproducible film results; no tedious preparation or messy cleanup; fewer patient problems because redi-*FLOW* goes down deliciously.

redi-*FLOW*—the simpler, safer, barium sulfate suspension . . . available in Gastric and Colonic forms.

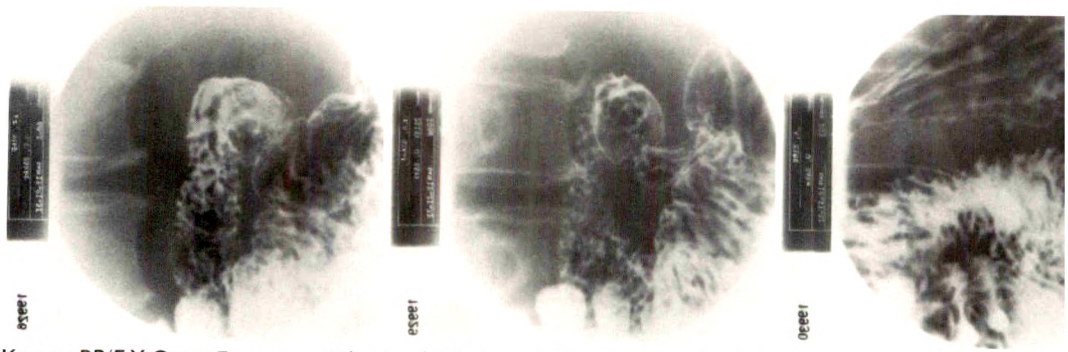
Flow Pharmaceuticals, Inc.
936 Commercial Street
Palo Alto, Calif. 94303

**Escherichia coli, Klebsiella, Streptococcus faecalis, Pseudomonas, Achromobacter, Aerobacter, Clostridia.*

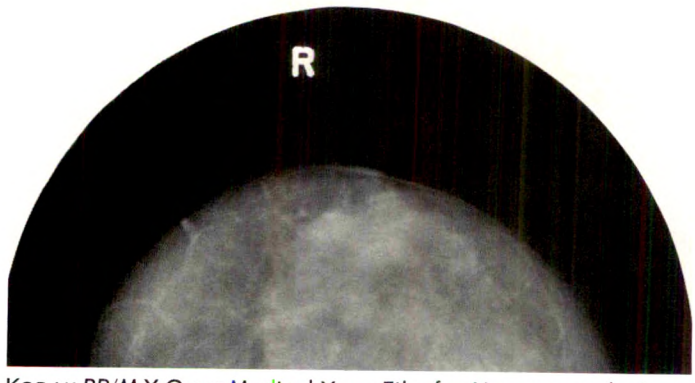
[1] Radiology, 97:182-3, Oct. 1970.

Quality commitmer

The common denominator for all Kodak special-purpose x-ray is quality—just as it is for your own special radiographic nee



KODAK *RP/F* X-OMAT FLUROSPOT Film is a high-speed, 90-second automatic processable film capable providing excellent contrast and definition. This is demonstrated in the above views where the mor of the pyloric end of the stomach and the first portion of the duodenum are well shown.



KODAK *RP/M* X-OMAT Medical X-ray Film for Mammography is an extra fine grain, high-contrast film designed for radiography of the breast. The views illustrated here show dense fibrocystic disease throughout. Note that the veins near the outer border, the retracted nipples, and skin layer are clearly defined.



When your diagnostic program calls for special radiographic or photographic applications, choose from a broad range of quality films designed to meet your specific needs.

For radiographic examinations of the breast, you can choose KODAK *RP/M* X-OMAT Medical X-ray Film for Mammography. It's an extremely fine grain, high-contrast, direct-exposure film that gives you excellent radiographic detail.

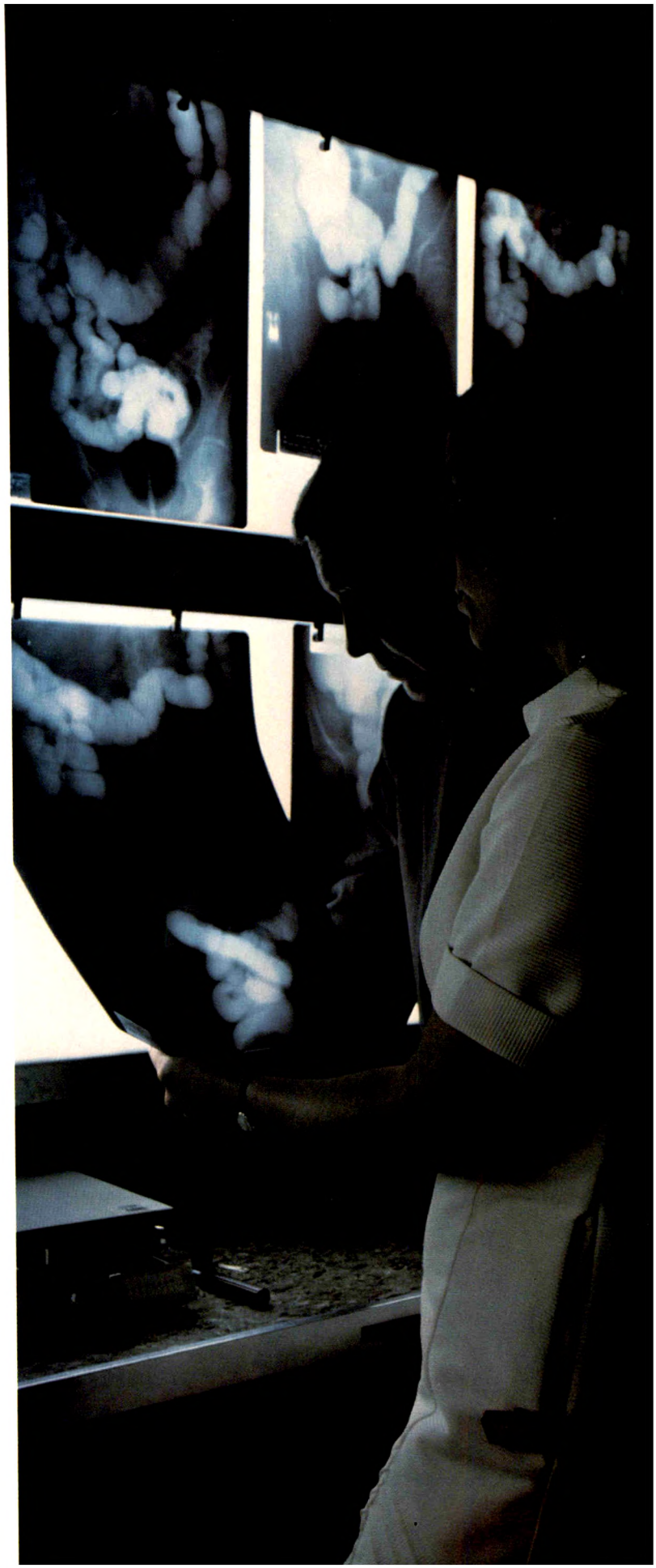
When preparing the mask in the subtraction of radiographic images, choose KODAK *RP/SU* X-OMAT Subtraction Film. Compared to KODAK Commercial Film 4127 (ESTAR Thick Base), its reduced speed simplifies and allows more precise exposure control of the film in a printing frame or in a mechanical contact printer.

And we also have special films you can use for spot technics, for therapy localization procedures, and for duplicating radiographs of patient records for teaching aids. Like all Kodak medical x-ray films, they provide consistent high quality and excellent image detail.

Each of these films is designed to give you the timesaving advantages of automatic 90-second processing.

Your Kodak Technical Sales Representative is available to demonstrate their advantages. Or you can consult your x-ray products dealer.

EASTMAN KODAK COMPANY
Radiography Markets Division
Rochester, New York





IT'S JUST ANOTHER PLASTIC BAG WITHOUT ITS SOL-O-PAKE™

Sure our **E-Z-EM®** Prepackaged Disposable Barium Enema is among the finest available. The point is, it wouldn't be if the Sol-O-Pake barium inside weren't also among the finest available.

And the reason we know it is, is that *it's* our product too. A premium barium, providing superior performance on two important counts. One, Sol-O-Pake mixes instantly and remains in suspension. So there are no clumps to clog tubing, or produce artifacts in your film. And two, Sol-O-Pake coats excellently over a wide range of densities. So in high or low kilovoltage procedures . . . filled or postevacuation examinations . . . routine or double contrast studies, you get the fine mucosal detail you need.

Now that you know about Sol-O-Pake, remember there's an upper G.I. form as well.

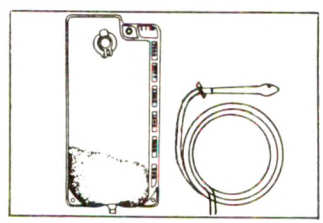
SOL-O-PAKE™

In Upper G.I. Cup



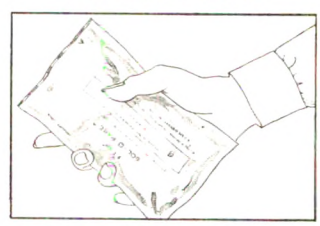
Maximum convenience. Fast flow through small bowel. Vanilla, chocolate, fruit or unflavored.

For Lower G.I.



Prepackaged in the amount you specify in E-Z-EM kits. For use with or without an evacuant.

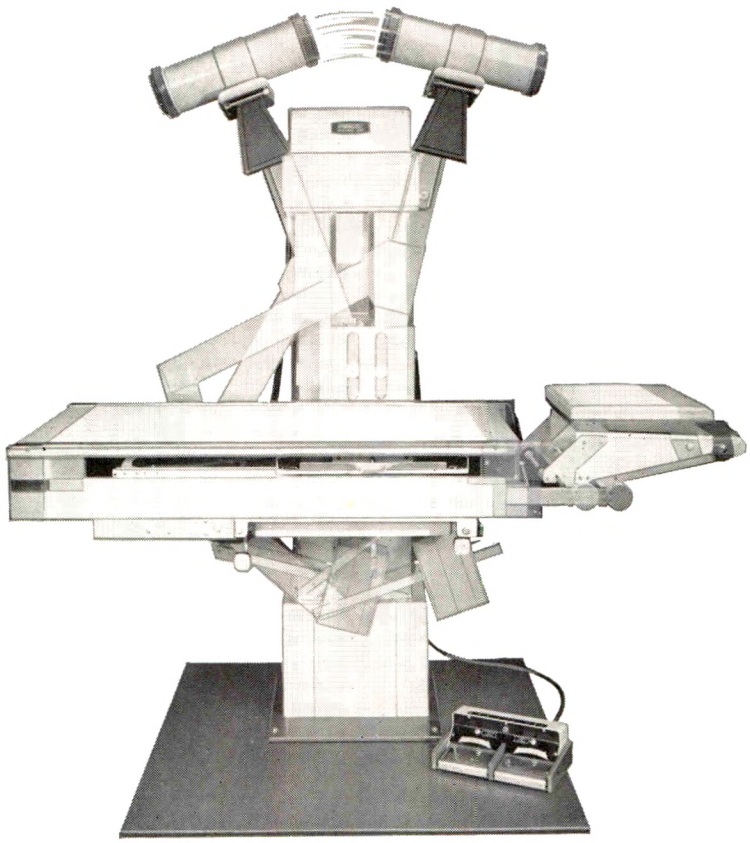
In Individual Packets



Economical. Packaged in individual four or six ounce packets. Vanilla, chocolate, fruit or unflavored.

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TOMOGRAPHY



. . . a new Capability for the Hydradjust!

Now, the versatile Hydradjust Urological Table can be equipped for tomographic studies to further extend its capabilities in the hospital or clinic.

With the tomographic attachment, body section radiography can be accomplished with a single film cassette of desired size, or with a multi-film cassette "book", 10 x 12 inch or 14 x 17 inch film size.

The new tomographic attachment is engineered for simple, positive "lock-in" of the tube arm with the table; however, it may be just as easily disengaged, to return the Hydradjust to all its original capabilities.

Hydradjust tables now in use can be fitted easily with the new tomographic attachment. Ask your x-ray equipment supplier or write direct for literature.

LIEBEL-FLARSHEIM
SYBRON CORPORATION
111 East Amity Road, Cincinnati, Ohio 45215 U.S.A.

Products for Better Health from Sybron

The Clinac® 4's first innovation was reliability.

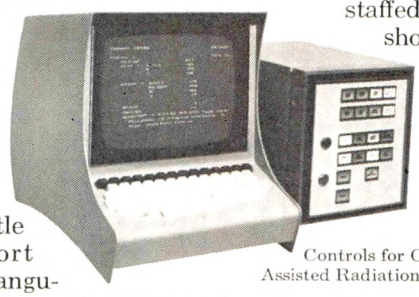
We didn't stop there.

And we haven't stopped yet.

When linear accelerators were first introduced they were used almost exclusively by hospitals with extensive physics support. Therapists were concerned that linear accelerators might require frequent tuning, and maintenance. Then Varian introduced the Clinac 4. Today it's so trusted that the Clinac 4 is the only therapy machine in over half of our eighty installations.

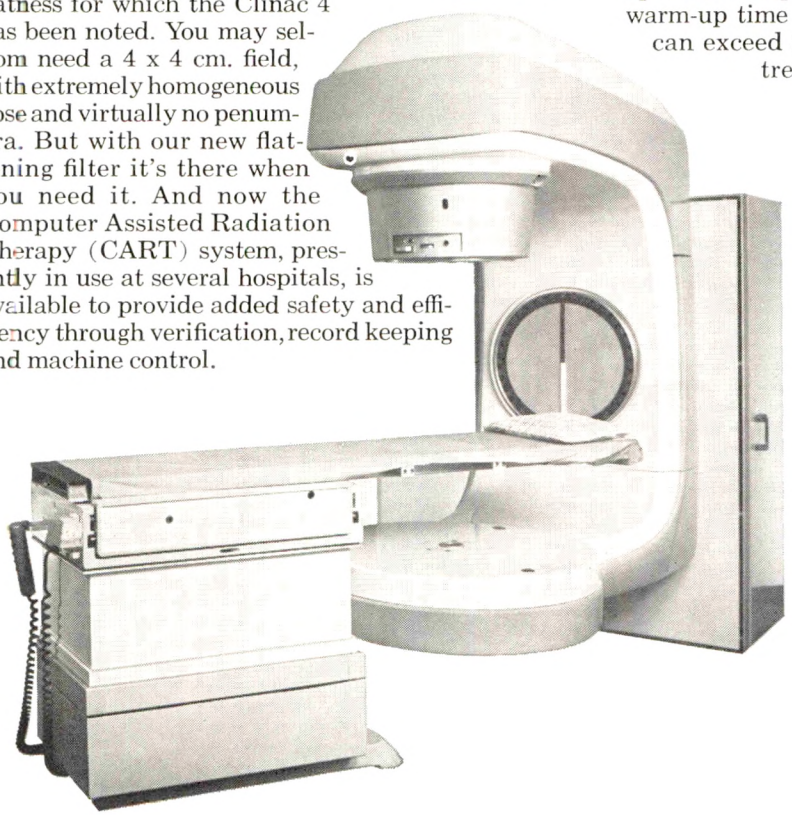
The reason is simple. By utilizing non-bent beam geometry, the Clinac 4 achieves the stability and dependability required in a heavy usage therapy machine.

Reliability without sacrificing performance. The Clinac 4 will do all a busy radiotherapist expects from a heavy-duty therapy machine. From precise small-field rotational treatments of the prostate to extra-large mantle fields. From complex multi-port wedged treatments to simple rectangular fields. All with the precision, reliability and repeatability needed by the therapists who use it.



Controls for Computer Assisted Radiation Therapy

Radiation therapy is constantly being improved. So is the Clinac 4. Our recently announced new flattening filter gives exceptional small-field flatness, extra-small penumbra, improved central-axis depth dose characteristics, and maintains the excellent large-field flatness for which the Clinac 4 has been noted. You may seldom need a 4 x 4 cm. field, with extremely homogeneous dose and virtually no penumbra. But with our new flattening filter it's there when you need it. And now the Computer Assisted Radiation Therapy (CART) system, presently in use at several hospitals, is available to provide added safety and efficiency through verification, record keeping and machine control.



Our Continuing Engineering Program has developed new accessories, including the wire mesh shadow block holder and laser backpointer, and has incorporated into new production models improvements based on experience from over 100 operating Clinacs.

We eliminated the service hassle. When you need assistance, a member of our extensive service group can either help you over the phone, or arrive within hours. Established field offices near our customers are staffed with men extensively trained in trouble shooting and maintenance. In addition, computerized analysis of machine malfunctions, preventive maintenance programs, and the modular construction of the Clinac all contribute to a low down time record.

We minimized construction problems and the need to rebuild. The Clinac 4 requires only slightly more shielding than cobalt and can usually be put into rooms designed for a rotational cobalt machine. Power and water requirements, access dimensions, and installation time for a Clinac 4 are planned for minimum inconvenience.

We designed in patient reassurance. The Clinac's clean design, small size, and smooth, quiet operation help relax your patients. Since there's no warm-up time before each treatment, and dose rates can exceed 350 rads/min. at isocenter, a Clinac treatment customarily takes little more than one minute.

Don't take our word about the Clinac 4. Call any of the radiotherapists now using one. Write us and we'll provide a list along with full information on the Clinac 4.

Varian Radiation Division, 611 Hansen Way, Palo Alto, California 94303, tel. 415-493-4000; Springfield, N. J., tel. 201-376-6632; Detroit, Mich., tel. 313-455-1570; Chicago (Park Ridge), Illinois, tel. 312-825-6232; In Europe: Zug, Switzerland, tel. 042-23-25-75.

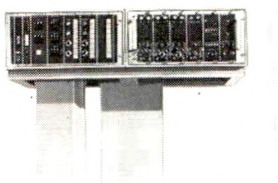
varian
radiation
division



2000 a.d.

...you've conquered cancer and heart disease, and switched to preventive medicine...









...the tools for getting there are here today.

The journey won't be easy. You'll have to travel past the limitations of your five senses. And be extra-familiar with the submolecular, as well as the intracellular, world.

We have the instrumentation to take you there. For instance, Nuclear-Chicago's Pho/Gamma Scintillation Camera. It's the choice of more than 95% of U.S. teaching hospitals and medical schools. They like its high resolution, ease of patient positioning, and its choice of 12 specialized collimators allowing one to switch from routine, "bread-and-butter" imaging to highly sophisticated procedures.

Our Pho/Dot is the world's most proven rectilinear scanner. Our Liquid Scintillation and Automatic Gamma Counters embody the newest ideas in capability-expansion for radioimmunoassay and competitive binding tests. And we could say equally good things about our Pho/Gamma Tomocamera, Data/Store Playback System, and similar products. But that wouldn't be modest.

When the last of the Great Plagues that afflict humanity has been wiped out, it will be because dedicated people have pin-pointed the method of attack. With instruments like these.

NUCLEAR-CHICAGO CM-276

A SUBSIDIARY OF G. D. SEARLE & CO.

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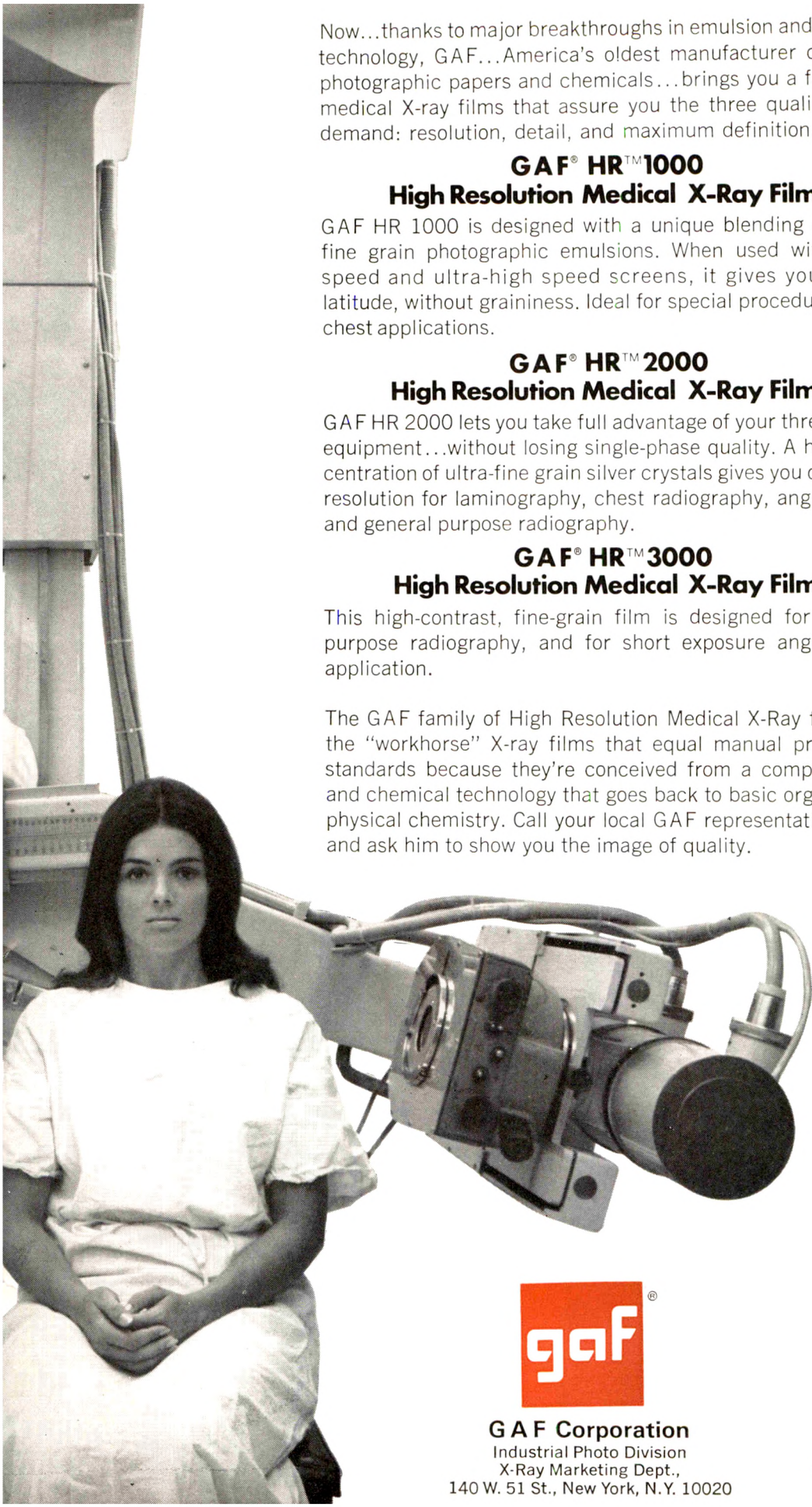
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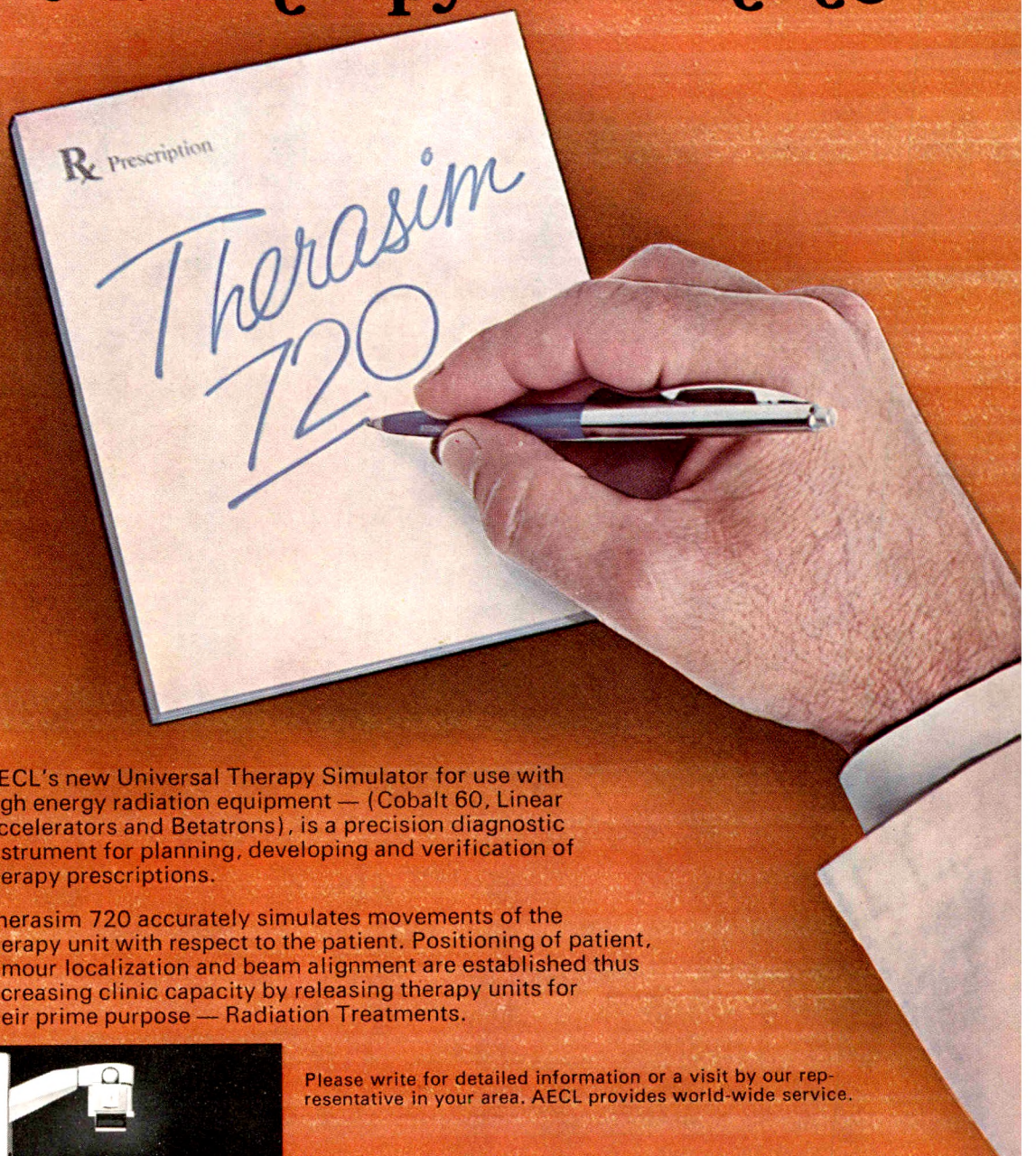


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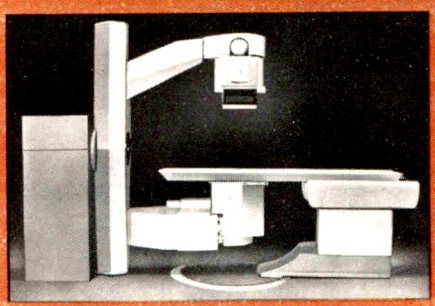
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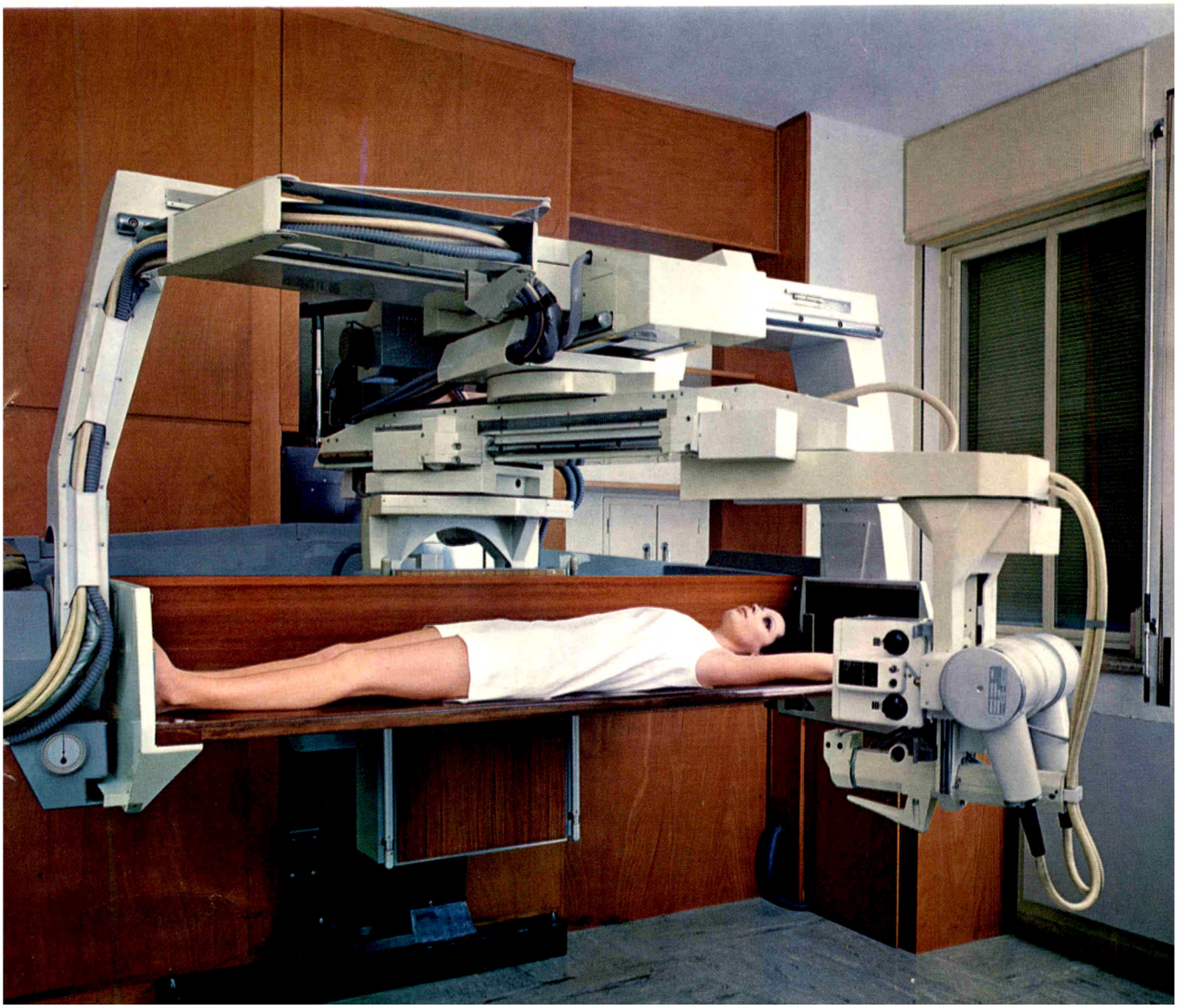
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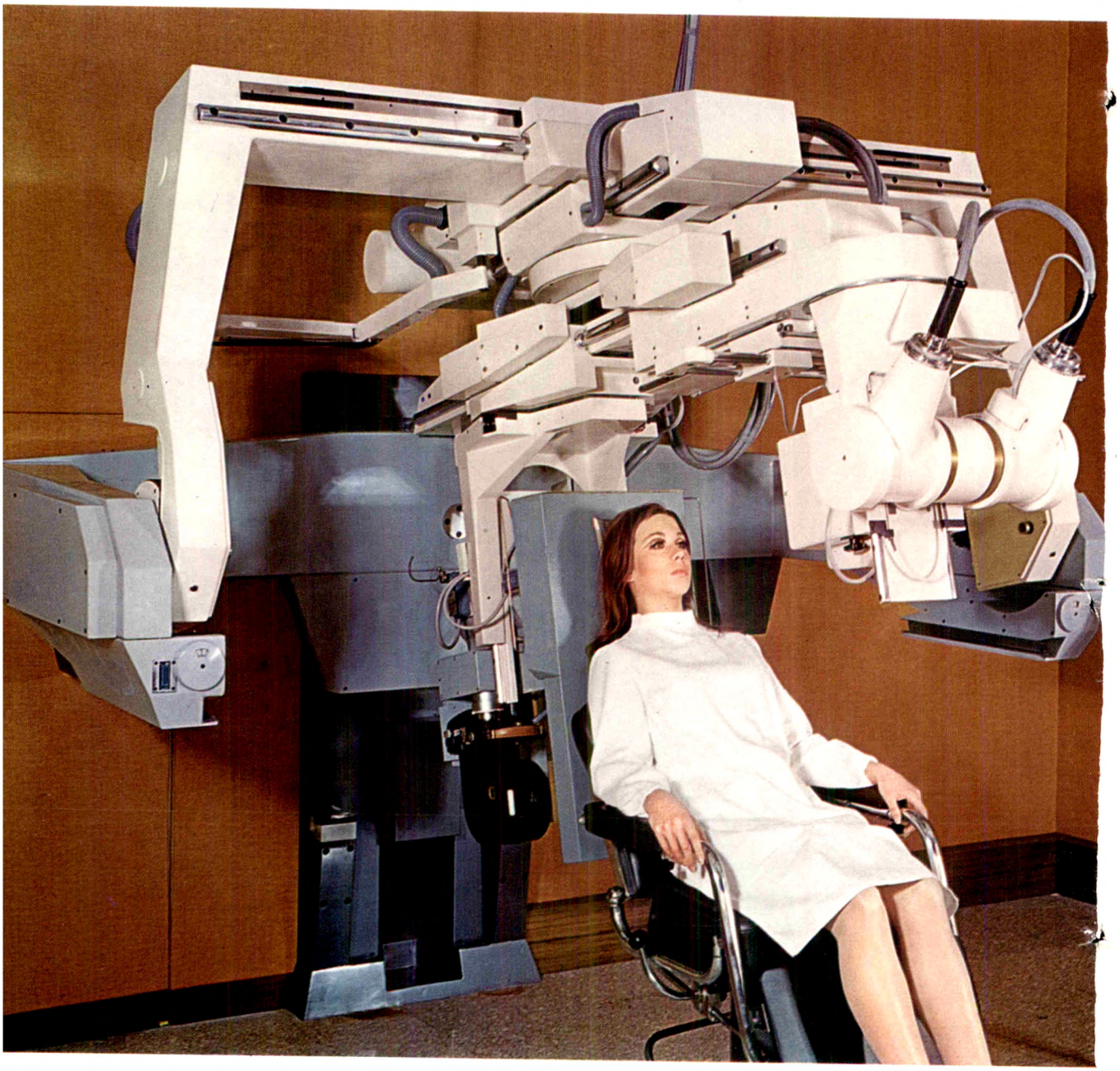
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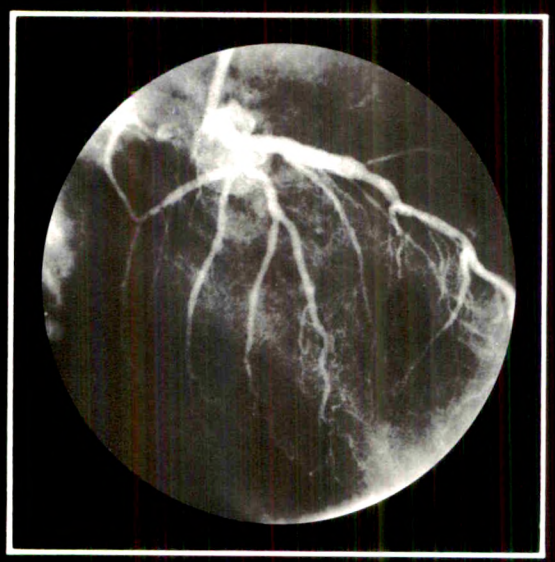
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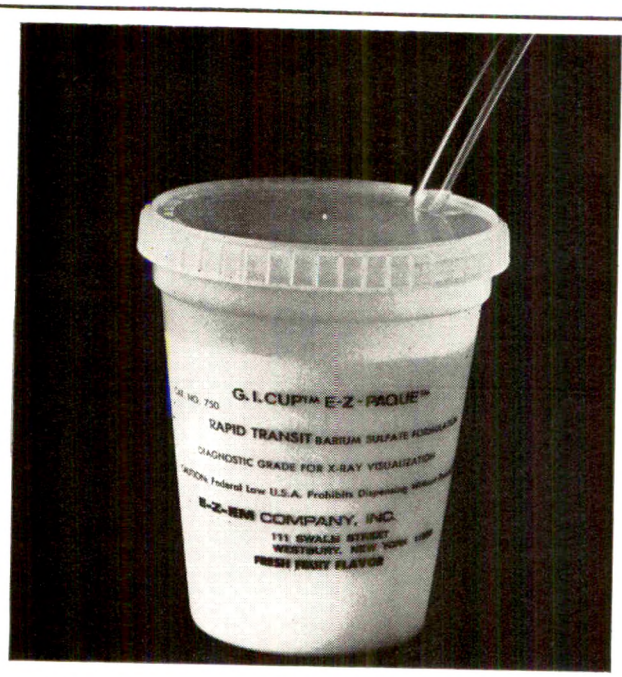
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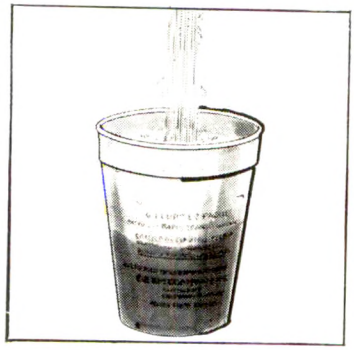
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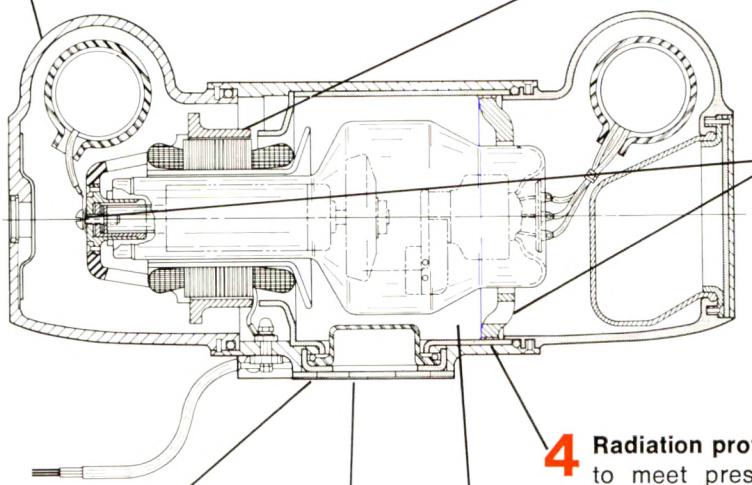
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Here are a few of the important protocols being done. And some unique capabilities.

Flood Correction

Eliminates field artifacts and reduces the possibility of false negatives and false positives (due to gamma camera sensitivity non-uniformity) to a minimum. Many hospitals accustomed to the value of flood correction won't look at a study until it has been corrected.

Increased Resolution

Increased resolution is a natural offshoot of field uniformity or flood correction. Most gamma cameras are detuned off the photopeak to avoid the phototube rosette pattern artifact. In other words, the cameras are "compromise tuned" for concomitant optimization of field uniformity and resolution. However, some gamma cameras, such as the Radicamera, can also be operator tuned right on the photopeak to ensure the highest possible resolution. Subsequent flood correction by the image data processing system can then be used to eliminate camera uniformity variations.

Residual Isotope Subtraction

Permits you to subtract the data remaining from a previous study before you evaluate the followup.

Data Exponentiation

Allows you to substantially enhance subtle contrast differences by squaring, cubing or raising the data in each image channel to a given power.

Image Frame Condensation

Lets you add image data in successive frames to optimize statistics in subsequent time-activity histograms.

Automatic Study Sequencing

Permits you to automatically acquire a dynamic frame sequence followed by a static image. With a protocol such as this, you can automatically acquire half second sequential frames for a specified number of seconds—followed by a ten second static image—when doing dynamic cerebrovascular transit studies.

Exposure Optimization

Can be achieved by telling the system to continue data acquisition until one of the data points comprising the image contains a given number of counts. With this capability, different views of the liver, for example, all contain the same "brightest level." Much better (more uniform) image intensity/exposure control from view to view is obtained in this manner than is possible when taking views to preset time or total image field counts.

Quick Histogram Generation For Multiple Areas Of Interest

A particularly important capability of image processing systems. Since much of the work done with such systems is dynamic and involves histograms, speed is of the essence. Some image processing systems can generate separate time/activity curves for each area you've defined within a 25 minute renal function series in just seconds. When compared to conventional recording systems, over a period of a year an image processing system can save you a few hundred hours in histogram generation time alone.

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Another primary feature. You can not only store and replay a multi-frame study, but also show only the *change* from frame to frame, generate *isocontour plots* for successive frames, quickly define and view *profiles* across each image frame, and print out an activity map showing the *number of counts comprising each image data point.*

Ability To Do Several Clinical Routines Not Otherwise Possible

This is probably the most important capability of an image processing system. Because most such systems are programmable, everything from isotope inventory to fractional clearance rate calculations is possible. Image processing systems are being used for clinical protocols ranging from determination of hepatic phagocytic, metabolic and blood transit changes to verification of the existence and degree of septal defects. Cardiac ejection fraction studies are being made routinely in some hospitals as part of the serial evaluation of, for example, mitral valve disfunction characterized by valvular regurgitation, and left heart failure associated with myocardial infarction. While it is sometimes possible to obtain function indices using alternative nuclear medicine procedures, the speed, convenience, accuracy and replicability available with a good image data processing system cannot be duplicated.

Conclusion

There are literally dozens of procedures—vital ones—which are only possible with a programmable image processing system. Such systems have graduated from the research phase of their development. They belong in any up-to-date clinical nuclear medicine imaging laboratory. Image processing is no longer image obfuscation. It is better resolution. Organ function indices. Scintigraphs free of instrumentation artifacts. Region-of-interest analysis. Exposure optimization. Contrast enhancement. Activity maps. Tumor to non-tumor ratios. Pre- and post-operative comparisons. Improved pancreas visualization. And a host of other clinically valuable capabilities.

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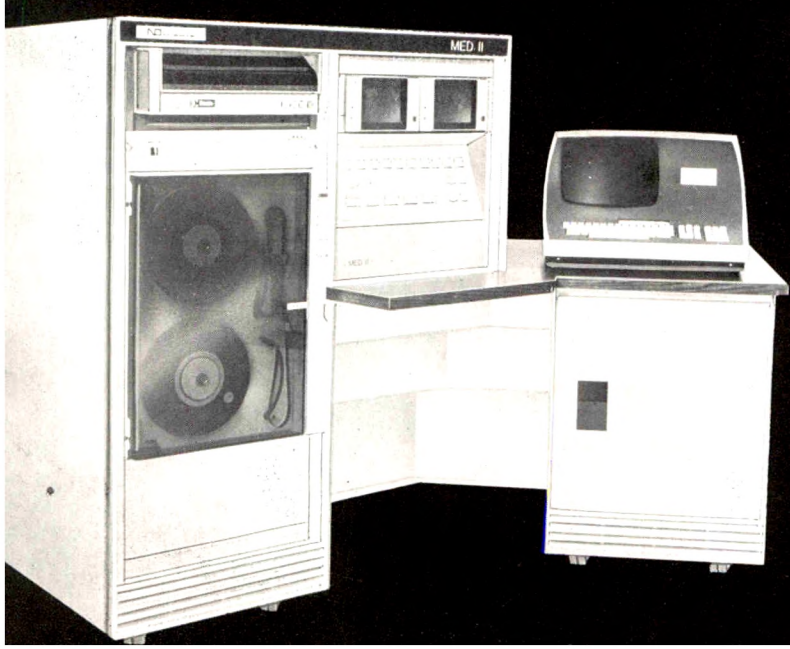
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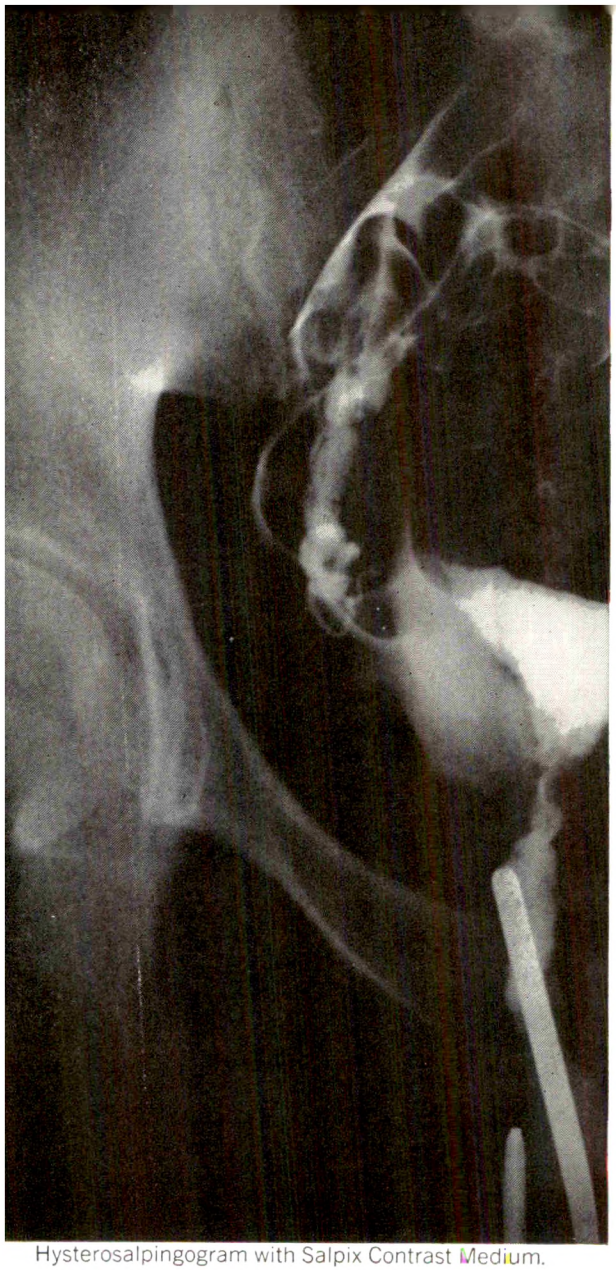
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VOL. 116 SEPTEMBER, 1972 NO. 1

RESULTS OF ARTIFICIAL SLOW FLOW ANGIOGRAPHY WITH ARTERIOVENOUS ANEURYSMS IN THE SUPPLY AREA OF THE EXTERNAL OR INTERNAL CAROTID ARTERIES*

By HENK VERBIEST
UTRECHT, THE NETHERLANDS

ARTIFICIAL slow flow carotid angiography, a method described in a previous paper,[15] is performed by means of percutaneous carotid injection of the contrast medium with digital compression of the common carotid artery below the site of puncture. We have called this method proximal compression angiography (P.C. angiography).

METHOD AND CONTRAINDICATIONS

The use of the term slow flow angiography may give rise to the question of possible dangers resulting from prolonged contact time of the contrast medium with the neural elements. This danger would be present in case of prolongation of the capillary phase (intermediate phase according to Taveras and Wood[13]), because it is during this phase that noxious effects of the contrast media on the neural structures are supposed to occur.

With the present technique it is only the arterial phase which is prolonged.

Analysis of P.C. angiography in 60 cases of various conditions, reported in a previous paper, revealed that:

(a) in one-half of the cases the contrast medium was arrested in the internal carotid siphon so that there was no intracranial transport of the contrast medium during the 3 second slow flow angiography. The remaining contrast medium escaped through the external carotid circulation;

(b) in one-third of the patients there was rapid transport of the contrast medium through the middle cerebral artery, due to collateral circulation acting on the carotid siphon. These effects may be compared with those occurring during the frequently applied method of examining cross circulation (injection into one carotid artery during digital compression of the contralateral carotid artery in the neck);

(c) in one-sixth of the cases there was prolonged staining of cerebral ar-

* From the Neurosurgical Department, State University of Utrecht, The Netherlands.

teries (in most cases of the middle cerebral artery and in a few instances of both the anterior and middle cerebral arteries). This was called a frozen appearance, since there was no transport of the contrast medium during the 3 second compression of the common carotid artery. Subtraction pictures of the 3 roentgenograms did not show regional blushing due to filling of fine arteries or capillaries.[1]

After releasing compression of the carotid artery there was no prolonged intermediate phase.

In conclusion, the present slow flow angiography technique is limited to the arterial phase and there is no reason to expect much harm from the 3 second contact of contrast medium with normal arterial walls.

A similar form of angiography as described in this paper has been applied by various other investigators who, however, used another name and applied it for another purpose. It was called progress arteriography and was performed to estimate the size of intracranial aneurysms following ligation of the carotid artery. The method consisted of injection of the carotid artery distal to the site of permanent occlusion. Perusal of the literature revealed no reports of complications due to the progress arteriography.[2,3,5,6,8–10,14]

Based on present knowledge, and observing the contraindications described below, there is reason to believe that the selective prolongation of the arterial phase with P.C. angiography carries less risk of noxious influence of the contrast medium on the neural elements than when performing routine angiography in case of a prolonged intermediate (or so-called capillary) phase due to brain edema or other conditions.[9]

The *contraindications* of P.C. angiography are based on certain hemodynamic consequences of proximal occlusion of the common carotid artery. P.C. angiography is not performed in patients exhibiting electroencephalographic abnormalities following compression of the carotid artery. In all patients routine percutaneous angiography was made first. The angiograms were carefully studied, and, if they showed a diminished flow rate, arterial spasms, signs of intracranial vascular disease or injection of the contrast medium in the wall of the carotid arteries (a considerably reduced risk when using a Becton-Dickinson 18 G teflon cannula), use of P.C. angiography was considered contraindicated. The angiographies were performed under endotracheal anesthesia (nitrous oxide-oxygen-halothene). This depresses the baroreceptor reflexes. In case of untoward reflex responses, the proximal compression was discontinued.

By following these precautions, and observing the contraindications mentioned above, we have observed no untoward effects in over 380 cases examined up to the present.

TECHNICAL DETAILS

The common carotid artery was occluded using digital compression, when there was disappearance of the pulse in the ipsilateral temporal artery and cessation or considerable reduction of reflux of blood from the angiographic needle. Then, 8 ml. of 60 per cent urografin was injected. Injector pressures between 1.5–2 kg./cm.^{2} were employed. When 3 ml. was left in the syringe, the first film was exposed, followed by 2 other exposures with intervals of 1.5 seconds. After the third film had been exposed, the digital compression was released. With adequate experience the total duration of digital compression does not exceed 8 seconds.

The most reliable criterion of effective proximal occlusion of the common carotid artery in the P.C. angiograms was the visualization of the internal and external carotid arteries, or one of them, for 3 seconds (*i.e.*, in all 3 films) following injection of the contrast medium.

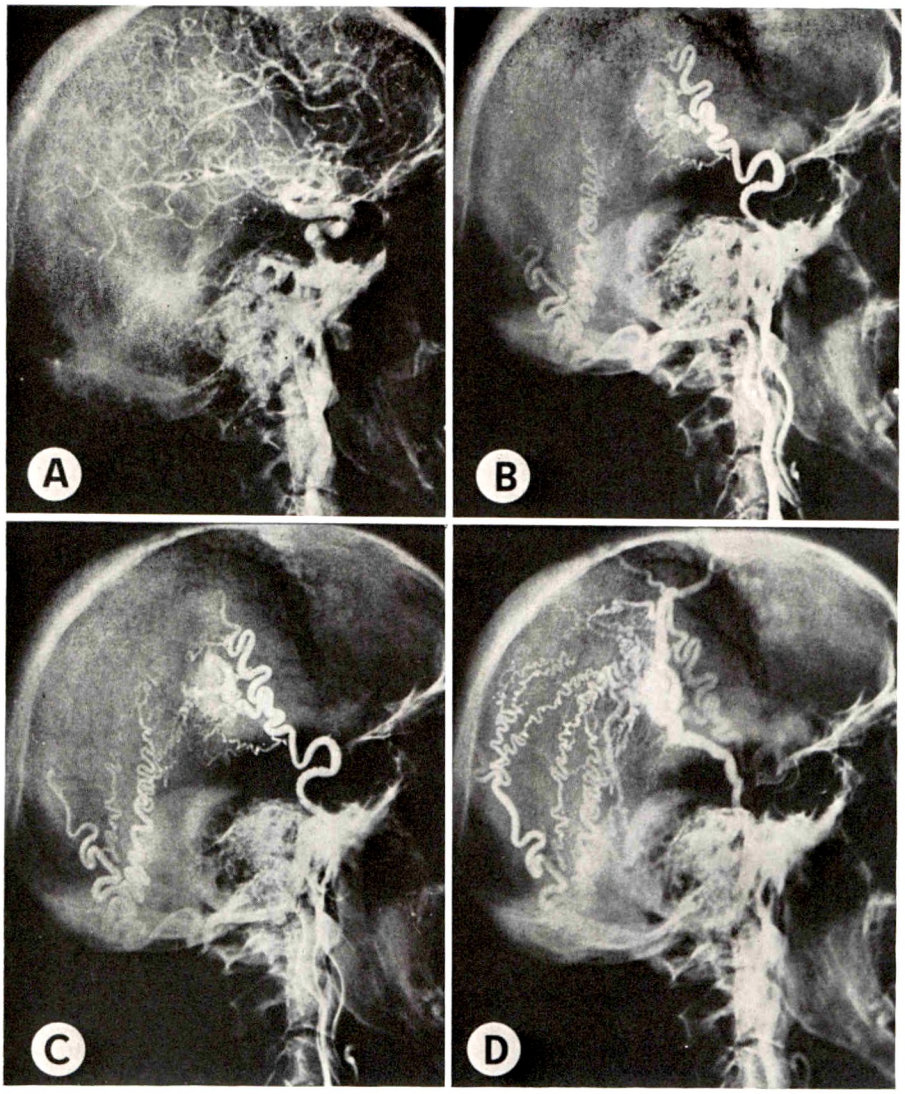


Fig. 1. (*A*) Routine angiogram with injection of contrast medium into the common carotid artery shows no staining of arteriovenous aneurysm (A.V.A.). (*B–D*) Proximal compression (P.C.) angiograms with 1.5 second intervals and injection of contrast medium at the same site as in *A*. Note complete suppression of intracranial circulation (*angiographic subtraction*) and preferential filling of A.V.A. All arterial feeders are visualized. Slow circulation (*angiographic addition*) of arterial feeders and venous drainage in *D*. Example of monovascular dependency of A.V.A., namely on main trunk of external carotid artery.

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RESULTS

Distal to the carotid occlusion there is a decrease in pressure and mean flow. Various

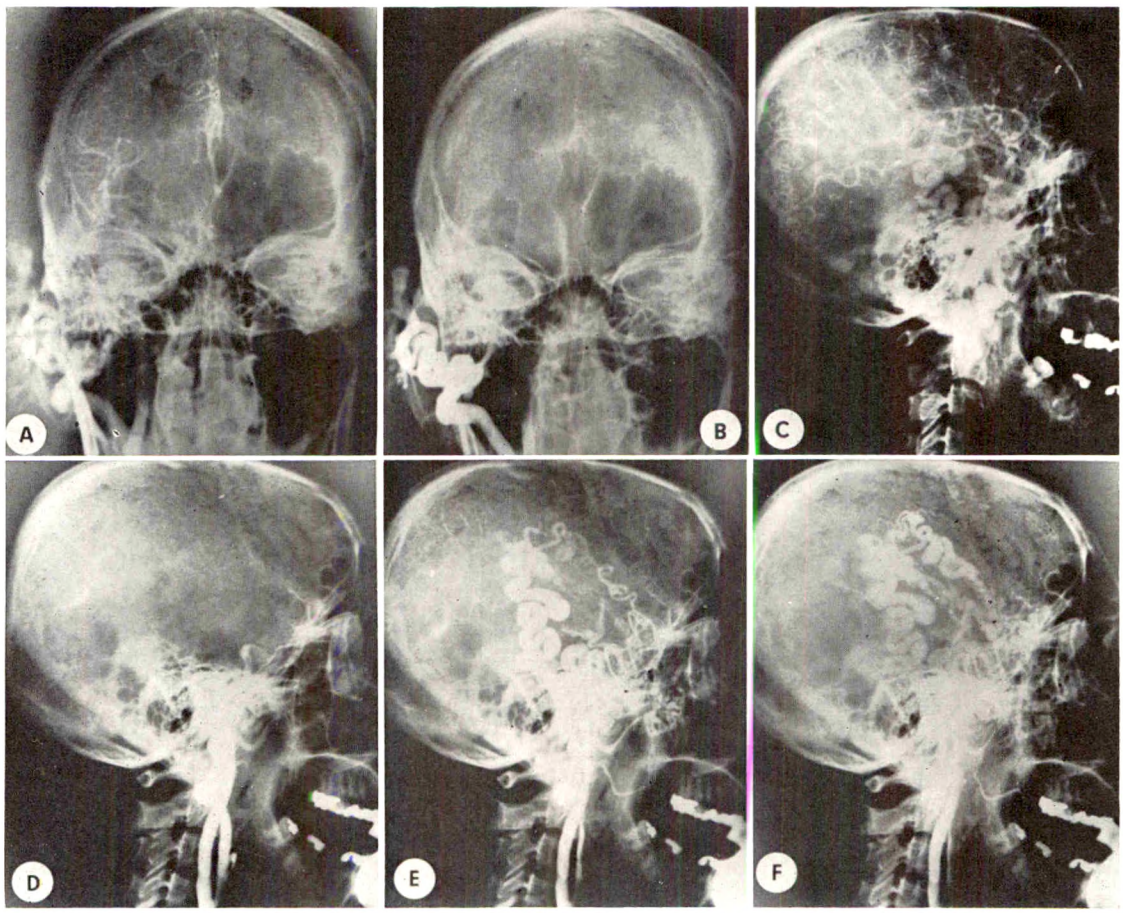


FIG. 2. (*A*) Anteroposterior and (*C*) profile routine angiograms. Injection of contrast medium into the common carotid artery. (*B*) Anteroposterior and (*D–F*) profile P.C. angiograms with injection of the contrast medium at the same site as in the routine angiograms. Note preferential filling of external carotid branches supplying the A.V.A.; angiographic subtraction of intracranial circulation. The interval between *D* and *E* is 1.5 seconds and between *E* and *F* is 60 seconds. The latter is a proof of great dependency of the A.V.A. on the feeding branches from the ipsilateral external carotid artery. Angiographic addition of arterial feeders and venous outflow in *F*.

unpredictable flow types have been observed with P.C. angiography, such as reversal of transport of contrast medium in the internal or external carotid arteries. The filling of some internal and/or external carotid branches is frequently suppressed. From a roentgenographic point of view this suppression may be called *angiographic subtraction*. In regard to the intracranial branches, the filling of the anterior or posterior cerebral arteries is frequently suppressed, and in some of the cases the contrast medium is completely arrested in the internal carotid artery.

On the other hand, P.C. angiography may produce *angiographic addition*, such as greater density of the vascular pattern or visualization of vessels not observed in the corresponding routine angiograms.

Although this unorthodox method of angiography produces "unnatural" effects, the latter proved to be of diagnostic and, in some instances, even therapeutic interest. Among the various conditions in which P.C. angiography has been found to have a useful application, the arteriovenous aneurysm (A.V.A.) occupies a special place deserving a separate discussion.

A general property of all P.C. angiograms, *i.e.*, greater density of the angiographic pattern, that of the smaller vessels included, may be very useful in the study of the A.V.A. Apart from this fact, the method produced interesting information with the following special forms of A.V.A.:

I. A.V.A. of the Scalp and Dural A.V.A. Supplied by Branches of the External Carotid Arteries

We have experience with 9 such cases: 5 A.V.A.s of the scalp; 1 dural A.V.A.; 2 combined forms, one localized in the scalp and the dura, and the other in the posterior cervical muscles and the basal dura of the posterior fossa; and 1 facial A.V.A.

P.C. angiography in this group revealed the following aspects:

(a) *Angiographic subtraction* (Fig. 1 and 2).

In 5 of the 9 cases, the contrast medium was arrested in the internal carotid artery so that the A.V.A. appeared free from a superimposed intracranial angiogram, while in the others the middle cerebral artery was faintly or only temporarily visualized. The same result can be achieved with selective external carotid angiography. P.C. angiography, however, is an easier procedure, since there is no need of moving the needle from the common carotid artery into the external carotid artery, or of selective puncture or catheterization of the latter. An absolute advantage of P.C. angiography over "routine" selective external carotid angiography is the changed dynamics of blood flow resulting in angiographic addition and demonstration of vascular dependency of the A.V.A., discussed in the following sections.

(b) *Angiographic addition* (Fig. 1 and 2).

Selective external carotid angiography in both cases of combined extracranial and dural A.V.A.s demonstrated only the extracranial shunting, whereas P.C. angiography revealed the presence of the dural A.V.A. as well. Other additional phenomena were denser opacification of the arterial feeders and demonstration of both the arterial feeders and venous effluents in one angiogram (frequently the 3rd angiogram). The latter phenomenon is related to monovascular dependency of the A.V.A. (see under c).

(c) *Vascular dependency of the A.V.A.*

In all cases bilateral carotid, and vertebral angiographies were performed. In 5 of 9 cases the A.V.A. appeared to be supplied by 1 external carotid artery only. The P.C. angiograms of these 5 patients demonstrated the same or more arterial feeders than the routine angiograms; the arterial feeders remained stained in all 3 successive P.C. angiograms, while these showed progressive filling of venous outflow. It was felt that these features may be signs of monovascular dependency (on the main trunk—the external carotid artery). In 1 of the patients, in whom the intracranial transport was suppressed in the P.C. angiograms, a repeat P.C. angiogram was made with a 60 second interval between the second and third angiogram taken. During this 1 minute interval, permanent filling of the A.V.A. was observed (Fig. 2, *E* and *F*).

These findings may have surgical implications as to the expected result of ligation of arterial feeders. In 3 of the 5 monovascular dependent A.V.A.s, the abnormal vascular mass, localized in the scalp, could be completely excised. The other 2 patients were treated by means of ligation of the arterial feeders. One of them, presenting with a dural A.V.A. supplied by the middle meningeal artery via the maxillary artery and suffering from exophthalmus and a pulsating bruit, was completely relieved from signs and symptoms following ligation of the ipsilateral external carotid and maxillary arteries. There were no signs of recurrence at a check-up 3.5 years after the operation. The other patient presenting with an A.V.A. of the scalp and the zygomatic area showed a complete collapse of the vascular mass following ligation of the external carotid, temporal, and occipital arteries. The internal maxillary artery could not be exposed without drastic

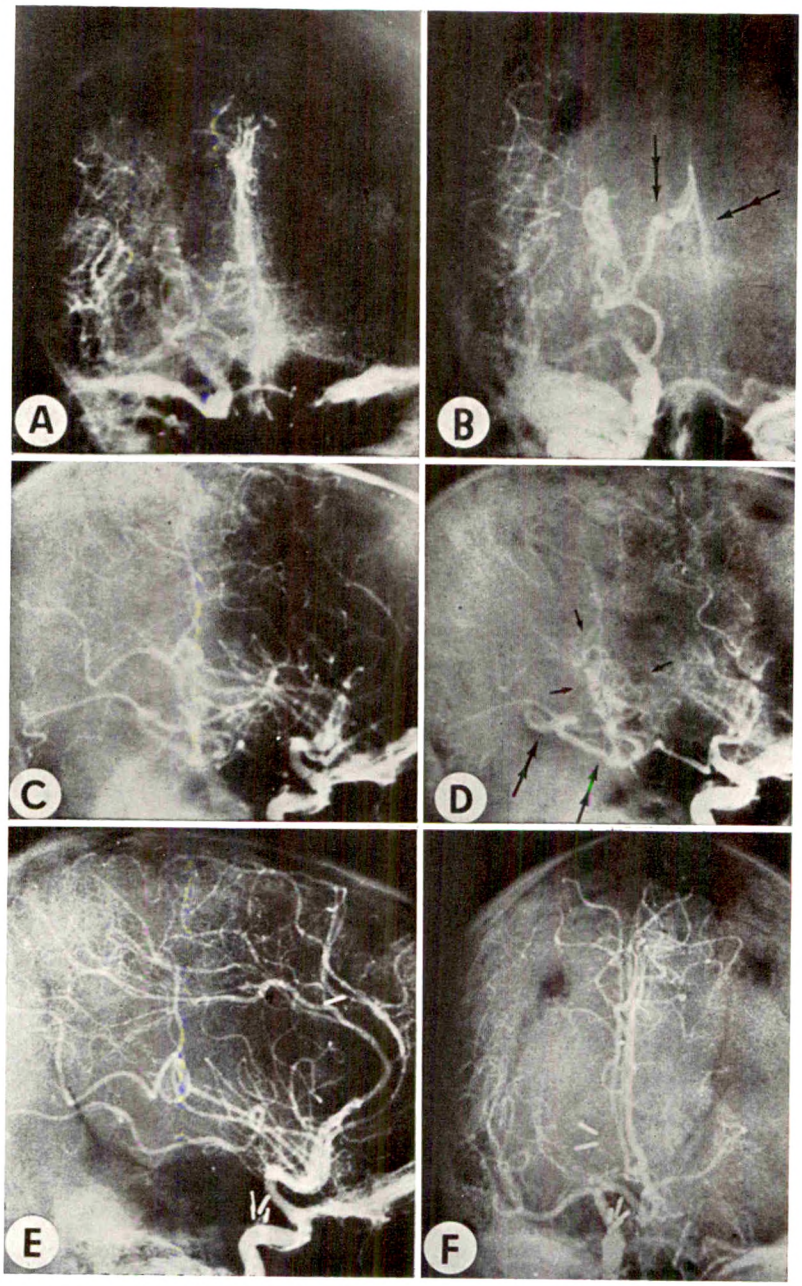


FIG. 3. A.V.A. of right basal ganglia supplied by anterior choroidal artery. (*A*) Anteroposterior routine angiogram. (*B*) Anteroposterior P.C. angiogram, which, because of angiographic subtraction of the anterior cerebral artery and its branches, demonstrates better visualization of A.V.A., the supplying anterior choroidal artery and the draining deep medial veins (arrows). (*C*) Profile routine and (*D*) profile P.C. angiograms. Arrows indicate A.V.A.; double arrows indicate draining vein. (*E* and *F*) Routine angiograms 2 years after clipping of anterior choroidal artery show exclusion of the A.V.A. In addition, *F* shows the extent of the craniotomy towards the opposite side used for transcallosal exploration of the floor of the right lateral ventricle before clipping of the artery. (*B–F* reproduced by permission of Butterworth & Co., Ltd.[16])

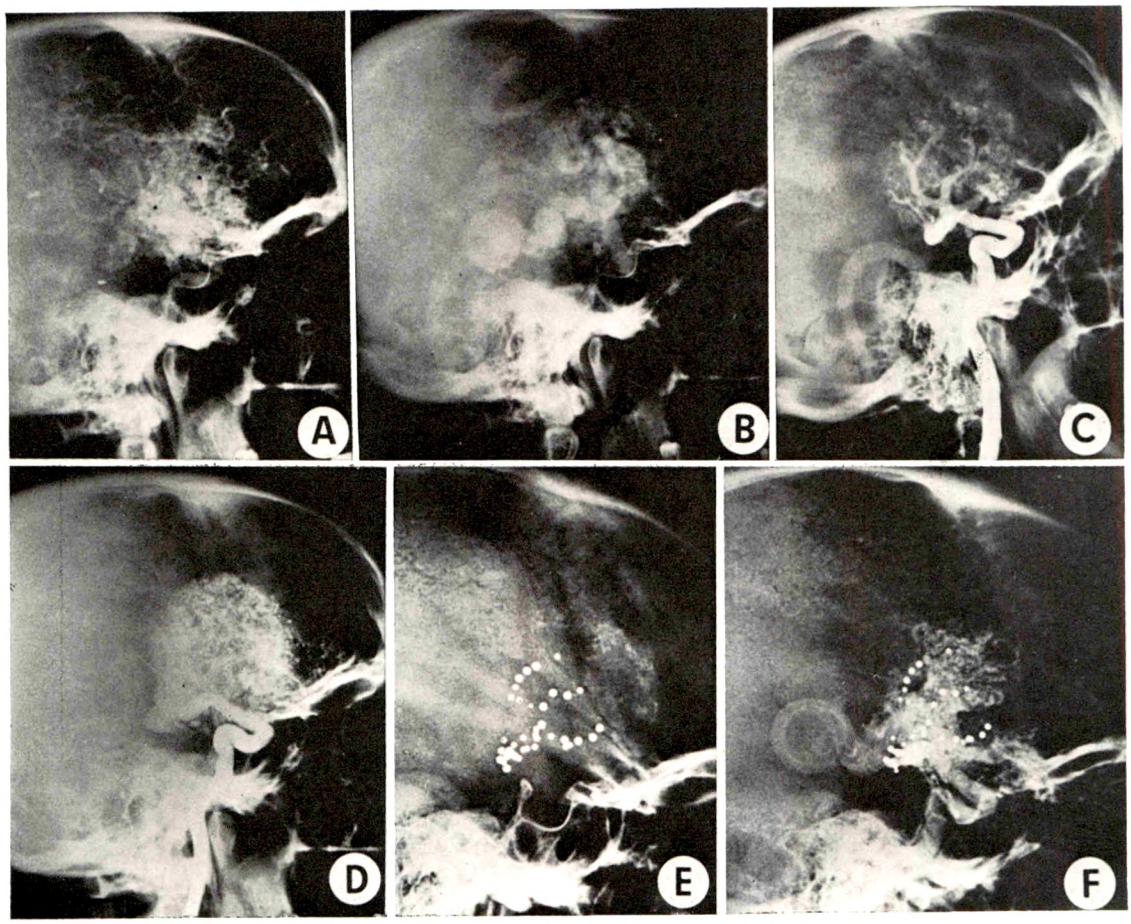


Fig. 4. A.V.A. supplied by the left middle cerebral artery. (*A* and *B*) Routine angiograms with 3 second interval. (*C* and *D*) P.C. angiograms with 3 second interval show, as compared to *A* and *B*, a much slower circulation. In addition, *C* shows heavily stained feeders inside A.V.A., allowing measurements of their diameters for artificial embolization. (*E*) Embolization of the principal arterial branches (see *C*) of the A.V.A. (*F*) Angiogram after embolization.

surgery and this vessel was therefore not ligated. The operation resulted in a collapse of the A.V.A. and disappearance of the bruit. Two years later there was only a small recurrence of the A.V.A. in the zygomatic area, which appeared to be due to an anastomosis between a dilated branch of the vertebral artery at the C 1–2 level with the maxillary artery not visualized in the previous angiograms. Following embolization and ligation of the anastomosis, the swelling in the zygomatic area collapsed. There has been no recurrence of the A.V.A. in the temporal area. This experience shows that even in the presence of monovascular dependency, the feeding branches should be obliterated as close to the A.V.A. as possible.

The P.C. angiograms of the other group of 4 patients, in whom the A.V.A. was supplied by both external carotid arteries, showed a reduced number of feeders of the A.V.A. as compared with the routine angiograms, while the successive P.C. angiograms showed signs of washing-out of the contrast medium. In some of the cases addition phenomena, such as denser arterial straining or visualization of details of venous outflow not demonstrated in the routine angiograms, were noted at the same time.

Our experiences with P.C. angiography

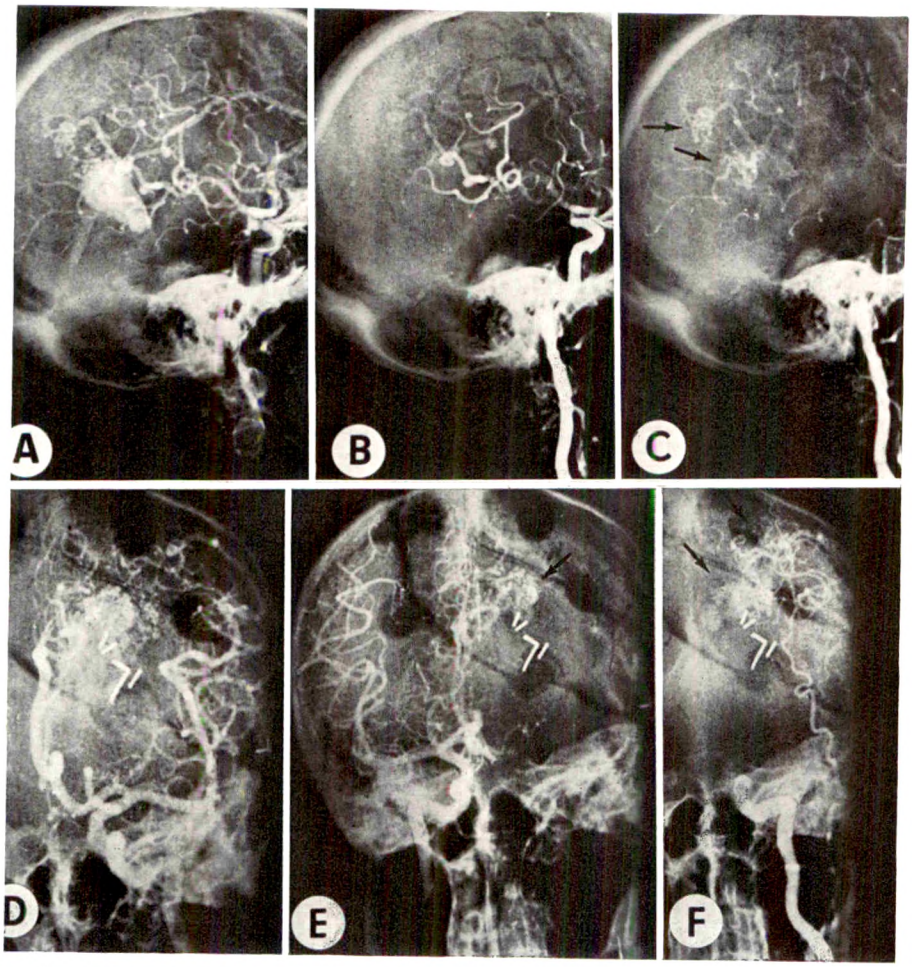


FIG. 5. Example of estimation of vascular dependency of *A.V.A. in the borderline supply areas* of the posterior cerebral artery and branches of the internal carotid artery. (*A*) Profile and (*D*) anteroposterior routine left carotid angiograms show location of A.V.A. (*B* and *C*) P.C. angiograms show suppressed filling of the anterior cerebral artery and its branches; the middle cerebral artery contributes to the supply (arrows in *C*) of the A.V.A. (see also *F*). (*E*) Anteroposterior right carotid angiogram, obtained during compression of the left carotid artery in the neck, shows supply of A.V.A. by left anterior cerebral artery (arrow). (*F*) Anteroposterior left P.C. carotid angiogram shows supply of A.V.A. by middle cerebral artery. (Angiograms *D* through *F* were made after surgical evacuation of the hematoma. The vertebral angiograms, not shown here, revealed supply of the A.V.A. by the left posterior cerebral artery.)

in this group of A.V.A.s induced us to extend the investigation of vascular dependency to A.V.A.s supplied by the internal carotid artery.

II. THE USE OF P.C. ANGIOGRAPHY FOR THE ESTIMATION OF VASCULAR DEPENDENCY OF INTRACRANIAL A.V.A. SUPPLIED BY THE INTERNAL CAROTID ARTERY

The one-artery dependent A.V.A.s in the supply area of the internal carotid artery, in which P.C. angiography may be a useful diagnostic aid, are those whose feeders are either the cavernous branches of the internal carotid artery or the anterior choroidal or middle cerebral arteries.

We had the following experiences with the practical value of P.C. angiography:

(a) *P.C. angiography as an aid for the indication of direct ligation of an arterial feeder surgically nonexcisable A.V.A.*

A one-artery dependent A.V.A. supplied

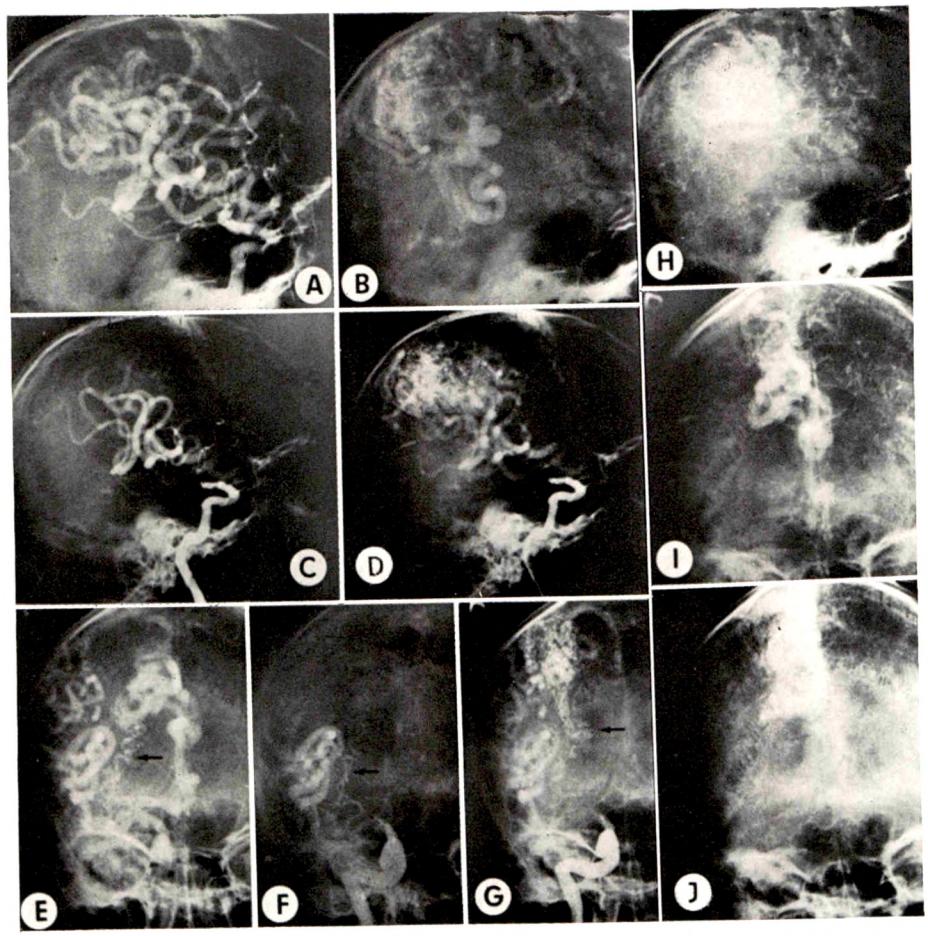


Fig. 6. Example of estimation of vascular dependency of *large A.V.A.* of right cerebral hemisphere. (*A* and *B*) Profile right routine carotid angiograms. Large and tortuous anterior and middle cerebral arteries in *A* and area of shunting in *B*. (*C* and *D*) Profile right P.C. carotid angiograms show suppressed filling of the right anterior cerebral artery. The area of the A.V.A. supplied by the middle cerebral artery is demonstrated in *D*. (*E*) Anteroposterior right routine carotid angiogram also shows participation of the anterior choroidal artery (arrow). (*F* and *G*) Right P.C. carotid angiograms. Suppressed filling of right anterior cerebral artery. The area of the A.V.A. supplied by the middle cerebral artery and the one supplied by the anterior choroidal artery (arrow) are demonstrated in *G*. (*H–J*) Left routine carotid angiograms during compression of the right carotid artery in the neck. (*I*) Poor filling of the left intracranial circulation. The right anterior cerebral artery and its branches are well filled. The area of the A.V.A. supplied by the right anterior cerebral artery is demonstrated 1.5 seconds after *I* in the profile roentgenogram *H* and anteroposterior roentgenogram *J*. (Vertebral angiography, not presented here, revealed supply of the A.V.A. by the right posterior cerebral artery.)

by cavernous branches of the internal carotid artery is presented in Figure 8.

The patient, a male 45 years of age, had his first subarachnoid hemorrhage in 1968 accompanied by temporary left hemiplegia and diplopia, followed by complete deafness of the right ear. A second hemorrhage occurred on March 24, 1970 and a third on April 14, 1970. The last hemorrhage was followed by coma for several days. The patient now complained of a pulsating bruit. A routine right internal carotid angiogram showed cavernous branches of the carotid artery and there was a faint indication of their being involved in an arteriovenous shunt. The left carotid and the vertebral angiograms showed no abnormalities.

Right 3 second P.C. angiography with puncture of the internal carotid artery showed arterial feeders arising from the intracavernous

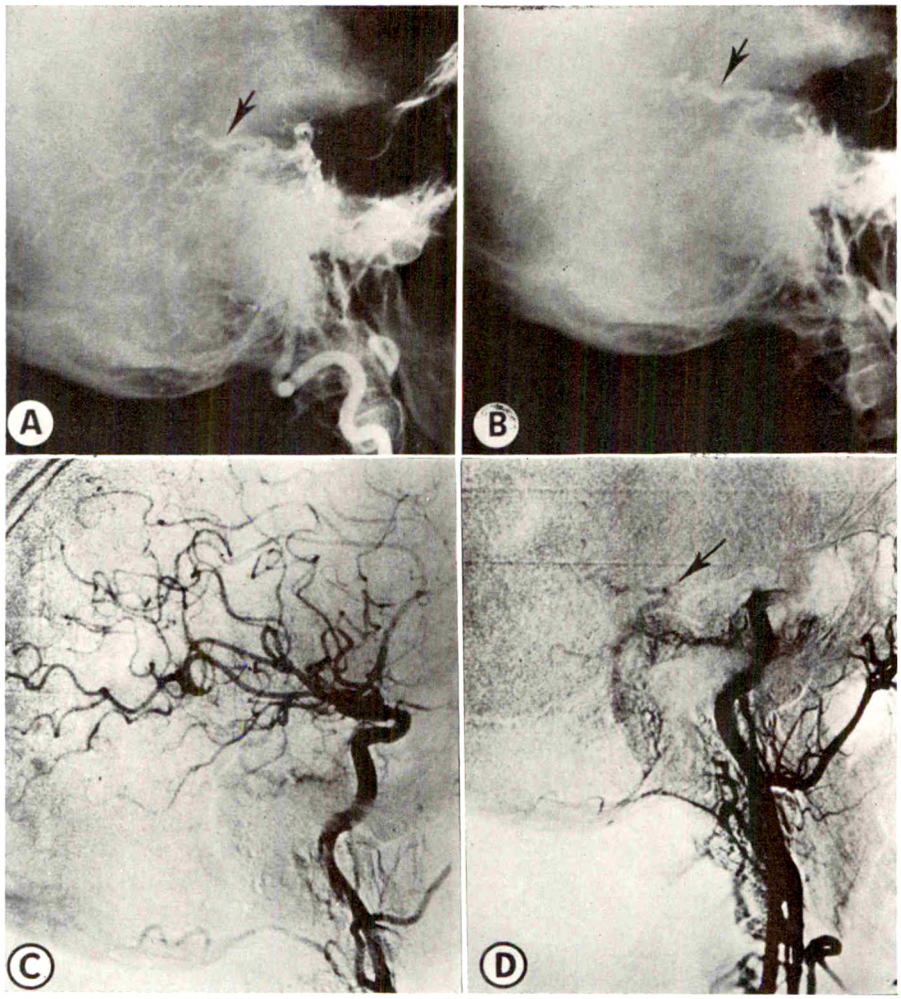


Fig. 7. (*A* and *B*) Vertebral angiograms show A.V.A. localized in the interpeduncular cistern, the petrosal apex and the dura covering these structures (verified at operation). (*C*) Subtraction image of routine right carotid angiogram shows faint indication of A.V.A. (*D*) Subtraction image of P.C. right carotid angiogram shows angiographic subtraction of intracranial circulation with exception of the cavernous branches of the internal carotid artery supplying the A.V.A. (arrow). The more complete and denser visualization of these branches is an example of angiographic addition in the internal carotid angiogram.

portion of the internal carotid artery and extensive venous effluents of the A.V.A. The transport of the contrast medium was very slow. It was the picture of a dural A.V.A. of the posterior fossa. The internal carotid artery was obliterated, using muscle pieces. This method, described by the author elsewhere,[16] resulted in occlusion of the cavernous branches of the A.V.A.

On check-up 2 years after the arterial occlusion, the intracranial bruit had disappeared and there had been no recurrence of subarachnoid hemorrhage.

A one-artery dependent A.V.A. supplied by the anterior choroidal artery is presented in Figure 3.

The patient, a girl 17 years of age, had a subarachnoid hemorrhage accompanied by coma and decerebrate rigidity. She recovered with residual left hemiplegia. The right carotid angiogram showed an A.V.A. supplied by the anterior choroidal artery. The left carotid and vertebral angiograms were normal.

Right P.C. carotid angiography resulted in better visualization of the malformation be-

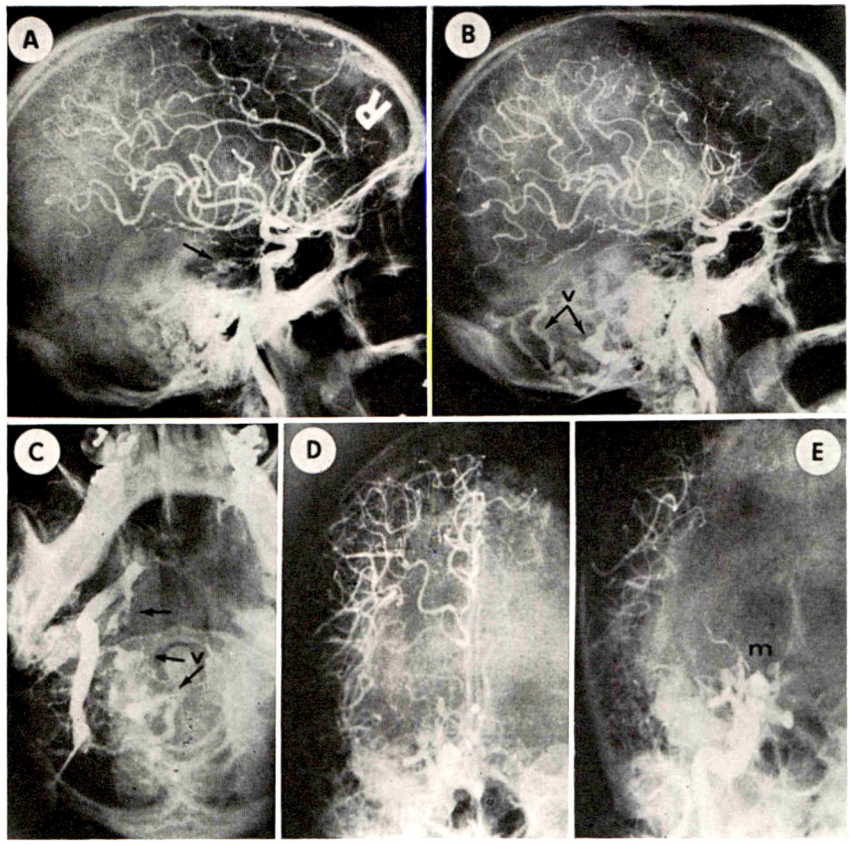


Fig. 8. Dural A.V.A. of posterior fossa supplied by cavernous branches of right internal carotid artery. (*A*) Profile and (*D*) anteroposterior routine right internal carotid angiograms show only small part of A.V.A. (arrow). (*B*) Profile, (*C*) basal, and (*E*) anteroposterior right P.C. carotid angiograms. *B* shows angiographic addition phenomena; *i.e.*, visualization of the arterial and venous (v arrows) components of A.V.A. during the arterial phase. In none of the routine angiograms was the venous drainage well visualized. *C*, basal view of P.C. angiogram, shows heavily stained cavernous branches (arrow) of the internal carotid artery and an extensive pattern of venous outflow (v arrows). In *E*, the drainage of the A.V.A. (m) into the deep veins is well visualized because of angiographic subtraction of the anterior cerebral artery (compare with the routine anteroposterior angiogram in D). (*A* and *C–E* reproduced by permission of Butterworth & Co., Ltd.[16])

cause of suppressed staining of the anterior cerebral artery. The arterial and venous components of the malformation were stained during 3 second P.C. angiography. These findings supported the diagnosis of an A.V.A. entirely dependent on the anterior choroidal artery. Therefore, this artery was exposed and clipped, after a transcallosal exploration of the lateral ventricle had not revealed any surface projection of the A.V.A.

Complete angiography 2 years later showed that the A.V.A. was completely excluded.

Comment. Clipping or ligation of feeding arteries of A.V.A.s rarely gives satisfactory results. Olivecrona and Ladenheim[11] stated that when angiography indicates that a single artery provides the arterial supply of the lesion, that artery may be ligated. But the authors considered angiography not infallible, since they had at times noted the presence of contributing vessels which had not been demonstrated in the angiograms. However, a few cases have been reported,[3,12] which improved after direct ligation of feeding arteries, although there were no reliable diagnostic means to predict the outcome of such ligations.

As far as A.V.A.s in the supply area of

the external or internal carotid arteries are concerned, P.C. angiography may sometimes demonstrate the presence of an A.V.A. dependent on 1 of these arteries or even on 1 of their branches. If such lesions are considered surgically nonexcisable, there is then reason to perform ligation of the nutrient vessels. This is demonstrated by the good clinical results in 2 cases of A.V.A.s in the supply areas of the external carotid (Section 1 c) and 2 others supplied by branches of the internal carotid arteries. In 1 of the latter the complete exclusion was demonstrated angiographically 2 years after ligation.

(b) *The value of P.C. angiography in the case of embolization of an A.V.A. supplied by branches from the proximal portion of the left middle cerebral artery.*

The patient, a male 34 years of age, suffered from progressive hemiparesis, aphasia and epileptic seizures.

The left carotid routine angiograms (Fig. 4) demonstrated a large A.V.A., situated on the proximal portion of the middle cerebral artery. P.C. angiography showed the great dependence of the A.V.A. on the middle cerebral artery resulting in slow circulation with considerable prolongation of the arterial phase, while the principal feeding branches, not distinguishable in the routine angiograms, were clearly visualized and heavily stained in the early P.C. angiogram. This allowed measurement of the required sizes of the emboli and demonstrated their successful placement.

(c) *The combination of P.C. angiography and other vascular compression tests for the evaluation of the number of feeding main arteries in supratentorial A.V.A. of the brain.*

This evaluation is of importance for the estimation of the operability or the surgical strategy in very large A.V.A.s and A.V.A.s situated in borderline supply areas of the posterior cerebral and branches of the internal carotid arteries. In such cases vertebral angiography shows the participation of the posterior cerebral artery. If the filling of the anterior cerebral artery is suppressed in the P.C. carotid angiograms, distal feeders of the middle cerebral artery may be visualized. Contralateral angiography with compression of the internal carotid at the site of the A.V.A. may show the supply by the anterior cerebral artery. Figure 5 shows the effect of this method with a borderline A.V.A., while Figure 6 demonstrates similar findings with a large A.V.A. Identical findings were obtained with another A.V.A. supplied by the anterior, middle and posterior cerebral arteries. Recently this method also proved useful in a case of A.V.A. of the pole of the right frontal lobe. Right P.C. carotid angiography showed suppressed filling of the anterior cerebral artery and supply of the A.V.A. by the orbito-frontal branches of the middle cerebral artery. Left carotid angiography with compression of the right carotid artery in the neck revealed crossed contrast medium filling of the right anterior cerebral artery, while the right middle cerebral artery was not visualized. This time the A.V.A. appeared to be contrast-filled through the orbital rami of the anterior cerebral artery.

The diagnostic value of P.C. angiography with this group of patients is limited to the demonstrated feeders from the middle cerebral artery. Their absence in the P.C. angiograms is no proof that such feeders do not exist. This may particularly be valid with A.V.A.s of the occipital lobe. These lesions may be supplied chiefly from the posterior cerebral artery system or from this and the distal portions of the middle and/or anterior cerebral artery systems.[4]

We have had experience with 3 A.V.A.s in this location. In 2 of them, localized in the medial portions of the occipital lobes, ipsilateral P.C. angiography and contralateral angiography during compression of the ipsilateral carotid artery did not demonstrate any feeders originating from the middle or anterior cerebral arteries, and there was not much evidence of such feeders at the surgical removal of the A.V.A.s.

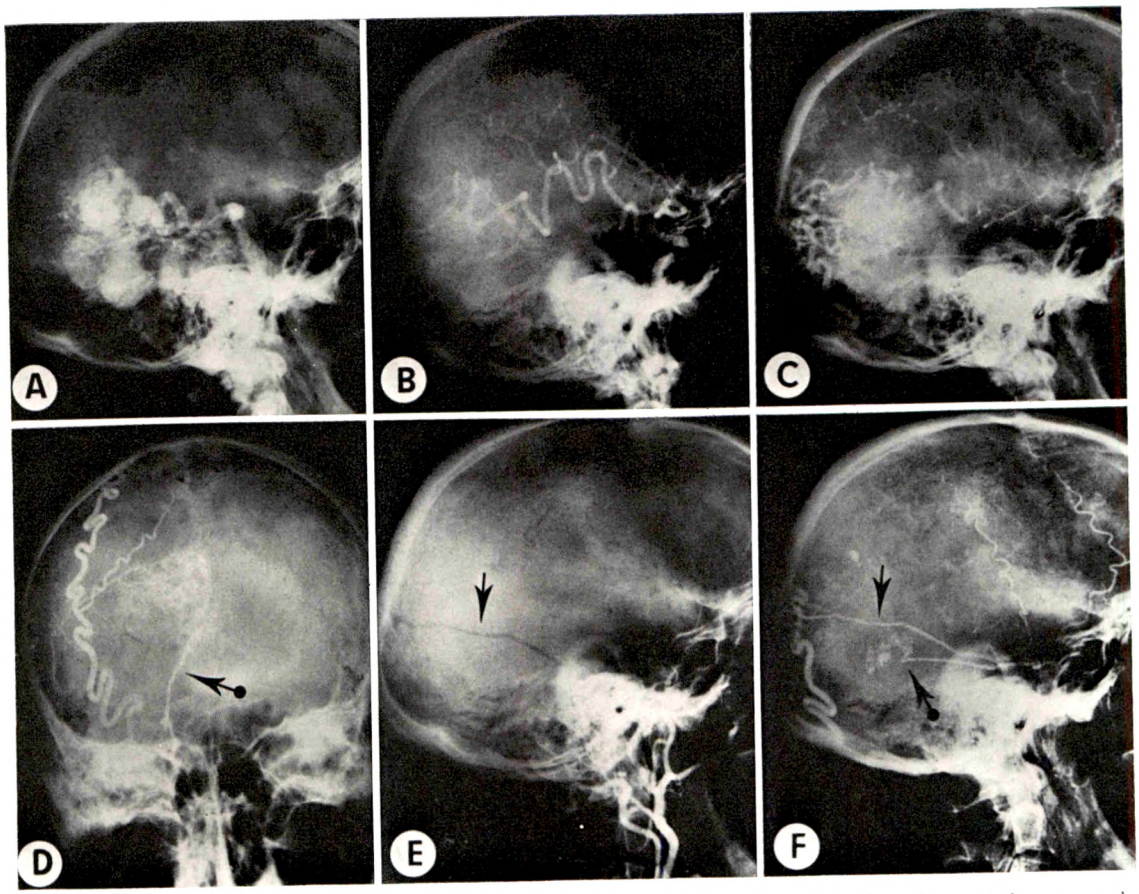


Fig. 9. A.V.A. of right occipital lobe, combined with dural A.V.A. (*A*) Vertebral angiogram shows supply of A.V.A. by right posterior cerebral artery. (*B* and *C*) Routine right carotid angiograms with injection of contrast medium into the common carotid artery show supply by the occipital artery. A tentorial artery is visualized. The posterior temporal branch of the middle cerebral artery appears to participate in the supply of the A.V.A. (*D–F*) Right P.C. carotid angiograms show angiographic subtraction of intracranial circulation with the exception of the tentorial artery. (*D*) Anteroposterior view shows the tentorial artery (arrow) and its branching inside the tentorium. The latter is an angiographic addition phenomenon not seen in the routine angiogram. Note the supply by the occipital and middle meningeal arteries. (*E*) First profile P.C. angiogram. Arrow points to the enlarged groove of the middle meningeal artery. The contrast medium has not yet entered into the intracranial vessels. (*F*) Profile view 3 seconds after *E*. Angiographic addition phenomena: branching of the tentorial artery inside the tentorium (point end arrow); and in *F* visualization of the middle meningeal artery (straight arrow) not seen in the routine angiograms.

The third case, however, not operated on because of the complexity of the lesion, clearly showed that absence of feeders in the P.C. angiograms is no proof that such feeders do not exist. The routine angiograms revealed that the malformation was supplied by the middle cerebral artery, whereas the filling of this artery was suppressed in the P.C. angiogram (Fig. 9).

This A.V.A. was clearly visualized in the vertebral angiograms. It extended more laterally than in the previous 2 cases. The routine carotid angiograms showed supply by the middle cerebral artery. Besides, a tentorial artery was stained and at its distal end a faint small abnormal vascular pattern was visualized. The external carotid artery participated by means of the occipital artery in the supply of the A.V.A.

P.C. angiography showed suppression of the middle and anterior cerebral arteries, but the

tentorial artery was well stained and appeared to supply a large tentorial A.V.A.

Of the external carotid artery circulation, the supplying occipital artery was heavily stained and the posterior branch of the middle meningeal artery, not demonstrated in the routine angiograms, also appeared to supply the A.V.A. (an addition phenomenon).

III. ANGIOGRAPHIC SUBTRACTION AND/OR ADDITION PHENOMENA WITH P.C. ANGIOGRAPHY OF INTRACRANIAL A.V.A.

(a) *Angiographic subtraction.*

In contrast to the well known methods of photographic or electronic subtraction, the subtraction of vessels in the P.C. angiogram, because of suppressed filling of certain vessels, is the consequence of the angiographic technique itself. Most frequently the suppressed filling involves the anterior and posterior cerebral arteries, but in some instances the filling of the middle cerebral artery is suppressed as well. This angiographic subtraction results in better visualization of abnormal patterns in the residual vessels in the angiogram. Examples of angiographic subtraction are given in Figures 3, 8 and 9.

The photographic or electronic subtraction also finds a place in P.C. angiography of A.V.A.s (Fig. 7). Immobilization of the head, essential condition for photographic subtraction of the intracranial angiogram, is especially difficult during P.C. angiography because the digital compression of the common carotid artery may induce a change in the position of the head.

(b) *Angiographic addition.*

The following phenomena of angiographic addition were observed with P.C. angiography:

1. Demonstration of *arterial feeders* not observed in the routine common, internal or external carotid angiograms. Of our material of over 380 P.C. angiographies, this addition phenomenon was only observed with A.V.A.s and meningiomas. In both conditions the addition was limited to dural arterial feeders. This addition phenomenon in cases of A.V.A. is demonstrated in Figures 7 and 9. The information thus obtained is invaluable for the determination of the operability of the lesion and the outlining of the surgical strategy.

2. Addition phenomena regarding the *venous outflow* of an A.V.A. These phenomena may be classified in 2 groups: Firstly, P.C. angiography may result in a more extensive visualization of the A.V.A. than occurred with routine angiography. We have observed this phenomenon in only 2 cases of dural A.V.A. (Fig. 8), and it was not noted in our cases of intracerebral A.V.A.s; and secondly, features of venous outflow demonstrated in successive routine angiograms may, using P.C. angiography, be observed on one film together with the arterial supply (Fig. 8). The phenomena of addition and subtraction may both appear in the same angiogram, resulting in better visualization of the addition phenomenon.

GENERAL REMARKS

In over 380 P.C. angiographies we have been confronted with a certain unpredictability of the distribution of the contrast medium. This is, however, compensated by other angiographic findings. The meningiomas and A.V.A.s have a special place in our series of P.C. angiographies, since in the great majority of meningiomas, P.C. angiography made a valuable contribution to the diagnosis, while in 20 out of 22 A.V.A.s examined up to the present, P.C. angiography facilitated the diagnosis or supplied valuable information which could not have been obtained by the routine methods of angiography. More than with other neurosurgical lesions, the P.C. angiographic examination of A.V.A.s opens new perspectives concerning the hemodynamics involved, which encourages further investigation on a larger scale.

SUMMARY

Our experience with artificial slow flow carotid proximal compression angiography (P.C. angiography) in cases of A.V.A.s in

the supply area of the internal and/or external carotid arteries is described.

The method consists of percutaneous puncture of the common carotid artery with digital compression of the artery proximal to the site of injection.

Following a description of technical details, more extensively reported in a previous paper, attention is paid to the specific properties of P.C. angiography in A.V.A.s, such as: (a) preferential staining of A.V.A.s of the scalp and/or the dura mater in the supply area of the external carotid artery; (b) *angiographic subtraction* due to suppressed filling of certain intracranial arteries, which may result in better visualization of the malformation; and (c) *angiographic addition*, consisting of increased density in staining, more extensive staining of the arteriovenous flow pattern than visualized in the routine angiograms, or addition of flow features demonstrated in successive routine angiograms in 1 of the P.C. angiograms, and finally demonstration of dural feeders not visualized in the routine angiogram.

The use of P.C. angiography for the evaluation of the vascular dependency of extra- and intracranial A.V.A.s is also discussed.

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REFERENCES

1. Decker, K., and Backmund, H. Angiography of Cerebral Circulation. Georg Thieme Verlag, Stuttgart, 1968.
2. Ecker, A., and Riemenschneider, P. Deliberate thrombosis of intracranial arterial aneurysms by partial occlusion of carotid artery with arteriographic control: preliminary report of a case. *J. Neurosurg.*, 1951, *8*, 348–353.
3. French, L. A., and Chou, S. N. Conventional methods of treating intracranial arteriovenous malformations. *Progress in Neurol. Surg.*, 1969, *3*, 275–316.
4. Gass, H. The Importance of the Posterior Communicating Artery in the Treatment of Posterior Supratentorial Arteriovenous Malformations of the Brain. Abstracts. Fourth Europ. Congr. Neurosurg., 1971, n-94. Avicenum Czechoslovak Medical Press.
5. Gibbs, J. R. Effects of carotid ligation on size of internal carotid aneurysms. *J. Neurol., Neurosurg. & Psychiat.*, 1965, *28*, 383–394.
6. Harris, P., and Udvarhelyi, G. B. Aneurysms arising at internal carotid-posterior communicating artery junction. *J. Neurosurg.*, 1957, *14*, 180–191.
7. Leeds, N. E., and Taveras, J. M. Dynamic Factors in Diagnosis of Supratentorial Brain Tumors. W. B. Saunders Company, Philadelphia, 1969.
8. Luessenhop, A. M., Mora, F., and Sweet, W. H. Subarachnoid hemorrhage, intracranial aneurysms and arteriovenous anomalies; treatment by cervical carotid occlusion. In: Pathogenesis and Treatment of Cerebrovascular Disease. Edited by W. S. Fields. Charles C Thomas, Publisher, Springfield, Ill., 1961, pp. 516–533.
9. Mount, L. A., and Taveras, J. M. Results of surgical treatment of intracranial aneurysms as demonstrated by progress arteriography. *J. Neurosurg.*, 1956, *13*, 618–626.
10. Odom, G. L., Woodhall, B., Tindall, G. T., and Jackson, J. R. Changes in distal intravascular pressure and size of intracranial aneurysm following common carotid ligation. *J. Neurosurg.*, 1962, *19*, 41–50.
11. Olivecrona, H., and Ladenheim, J. Congenital Arteriovenous Aneurysms of the Carotid and Vertebral Arterial Systems. Springer-Verlag, Berlin, 1957.
12. Pool, J. L., and Potts, D. G. Aneurysms and Arteriovenous Anomalies of the Brain. Paul B. Hoeber, Inc., New York, 1965.
13. Taveras, J. M., and Wood, E. H. Diagnostic Neuroradiology. Williams & Wilkins, Baltimore, 1964.
14. Tindall, G. T., Goree, J. A., Lee, J. F., and Odom, G. L. Effect of common carotid ligation on size of internal carotid aneurysms and distal intracarotid and retinal artery pressures. *J. Neurosurg.*, 1966, *25*, 503–511.
15. Verbiest, H., and Dokkum, Th.A. Artificial slow flow carotid angiography with compression proximal to site of injection. *J. Neurosurg.*, 1970, *32*, 180–196.
16. Verbiest, H. Arterio-Venous Malformations. In: Operative Surgery. Second edition. Neurosurgery. Butterworth & Co., Ltd., London, 1971, pp. 130–141.
17. Verbiest, H. Arteriovenous Aneurysms of the Posterior Fossa. In: Cerebral Circulation. Edited by W. Luyendijk. Progress in Brain Research. Elsevier Press, Inc., Amsterdam, 1968, *30*, 383–396.

PSEUDOXANTHOMA ELASTICUM ASSOCIATED WITH CAROTID RETE MIRABILE*

CASE REPORT

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PSEUDOXANTHOMA elasticum (PXE) is an uncommon disease causing diffuse vascular involvement in young patients. The pathogenesis and clinical manifestations, as well as the varied roentgenographic findings have been reported previously.[5] The vascular changes of visceral organs associated with this condition have also been documented.[1] We have recently studied a patient with PXE who had bilateral carotid and vertebral rete mirabile with unilateral carotid-cavernous arteriovenous fistula and a vascular malformation in the spleen. Such an association, as far as we know, has not been reported heretofore.

REPORT OF A CASE

A 20 year old carpenter was admitted to the University of California Medical Center for evaluation of a protruded red right eye of 2 months' duration. The patient had been relatively asymptomatic until 1 week before admission when the protrusion increased rapidly and was associated with decreased visual acuity. He denied double vision, pain, or a subjective bruit.

Physical examination revealed the blood pressure to be 140/90 mm. Hg, pulse 88 per minute, and temperature 37.8°C. The right eye was protruded moderately (23 mm. O D, 17 mm. O S on the Hertel scale) and the conjunctival and episcleral veins were dilated. Visual acuity was decreased in the involved eye and a loud bruit was heard directly over the eyeball. The retinal veins in the right eye were slightly enlarged and tortuous, and hemorrhages involved the macula. "Angioid streaks" were also noted in both fundi, more marked in the right eye. Symmetric plaques of papular, reticulated yellowish lesions were noted on the lateral aspects of the neck and both axillae. The results of the neurologic examination were normal. Peripheral pulses were normal. Laboratory data included hemoglobin of 15.7 gm. per cent; hematocrit of 47 per cent; normal urinalysis, coagulation studies and electrolyte series; and serum cholesterol of 170 mg. per cent.

A biopsy specimen of the skin of the left neck area was diagnosed as pseudoxanthoma elasticum. Bilateral external and internal carotid, vertebral, and thyrocervical angiograms were obtained the following day (Fig. 1–6). Right internal carotid arteriograms (Fig. 1, *A* and *B*) showed a carotid-cavernous sinus arteriovenous fistula. The contrast medium was shunted predominantly into the superior ophthalmic vein as well as into the petrosal and jugular veins posteriorly. No filling of the distal supraclinoid internal carotid artery was present. The precavernous portion of the internal carotid artery was composed of several small branches (rete mirabile). Right external carotid arteriograms (Fig. 2, *A* and *B*) showed enlargement of the internal maxillary artery. An arterial network (rete mirabile), which arose from the internal maxillary artery, supplied the carotid siphon and its intracranial branches. A rete mirabile arising from the internal (Fig. 3, *A* and *B*) as well as from the external (Fig. 4, *A* and *B*) carotid arteries could also be seen clearly on the left side. The ascending cervical (Fig. 5) and deep cervical arteries (Fig. 6) were enlarged on the right side and gave rise to anastomotic vessels that supplied the distal right vertebral artery. A similar network between the cervical branches of the subclavian artery and the distal vertebral artery was also noted on the left. The proximal vertebral arteries were hypoplastic bilaterally.

Celiac angiograms showed a similar vascular anomaly in which the distal portion of the splenic artery gave rise to a network of vessels

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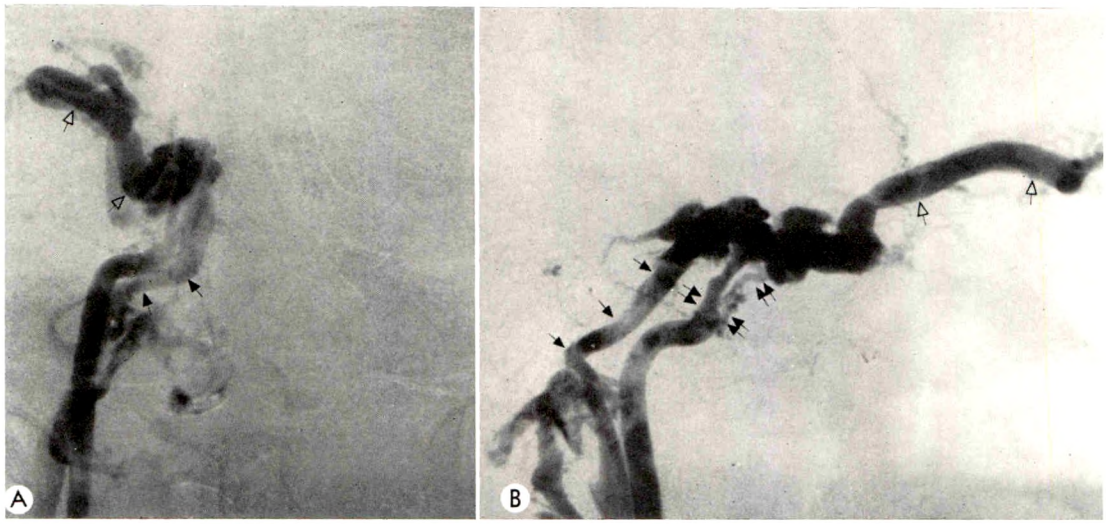


Fig. 1. Right internal carotid arteriograms. (*A*) Anteroposterior and (*B*) lateral views show carotid-cavernous sinus fistula, with drainage of contrast medium anteriorly into the superior ophthalmic vein (open arrows). Posteriorly the contrast medium is shunted into the inferior petrosal sinus (solid arrows) and internal jugular vein. There is no filling of the internal carotid artery distal to the cavernous sinus. The precavernous portion of the internal carotid artery is hypoplastic and is composed of several small arterial channels (double arrows).

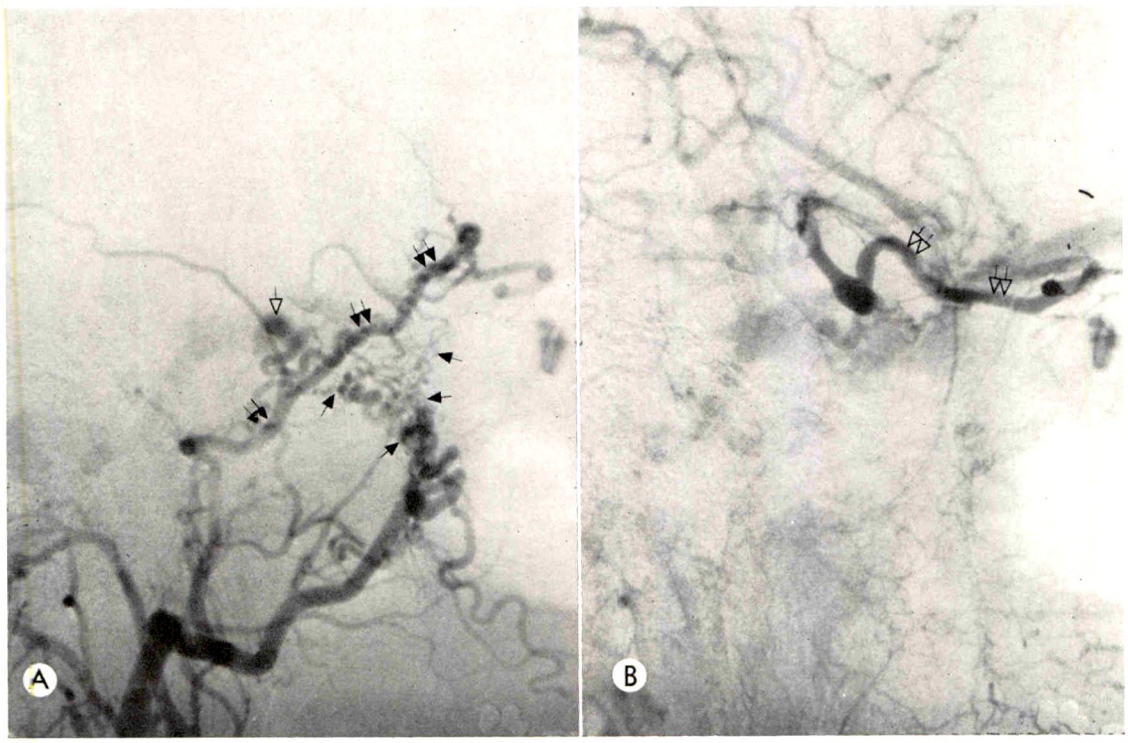


Fig. 2. Right external carotid arteriograms, lateral projection. (*A*) Early and (*B*) late arterial phases. The internal maxillary artery is enlarged. It gives rise to multiple small arterial channels (rete mirabile) (solid arrows) which fill the carotid siphon (open arrow). The carotid siphon, however, is primarily filled from the ophthalmic artery (double open arrows) which is filled in a retrograde manner from an enlarged ascending pharyngeal artery (double closed arrows). The superior ophthalmic vein and inferior petrosal sinus are faintly opacified from the internal carotid siphon.

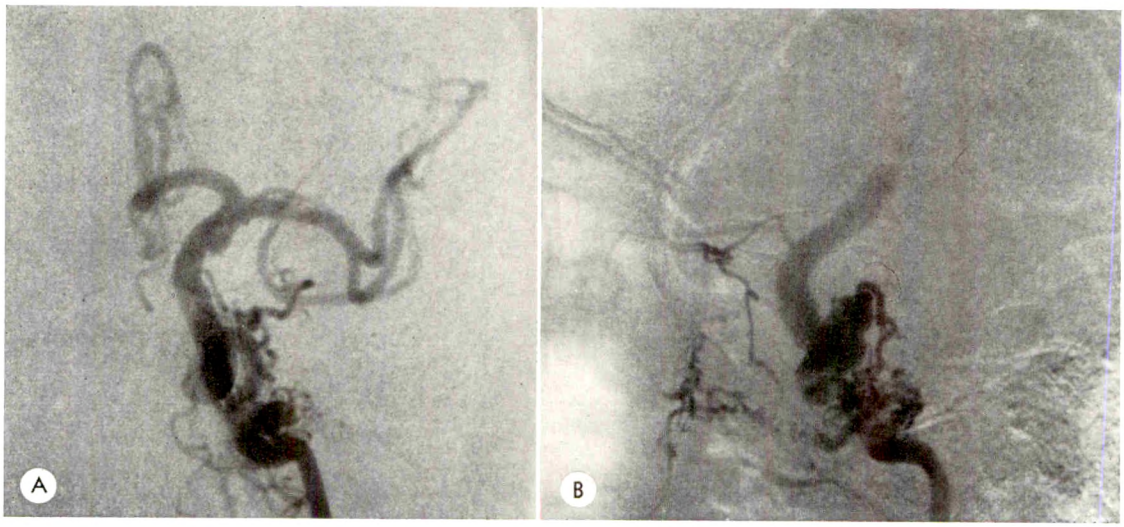


FIG. 3. Left internal carotid arteriograms. (*A*) Frontal and (*B*) lateral views. The precavernous portion of the internal carotid artery is replaced by multiple small branches (rete mirabile). These small branches reform a normal distal internal carotid artery. No arteriovenous shunting is seen.

that converged toward the hilum and supplied the spleen (Fig. 7, *A* and *B*).

An electrothrombosis of the cavernous sinus was performed in 2 stages. Anterior obliteration was achieved by placement of 3 copper wires through the dilated superior ophthalmic vein.

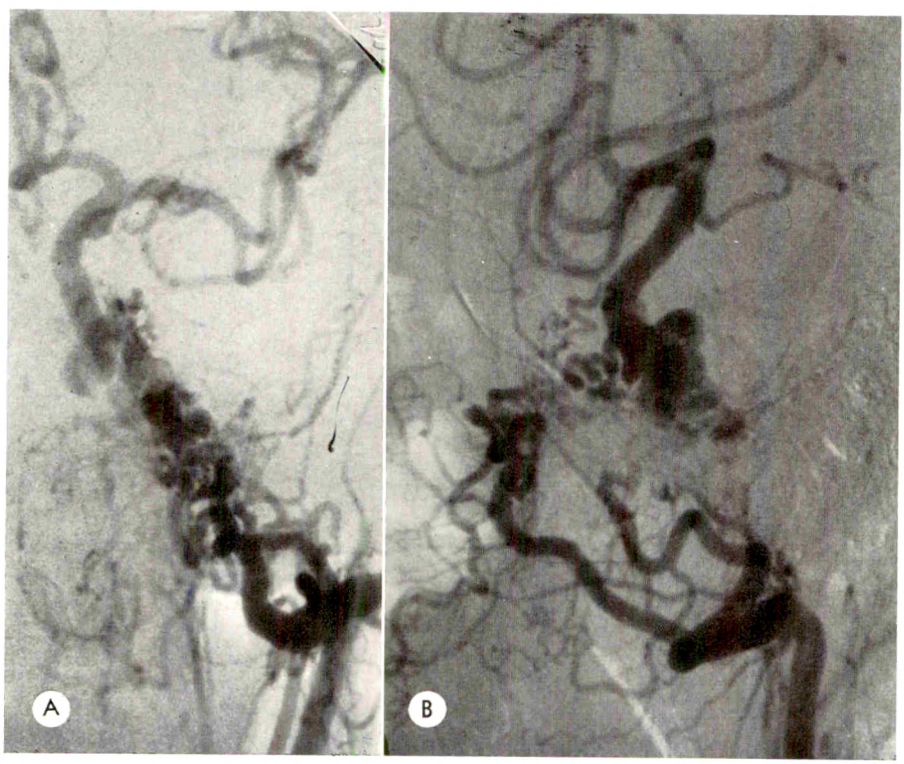


FIG. 4. Left external carotid arteriograms. (*A*) Anteroposterior and (*B*) lateral views. The enlarged internal maxillary and ascending pharyngeal arteries give rise to multiple arterial channels (rete mirabile) which fill the supraclinoid carotid artery.

The posterior cavernous sinus was approached through a craniotomy and the copper wires were placed directly into the cavernous sinus. The surgical procedure is described in detail by Hosobuchi.[4] Signs and symptoms referable to the arteriovenous fistula resolved completely after surgery. Postoperative angiography performed several weeks later showed obliteration of the carotid-cavernous fistula. The rete mirabile could now be seen more clearly (Fig. 8).

DISCUSSION

Pseudoxanthoma elasticum is a hereditary disorder of connective tissue. It is characterized by premature degeneration and calcification of elastic tissue primarily of the skin, eyes, and the cardiovascular system.[7] The granular, basophilic material with calcium deposits in the dermis and the angioid streaks of the retina surrounding the disk are characteristic of this disorder. The combination of retinal angioid streaks and calcification in the dermis is known as the Grönblad-Strandberg syndrome. The changes in the walls of medium-sized arteries, however, are indistinguishable from those found in atherosclerosis. Degeneration of elastic tissue has been seen in the aorta, in medium-sized vessels of the jejunum and ileum, and in the coronary artery.[14] Calcification is a secondary phenomenon in areas of elastic degeneration and destruction. Carlborg[2] demonstrated many patients with reduced peripheral pulses and symptoms of claudication. Some of his

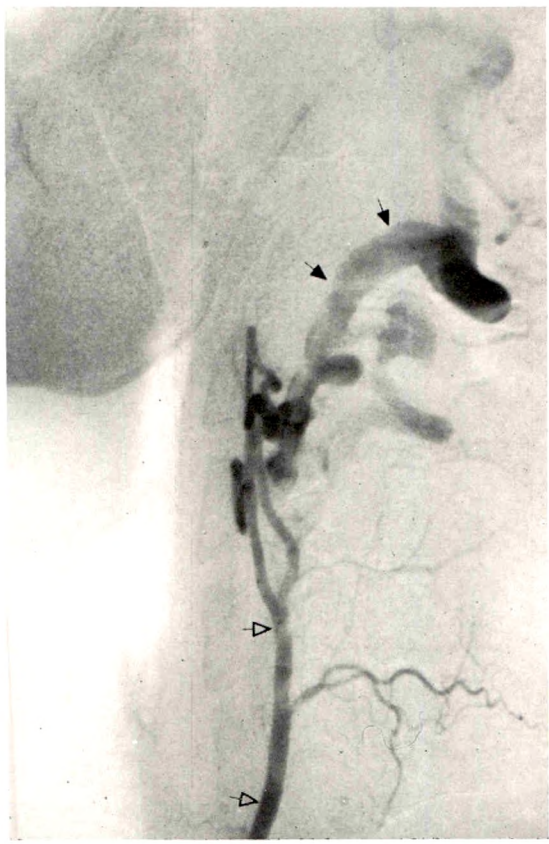


Fig. 5. Selective right deep cervical arteriogram, lateral view. The right deep cervical artery is enlarged, sending off numerous branches at the level of the second and third cervical vertebrae (open arrows). These branches opacify the distal vertebral artery (solid arrows) as the latter emerges from the transverse vertebral foramina. The proximal vertebral artery (not shown here) is diminished in caliber but is patent.

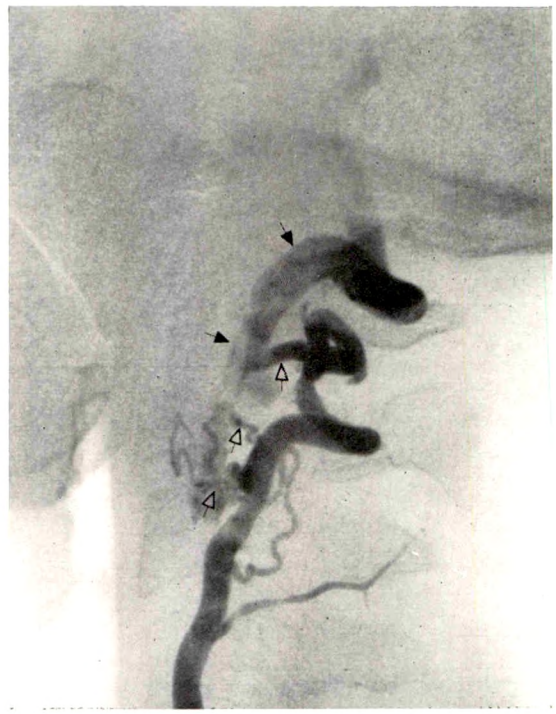


Fig. 6. Selective right ascending cervical arteriogram, lateral view. The right ascending cervical trunk (open arrows) is enlarged. It gives rise to multiple branches which opacify a large distal right vertebral artery (solid arrows). The basilar artery is also enlarged. It supplies the right cerebral hemisphere via the posterior communicating artery.

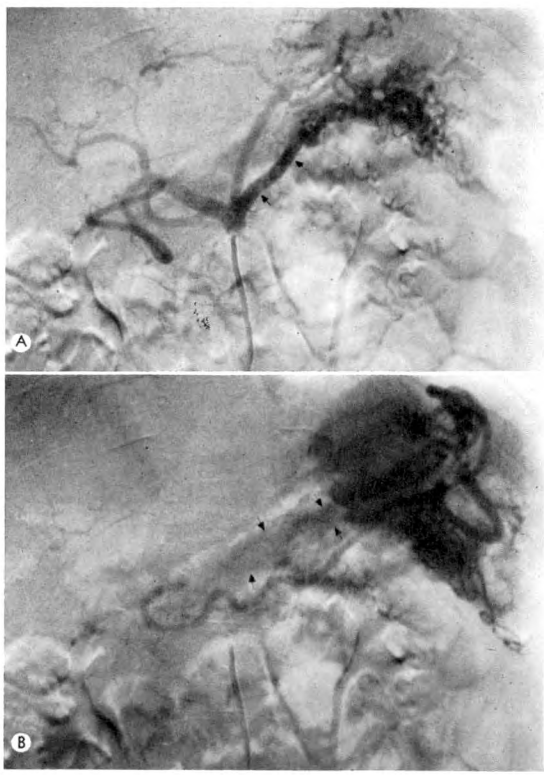


FIG. 7. Selective celiac arteriograms. (*A*) Early arterial phase. The splenic artery (arrows) breaks up distally into a network of arterial channels that converge toward the hilum and supply the spleen. (*B*) Early venous phase. The spleen is densely opacified and the splenic vein is now seen (arrows).

patients also had symptoms of angina pectoris, although atherosclerosis might have been an associated factor. The loss of arterial pulses was not considered the result of occlusion of the lumen, but of degeneration and fragmentation of elastic tissue in the vessel wall. The vessel thereafter is unable to transmit pulsation normally, even when the lumen is patent. Biopsy and necropsy materials not only confirm this degeneration and fragmentation, but also demonstrate well developed collateral circulation in areas where the vessel lumen may be partially obliterated from fragmented thrombi. Hypertension is present in about half the patients with PXE. Valvular heart disease, coronary and peripheral vascular thrombosis, congestive heart failure, and bleeding tendency in the gastrointestinal tract often further complicate the clinical picture.[3]

Carotid rete mirabile in man is a rare developmental anomaly.[8,12] This condition, however, is common in lower vertebrates such as the cat, goat, sheep, and pig (Fig. 9). A true rete mirabile as described in lower vertebrates is a primitive network of intertwined, freely anastomosing vessels. These vessels supply the circle of Willis from the internal carotid artery which is usually relatively small. The rete mirabile must be distinguished from the collateral channels that supply the internal carotid system from the external carotid artery following occlusion of the proximal internal carotid artery.[6,11] These collateral vessels arise mainly from the internal maxillary, ascending pharyngeal, and the occipital

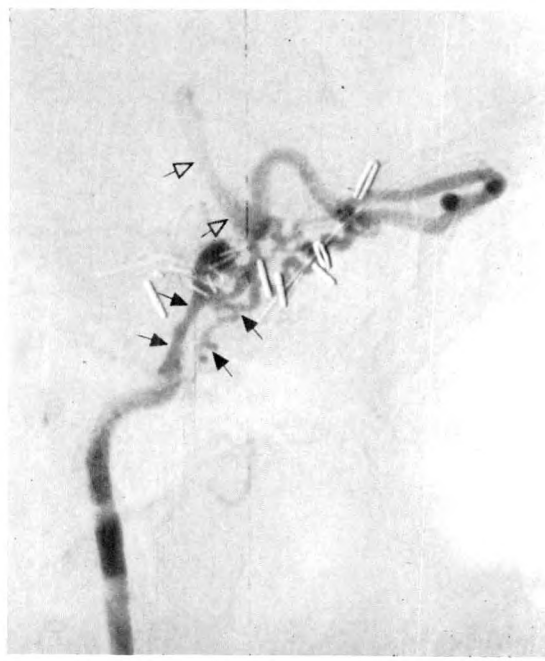


FIG. 8. Selective right internal carotid arteriogram, postoperative study. The copper wires (lying within the thrombosed, nonfilled superior ophthalmic vein) are partly subtracted. The carotid rete mirabile (closed arrows) can now be seen more clearly. Arteriovenous communication is no longer present. Filling of the supraclinoid portion of the internal carotid artery (open arrows) and beyond is now in an antegrade manner.

branches of the external carotid artery. The ophthalmic branch of the internal carotid artery is usually enlarged and is filled in a retrograde manner toward the carotid siphon. The carotid siphon may also be alternately filled directly from the meningeal branches that arise in the cavernous sinus.[12,13]

Caroticocavernous arteriovenous communications are often the sequelae of trauma. An increasing number of "spontaneous" arteriovenous fistulae is seen, however, in the region of the cavernous sinus.[9,10] These fistulae are now thought to be dural arteriovenous shunts between the meningeal branches of either the internal or the external carotid artery and the cavernous sinus itself.

The carotid and vertebral rete mirabile in the patient of this report is probably an incidental developmental anomaly associated with an unusual underlying disorder of connective tissue. A small anastomosing vessel of the rete mirabile in the region of the cavernous sinus on one side, probably ruptured spontaneously. This rupture resulted in the classic clinical triad of unilateral proptosis, engorged conjunctival and episcleral veins, and a loud bruit of a caroticocavernous arteriovenous fistula.

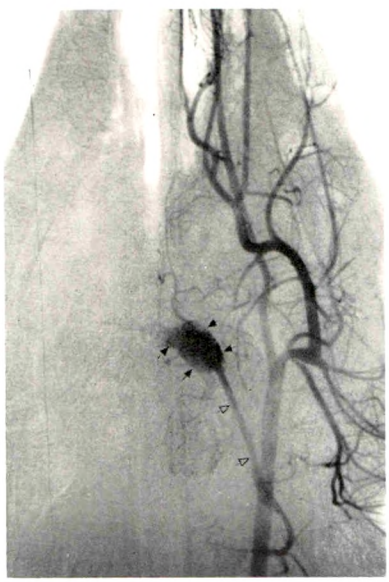


FIG. 9. Common carotid arteriogram in a pig. Anteroposterior view. The internal carotid artery (open arrows) is relatively small. A rete mirabile (closed arrows) consisting of a fine, lace-like arterial network is interposed between the extra and intracranial segments of the internal carotid artery.

SUMMARY

A patient with pseudoxanthoma elasticum associated with bilateral carotid and vertebral rete mirabile is reported.

Unilateral carotid-cavernous sinus fistula was the complication that led to the angiographic investigation.

An associated asymptomatic anomaly of the splenic artery was also observed.

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REFERENCES

1. Bardsley, J. L., and Koehler, P. R. Pseudoxanthoma elasticum: angiographic manifestations in abdominal vessels. *Radiology*, 1969, *93*, 559–562.
2. Carlborg, U. Studies of circulatory disturbances, pulse wave velocity and pressure pulses in larger arteries in cases of pseudoxanthoma elasticum and angioid streaks; contribution to knowledge of function of elastic tissue and smooth muscles in larger arteries. *Acta med. scandinav.*, 1944, Suppl. 151, 1–209.
3. Goodman, R. M., Smith, E. W., Paton, D., Bergman, R. A., Siegel, C. L., Ottesen, O. E., Pusch, A. L., and McKusick, V. A. Pseudoxanthoma elasticum: clinical and histopathological study. *Medicine*, 1963, *42*, 297–334.
4. Hosobuchi, Y. Electrothrombosis of carotid-cavernous fistula. In process for publication.
5. James, A. E., Jr., Eaton, S. B., Blazek, J. V., Donner, M. W., and Reeves, R. J. Roentgen findings in pseudoxanthoma elasticum (PXE). Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *106*, 642–647.
6. Margolis, M. T., and Newton, T. H. Collateral pathways between cavernous portion of internal carotid and external carotid arteries. *Radiology*, 1969, *93*, 834–836.
7. McKusick, V. A. Heritable Disorders of Connective Tissue. Second edition. C. V. Mosby Company, St. Louis, 1960, pp. 213–241.
8. Minagi, H., and Newton, T. H. Carotid rete

mirabile in man: case report. *Radiology*, 1966, *86*, 100–102.

9. NEWTON, T. H., and HOYT, W. F. Spontaneous arteriovenous fistula between dural branches of internal maxillary artery and posterior cavernous sinus. *Radiology*, 1968, *91*, 1147–1150.
10. NEWTON, T. H., and HOYT, W. F. Dural arteriovenous shunts in region of cavernous sinus. *Neuroradiology*, 1970, *1*, 71–81.
11. PARKINSON, D. Collateral circulation of cavernous carotid artery: anatomy. *Canad. J. Surg.*, 1964, *7*, 251–268.
12. VAN DER EECKEN, H. M., and ADAMS, R. D. Anatomy and functional significance of meningeal arterial anastomoses of human brain. *J. Neuropath. & Exper. Neurol.*, 1953, *12*, 132–157.
13. WALLACE, S., GOLDBERG, H. I., LEEDS, N. E., and MISHKIN, M. M. Cavernous branches of internal carotid artery. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1967, *101*, 34–46.
14. WOLFF, H. H., STOKES, J. F., and SCHLESINGER, B. E. Vascular abnormalities associated with pseudoxanthoma elasticum. *Arch. Dis. Childhood*, 1952, *27*, 82–88.

BILATERAL GIANT ANEURYSMS OF THE INTERNAL CAROTID ARTERY*

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ANEURYSMS of a diameter of 2.5 cm. or greater are defined as giant.[7,13] Even bilateral intracavernous giant aneurysms of the internal carotid artery are very rare. There have been 13 cases in the literature, as far as the authors know. Wilson and Myers[21] reported 1 case and collected 10 cases from the literature. Rischbieth and Bull,[15] and Morley and Barr,[13] recorded 1 case each, respectively. Cases diagnosed ante mortem by angiography were only 7.[1,9,11,13,15,16,21] Visual disturbances, although varying in nature and in degree, were noted in nearly all cases. In all, except 1[21] extraocular muscle palsies and/or cavernous sinus syndrome were recognized uni- or bilaterally, but in none was there evidence of hypopituitarism. Bruit was heard in 3[16,21] although in 1 there was a carotid-cavernous fistula on the right in addition to the huge aneurysm on the left.[1] No apparent subarachnoid hemorrhage was recorded. In the case of Seltzer and Hurteau[16] lumbar puncture yielded hemorrhagic cerebrospinal fluid (CSF), after occlusion of the intracranial internal carotid artery distal to the aneurysm in addition to gradual occlusion of the internal carotid artery in the neck.

Although varying from case to case, the roentgenograms of the skull showed some characteristic changes as described by Rischbieth and Bull,[15] including widening of the superior orbital fissure, erosion of the optic canal, erosion of the bone surrounding the pituitary fossa and calcification of the aneurysm. In 1 case[16] roentgenograms of the skull were normal and in another[13] there was no description as to the roentgenographic findings of the skull.

The only patient with bilateral intracavernous giant aneurysms in whom surgical treatment was carried out was that reported by Seltzer and Hurteau.[16] In their case, the indications for operative intervention were impairment of ocular movement, blurred vision, and throbbing headaches. The left internal carotid artery was occluded both proximal and distal to the symptomatic aneurysm. The eye movements were restored but fine movements in the right hand were impaired. According to Wilson and Myers,[21] the patient was re-admitted 4 years later with complete ophthalmoplegia on the right. The right common carotid artery was gradually occluded with a Silverstone clamp, which was opened 24 hours after its application because of the sudden development of left hemiplegia. There was slight improvement in the external ophthalmoplegia; however, the hemiplegia persisted. The patient died in a convalescent home several weeks later. No autopsy was obtained.

The pathogenesis of bilateral intracavernous aneurysms is unknown. Bull[4] considered that the aneurysms occurring in the cavernous portion and the terminal portion of the internal carotid artery are atherosclerotic, because they are quite different from congenital aneurysms in many respects. According to Bull,[4] they seldom rupture; they are not related to points of arterial bifurcation; they are seen almost entirely in the middle-aged and elderly patients; they frequently press upon cranial nerves; they tend to calcify and thus can often be recognized or suspected on plain roentgenograms; sometimes they present clinically as tumor masses rather than aneurysms; and they tend to be large. In addition many authors observed that the infraclinoid aneurysms occurred very

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frequently in hypertensive women.[10,12,15]

Bilateral giant aneurysms of the internal carotid artery, one infraclinoid and the other supraclinoid, are considered extremely rare and they have not been recorded in the literature, as far as the authors know. We are reporting such a case.

Bird *et al.*[3] described 5 cases of supraclinoid giant aneurysms, including a case (Case 3) in which there were a large aneurysm, measuring 5 cm. in diameter, arising from the supraclinoid portion of the right internal carotid artery and a smaller aneurysm, 1 cm. in diameter, on the left internal carotid artery. It was unknown, whether the latter arose from the supra- or infraclinoid portion of the internal carotid artery, because the smaller one was not illustrated. The giant aneurysm showed a spherical area of uptake of the radioisotope just to the right of the midline and above and anterior to the sella turcica, as shown in the present case. Binet and Loken,[2] also recorded a case of a large aneurysm at the internal carotid bifurcation, in which a scintiphotogram obtained 20 seconds after intravenous administration of Tc^{99m} showed an abnormal radioactivity in the suprasellar region.

REPORT OF A CASE

A 57 year old housewife was referred to the hospital on June 6, 1970, because of decreased visual acuity of the left eye. Six years prior to admission, she had suffered from nasal bleeding without cause, while being at work in the household. The bleeding had been profuse and she had been hospitalized for 2 weeks. Three years ago she again experienced nasal bleeding while sleeping. Bleeding occurred every time from the right nostril. The patient noticed for the first time in February, 1970 blurred vision of the left eye which remained stationary. She had had no headache or other neurologic complaints and had otherwise been well except for hypertension, for which she had been under medical care for the past 8 years. The family history was negative.

On examination, by Landolt's ring, vision in the left eye was reduced to 0.3 and could not be corrected. Vision in the right eye was 0.4 and corrected vision 1.5. She had bilateral optic atrophies, more pronounced in the left eye and very slight in the right eye. The visual field examination showed an upper quadrant temporal anopia of the left eye. The right visual field was entirely intact. The general neurologic examination was otherwise normal. There was no bruit. Physical examination was negative except for hypertension (136/100 mm. Hg). Electrocardiograms revealed ventricular premature beat and borderline ST depression; chest roentgenograms showed left ventricular hypertrophy of mild degree.

Laboratory data disclosed elevated total serum cholesterol (234 mg./dl.) and elevated blood sugar (124 mg./dl.). There was neither clinical nor laboratory evidence of hypopituitarism. Spinal tap revealed clear CSF with an opening pressure of 190 mm. of water. The CSF contained 340 mg./dl. of protein and 54 mg./dl. of sugar.

Roentgenograms of the skull, especially the laminagrams, revealed characteristic changes. The left anterior clinoid process and lesser wing of the sphenoid were eroded. The superior orbital fissure was enlarged bilaterally and the intervening bar between the superior orbital fissure and the optic canal was indistinct on the right. The contour of the optic canal was also indistinct bilaterally. The right side of the floor of the sella turcica and the medial aspect of the right middle cranial fossa were destroyed. Both posterior clinoid processes and the dorsum sellae were thinned.

Right common carotid angiograms revealed an infraclinoid intracavernous giant aneurysm, pea-shaped, 2.4×3.0×2.5 cm. in size, extending laterally toward the middle cranial fossa, which remained to be filled until the venous phase. In addition, there was slowed intracranial arterial filling and medial displacement of the cavernous portion of the internal carotid artery (Fig. 1*A*; 2*A*; and 3*A*).

Left common carotid angiograms demonstrated a supraclinoid giant aneurysm, elliptical in shape, 2.5×2.1×2.1 cm. in size, of the internal carotid artery, extending superomedially toward the suprasellar region, which emptied rapidly and was not filled in the phlebograms (Fig. 1*B*; 2*B*; and 3*B*). Both right and left carotid angiograms demonstrated an ophthalmic artery, although Lombardi *et al.*[10] reported that the ophthalmic artery was demonstrated on angiograms in only 55 per cent of infraclinoid aneurysms.

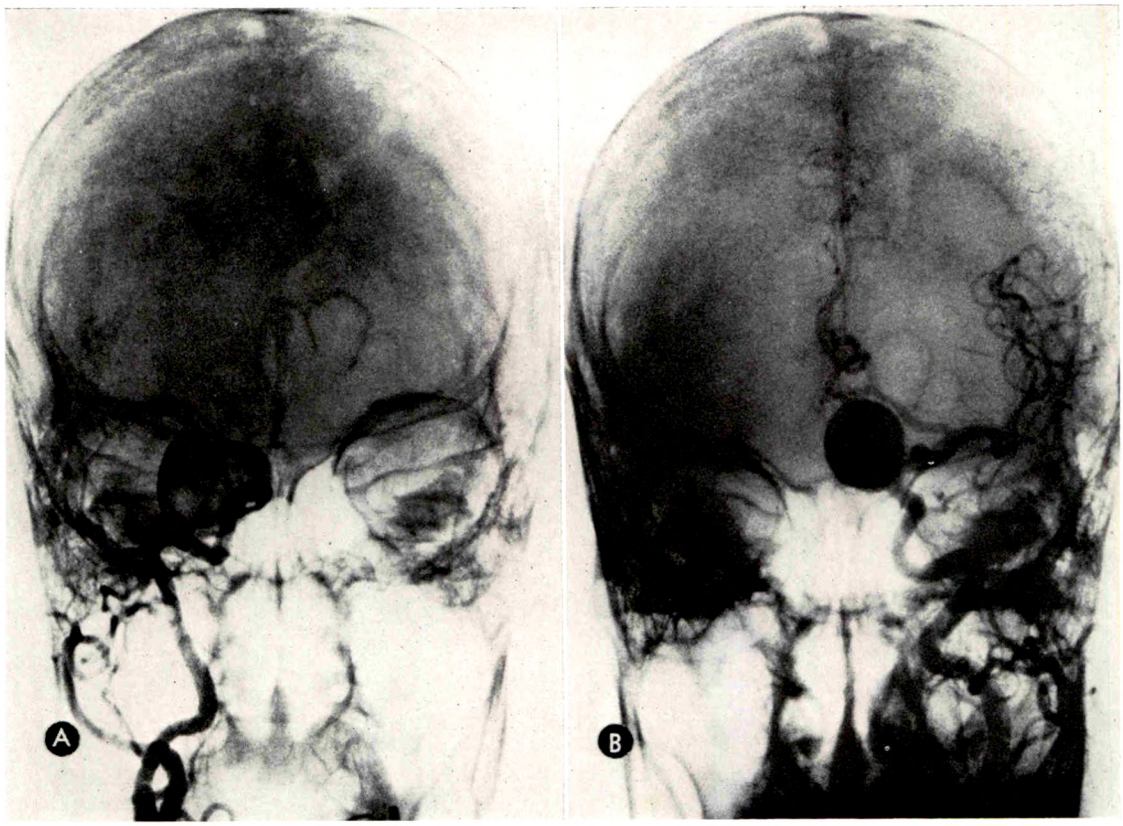


FIG. 1. Carotid angiograms, anteroposterior view. (*A*) Right common carotid angiogram, arterial phase. A giant aneurysm of the infraclinoid portion of the right internal carotid artery, extending laterally toward the middle cranial fossa. (*B*) Left common carotid angiogram, arterial phase. A giant aneurysm of the supraclinoid portion of the left internal carotid artery, extending superomedially toward the suprasellar region.

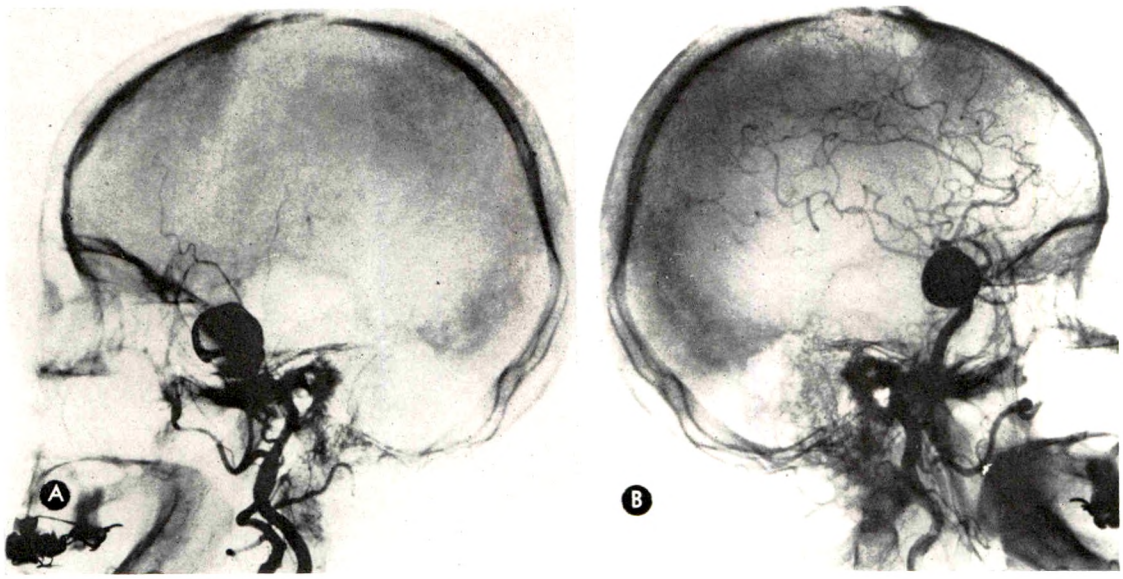


FIG. 2. Carotid angiograms, lateral view. (*A*) Right common carotid angiogram, early arterial phase. A giant aneurysm of the infraclinoid portion of the right internal carotid artery. The intracranial arteries are not yet filled. (*B*) Left common carotid angiogram, arterial phase. A giant aneurysm of the supraclinoid portion of the left internal carotid artery. The intracranial arteries are filled.

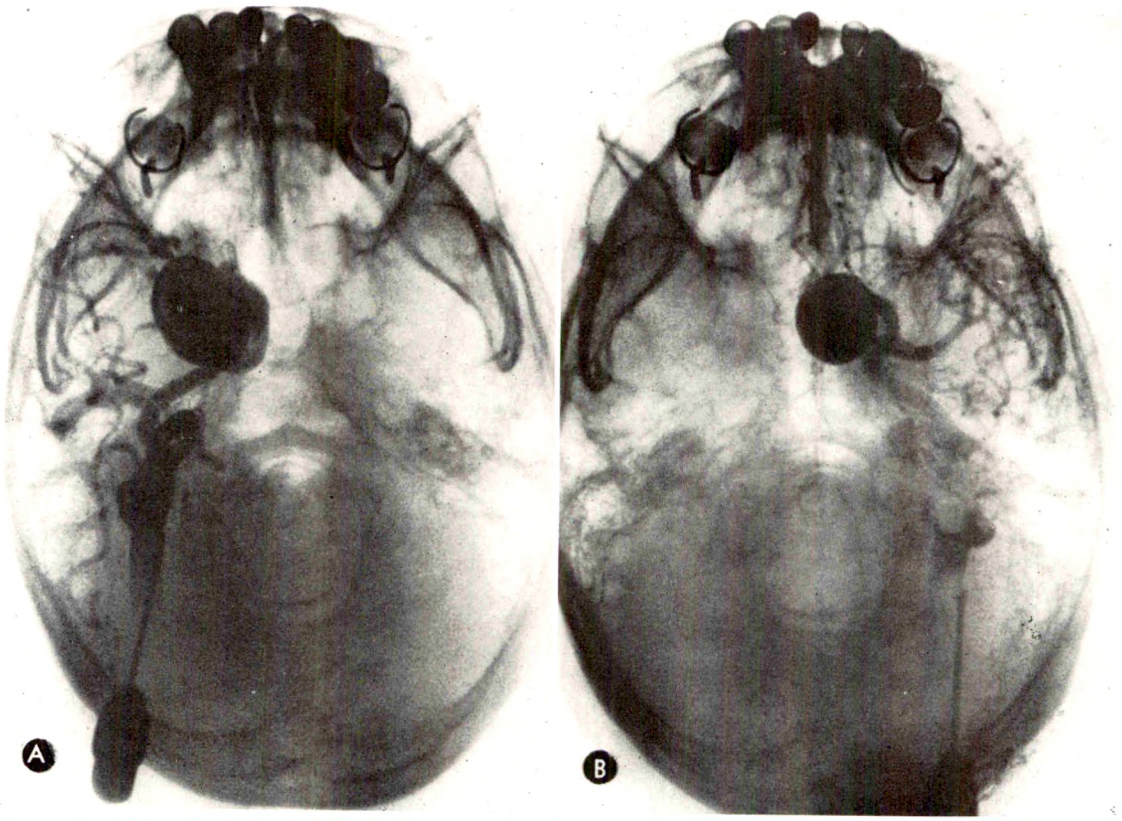


FIG. 3. Carotid angiograms, axial view. (*A*) Right common carotid angiogram, late arterial phase. The right infraclinoid giant aneurysm extends laterally and displaces medially the intracavernous portion of the right internal carotid artery. (*B*) Left common carotid angiogram, arterial phase. The left supraclinoid giant aneurysm extends medially and displaces laterally the intracavernous portion of the left internal carotid artery.

Right retrograde brachial angiograms showed no vascular anomalies except for the infraclinoid intracavernous giant aneurysm of the right internal carotid artery (Fig. 4, *A* and *B*) which could not be visualized with digital compression on the right carotid artery (Fig. 4*C*), although so-called megadolichobasilar anomaly was noted (Fig. 4, *A–C*).

Cavernous sinus venograms demonstrated interesting findings. In the axial view, the left cavernous sinus was filled, but became narrowed distally and its anterior portion was displaced laterally. The usual shadow defect in the cavernous sinus by the internal carotid artery was seen, although not so clearly. On the other hand, the right cavernous sinus was not fully filled and there was a clear shadow defect by the infraclinoid aneurysm near the junction of the inferior petrosal sinus and the cavernous sinus (Fig. 5*A*). In the anteroposterior view the posterior portion of both cavernous sinuses was seen with the shadow defect caused by the internal carotid artery, and the left cavernous sinus was flattened by the left supraclinoid giant aneurysm (Fig. 5*B*).

Pneumoencephalograms revealed that there was an elevation of the anterior portion of the third ventricle by the left supraclinoid aneurysm and of the posterior portion of the third ventricle by the megadolichobasilar anomaly, and that the air in the suprasellar cistern appeared to surround the upper portion of each aneurysm.

Radioactive scan with intravenous administration of 10 mc of Tc^{99m} showed a spherical area of abnormal uptake immediately after, and 20 seconds after, intravenous injection near the midline just above the sella turcica. This uptake was assumed to be due to the left supraclinoid giant aneurysm, extending to the suprasellar region.

The patient was discharged on July 4, 1970,

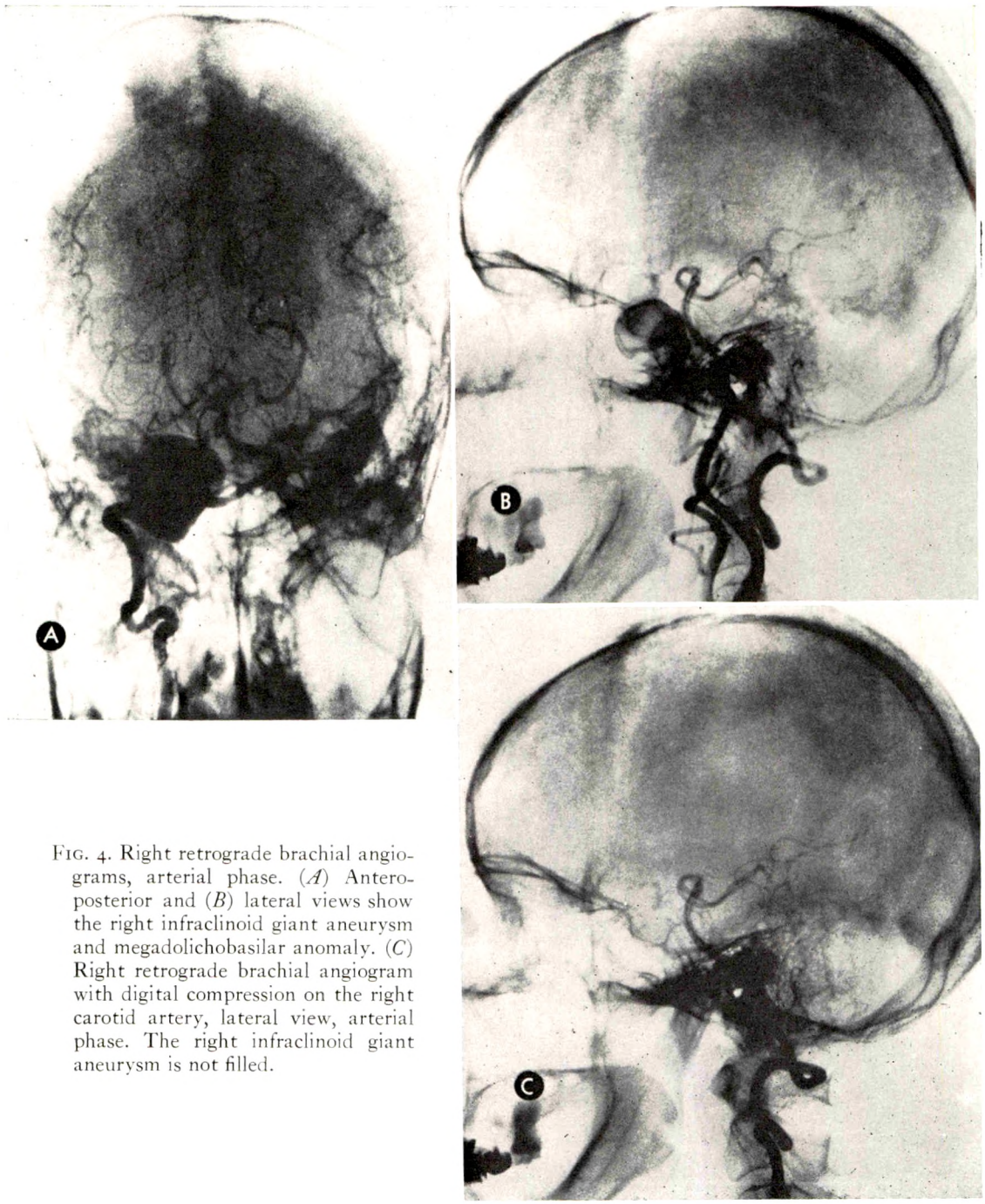


Fig. 4. Right retrograde brachial angiograms, arterial phase. (*A*) Anteroposterior and (*B*) lateral views show the right infraclinoid giant aneurysm and megadolichobasilar anomaly. (*C*) Right retrograde brachial angiogram with digital compression on the right carotid artery, lateral view, arterial phase. The right infraclinoid giant aneurysm is not filled.

without surgical intervention. Although the patient and her family wanted to have surgical treatment, we decided against it. The following are the reasons why we made up our minds not to operate: (1) the visual acuity of the left eye has been impaired but her blurred vision has not been progressive, although there is an apparent optic atrophy on the left and in addition there is another possibility that the right eye vision may be impaired in the near future; (2) both carotid arteries would have to be ligated for both giant aneurysms, because direct attack on giant aneurysms is seldom possible or successful, and both supra- and infraclinoid

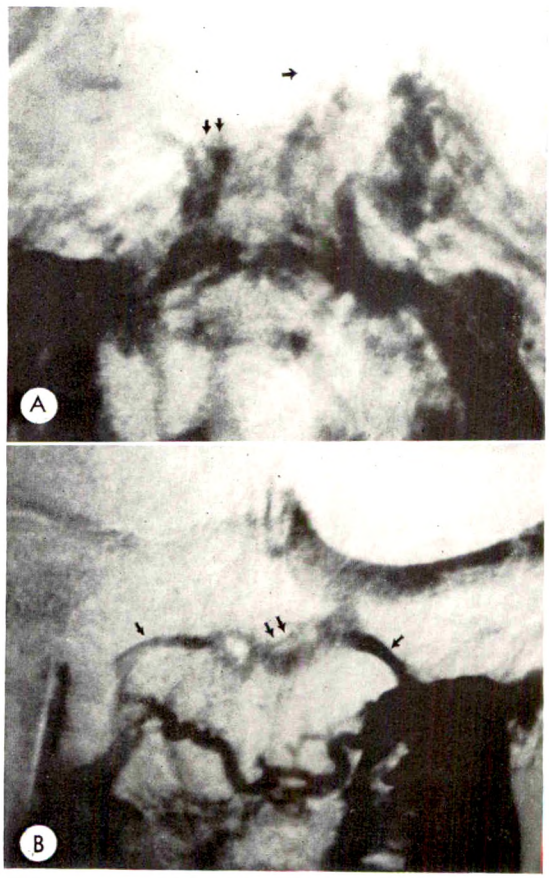


FIG. 5. Cavernous sinus venograms. (*A*) Axial view (electronic subtraction). The left cavernous sinus is narrowed and displaced laterally by the left supraclinoid giant aneurysm (→) and the shadow defect in the cavernous sinus by the intracavernous portion of the internal carotid artery is seen, although not so clearly. The right cavernous sinus is not visualized in its entire length and only the most posterior portion is filled. There is seen a clear shadow defect caused by the right infraclinoid giant aneurysm near the junction of the inferior petrosal sinus and the cavernous sinus (↓ ↓). The intercavernous sinuses are not visualized. (*B*) Anteroposterior view (electronic subtraction). The posterior portion of the cavernous sinus with the shadow defect by the intracavernous portion of the internal carotid artery is seen bilaterally. The left cavernous sinus is flattened by the left supraclinoid giant aneurysm (↓ ↓). The single arrows point to the inferior petrosal sinus.

giant aneurysms of the internal carotid artery must be treated by carotid ligation,[3,13,19] although untoward effects of carotid artery ligation have occasionally been reported.[8,18] Massive enlargement of intracranial aneurysm after carotid artery ligation has also been recorded.[5] Matas' test was tolerated by the patient for over 10 minutes, but the right carotid angiograms revealed slowed intracranial arterial filling and the right brachial angiograms under digital compression of the right carotid artery did not show a satisfactory filling of the right internal carotid artery territory through the posterior communicating artery (Fig. 4*C*). Therefore, we were afraid that ligation of both carotid arteries in this patient might severely compromise the cerebral circulation. There were also the possibilities that the right intracavernous aneurysm may rupture and uncontrollable epistaxis may occur, and that the left aneurysm may give rise to hypopituitarism.

DISCUSSION

The giant aneurysm on the right involved the infraclinoid portion of the internal carotid artery, extending laterally toward the middle cranial fossa. The left giant aneurysm was of supraclinoid origin and extended superomedially toward the suprasellar region.

Using cavernous sinus venography,[6,14,17,20] the left cavernous sinus was slowly filled; its anterior portion was displaced laterally by the left suprasellar giant aneurysm. The right cavernous sinus had only its posterior portion filled. There was a clear shadow defect near the junction of the right inferior petrosal sinus and the right cavernous sinus caused by the infraclinoid giant aneurysm.

We believe that the present case is the first in which bilateral giant aneurysms, one suprasellar and the other infraclinoid, of the internal carotid artery were found by cavernous sinus venography, performed for giant aneurysms of the internal carotid artery.

Furthermore, it is interesting that in the present case bilateral giant aneurysms of the internal carotid artery, one infra- and the other supraclinoid, were associated with megadolichobasilar anomaly. We assume that atherosclerosis might play an important role in forming such giant aneurysms, at least in this patient. Recently, we have

examined a case of megadolichobasilar anomaly associated with an aneurysm at the junction of the posterior inferior cerebellar artery and the vertebral artery. The relationship between magadolichobasilar anomaly and aneurysmal formations will be discussed elsewhere.

SUMMARY

An unusual case of bilateral giant aneurysms of the internal carotid artery, one arising from the intracavernous portion of the right internal carotid artery and the other from the supraclinoid portion of the left internal carotid artery, is reported. So-called megadolichobasilar anomaly is associated with such giant aneurysms of the internal carotid artery.

Characteristic findings were seen in plain roentgenograms of the skull, especially in the laminagrams, bilateral carotid angiograms, right retrograde brachial angiograms, pneumoencephalograms, cavernous sinus venograms and brain scans.

In contrast to the many roentgenologic findings, the clinical manifestations were few. There was a history of epistaxis and blurred vision of the left eye. Neurologic examination revealed bilateral optic atrophies and decreased visual acuity of the left eye with an upper quadrant temporal anopia. The patient had no extraocular muscle palsy, no olfactory trouble, and no evidence of hypopituitarism.

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REFERENCES

1. Alpers, B. J., Schlezinger, N. S., and Tassman, I. M. Bilateral internal carotid aneurysms involving cavernous sinus; right carotid-cavernous sinus fistula and left saccular aneurysm. *Arch. Ophth.*, 1951, *46*, 403–407.
2. Binet, E. F., and Loken, M. K. Scintiangiography of cerebral arteriovenous malformations and aneurysms. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *109*, 707–713.
3. Bird, A. C., Nolan, B., Gargano, F. P., and David, N. J. Unruptured aneurysm of supraclinoid carotid artery; treatable cause of blindness. *Neurology*, 1970, *20*, 445–454.
4. Bull, J. W. D. Contribution of radiology to study of intracranial aneurysms. *Brit. M. J.*, 1962, *2*, 1701–1708.
5. Cuatico, W., Cook, A. W., Tyshchenko, Y., and Khatih, R. Massive enlargement of intracranial aneurysms following carotid ligation. *A.M.A. Arch. Neurol. & Psychiat.*, 1967, *17*, 609–613.
6. Hanafee, W., Rosen, L., Weidner, W., and Wilson, G. Venography of cavernous sinus, orbital veins, and basal venous plexus. *Radiology*, 1965, *84*, 751–753.
7. Locksley, H. B. Natural history of subarachnoid hemorrhage, intracranial aneurysms and arteriovenous malformations, based on 6368 cases in cooperative study. Report on cooperative study of intracranial aneurysms and subarachnoid hemorrhage. Section V, Part II. *J. Neurosurg.*, 1966, *25*, 321–368.
8. Levitt, J. M. Aneurysm of internal carotid artery simulating tumor; report of case. *Arch. Ophth.*, 1950, *43*, 500–503.
9. Logue, V. Saccular aneurysms of internal carotid artery in cavernous sinus occurring bilaterally. *Brit. J. Surg.*, 1951, *39*, 181–182.
10. Lombardi, G., Passerini, A., and Migliavacca, F. Intracavernous aneurysms of internal carotid artery. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1963, *89*, 361–371.
11. Mahoudeau, D., Daum, S., George and Rosier. Ophthalmoplégie double révélatrice d'un anévrysme intracranien bilatéral de la carotide interne. Intérêt diagnostique de l'artériographie. *Bull Soc. méd Hôp.*, 1949, *65*, 503–509.
12. Meadows, S. P. Intracavernous aneurysms of internal carotid artery. *A.M.A. Arch. Ophth.*, 1959, *62*, 566–574.
13. Morley, T. P., and Barr, H. W. K. Giant intracranial aneurysms: diagnosis, course, and treatment. *Clin. Neurosurg.*, 1969, *16*, 73–94.
14. Rand, R. W., and Hanafee, W. N. Cavernous sinus venography and stereotaxic cryohypophysectomy. *J. Neurosurg.*, 1967, *26*, 521–526.
15. Rischbieth, R. H. C., and Bull, J. W. D. Significance of enlargement of superior orbital (sphenoidal) fissure. *Brit. J. Radiol.*, 1958, *31*, 125–135.
16. Seltzer, J., and Hurteau, E. F. Bilateral symmetrical aneurysms of internal carotid artery within cavernous sinus; case report. *J. Neurosurg.*, 1957, *14*, 448–451.

17. SHIU, P. C., HANAFEE, W. N., WILSON, G. H., and RAND, R. W. Cavernous sinus venography. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1968, *104*, 57–62.
18. STRENGER, L. Neurological deficits following therapeutic collapse of intracavernous carotid aneurysms; report of two cases. *J. Neurosurg.*, 1966, *25*, 215–218.
19. TYTUS, J. S., and WARD, A. A., JR. Effect of cervical carotid ligation on giant intracranial aneurysms. *J. Neurosurg.*, 1970, *33*, 184–190.
20. WAGA, S., KIKUCHI, H., HANDA, J., and HANDA, H. Cavernous sinus venography. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *109*, 130–137.
21. WILSON, C. B., and MYERS, F. K. Bilateral saccular aneurysms of internal carotid artery in cavernous sinus. *J. Neurol., Neurosurg. & Psychiat.*, 1963, *26*, 174–177.

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ANEURYSMS OF THE CERVICAL INTERNAL CAROTID ARTERY*

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IN THE pre-antibiotic era, erosive aneurysms of the cervical internal carotid artery were an uncommon but often fatal complication of peritonsillar infections.[40] Since the advent of antibiotics, cervical internal carotid aneurysms have become less common. Such lesions are easily confused with chemodectomas, peritonsillar abscesses, or peritonsillar neoplasms, and a review of the literature indicates that many have been biopsied prior to the correct diagnosis being made by carotid arteriography.

Case reports illustrating various presenting features of these aneurysms follow.

REPORT OF CASES

CASE 1. This 82 year old female was admitted with a history of difficult breathing of 3 months' duration.

On physical examination a 3×4 cm. mass was seen in the left tonsillar fossa displacing the uvula to the right (Fig. 1*A*). No mass was palpable on external examination of the neck. A biopsy was contemplated, but the pharyngeal mass was found to be pulsatile, and a bruit was detected. Plain film roentgenograms of the neck revealed a 4.5×7 cm. oval rim of calcification at the angle of the jaw. A left carotid arteriogram demonstrated that this represented a partially thrombosed aneurysm of the left internal carotid artery (Fig. 1*B*).

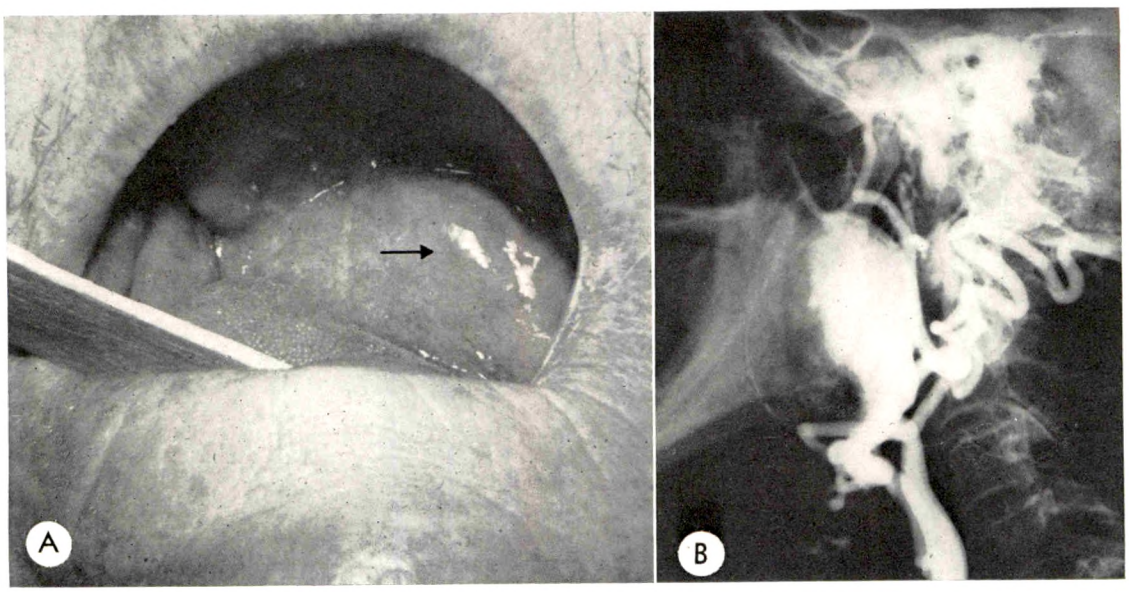


FIG. 1. Case 1. (*A*) View of the patient's oropharynx showing a soft tissue mass protruding into the left tonsillar fossa and displacing the uvula to the right. (*B*) Left common carotid arteriogram demonstrates a 4.5×7 cm. aneurysm of the internal carotid artery arising just below the angle of the jaw and extending to the skull base. Separation between the calcific rim of aneurysm and the contrast material indicates thrombus within the aneurysm.

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Fig. 2. Case II. Left common carotid arteriogram demonstrates a lobulated 1.5×2 cm. pseudoaneurysm of the internal carotid artery. The aneurysm lies well above the angle of the mandible. A bullet is seen posterior to the aneurysm.

Three weeks later the neck was explored, but the aneurysm was seen to extend to the base of the skull, and was unresectable.

The patient expired in the postoperative period after developing bronchopneumonia.

Case II. The patient, a 31 year old male, was admitted in shock and coma with bullet wounds of the head and right chest.

On physical examination entry wounds were present in the left and right occipital regions and in the right superior thorax. There was a right hemiparesis. Plain film roentgenograms showed a bullet lodged anterolateral to C1, and metallic fragments in the right frontoparietal region. There was no palpable neck or pharyngeal mass.

A left carotid arteriogram was obtained which demonstrated a $2\times1\frac{1}{2}$ cm. lobulated pseudoaneurysm of the cervical internal carotid artery adjacent to the bullet lodged in the neck (Fig. 2).

The intracranial angiographic series demonstrated swelling of the left cerebral hemisphere, without evidence of an extracerebral collection.

Further surgery was deferred because of the patient's clinical state.

The patient subsequently developed a right hemopneumothorax and bilateral aspiration pneumonitis and died 7 days after admission.

Case III. The patient, a 41 year old male, was admitted with a right hemiparesis and right hemianopsia which had developed the previous day. No mass was appreciated in the neck or pharynx. The Wassermann reaction was negative.

Common carotid arteriography was carried out with a needle placed at C5, well below the carotid bifurcation. A localized filling defect caused by an atheroma was noted in the internal carotid artery 1.5 cm. from its origin; 3 cm. from its origin a progressive constriction of the internal carotid artery was demonstrated (Fig. 3*A*).

In spite of anticoagulant therapy the patient's condition deteriorated and he died 4 days after the onset of his symptoms.

Postmortem sections of the brain demonstrated areas of infarction in the left frontal and parietal lobes, and the medial aspect of the right frontal lobe. Sections of the internal carotid artery demonstrated a dissecting hemorrhage of the wall of the artery extending over a 3–4 cm. segment (Fig. 3*B*); intimal tears were found at both the lower and upper ends of the dissection. Since the artery had been punctured well below the site of the dissection, this was considered to be spontaneous dissecting aneurysm.

Case IV. This 70 year old woman presented with the complaint of pain in the right ear and an enlarging mass at the angle of the right jaw of 1 month's duration.

The neck was surgically explored but a biopsy was not taken and the nature of the mass was not determined. A few weeks later the mass had enlarged in size, was obviously pulsatile, and a bruit was detected.

Right carotid angiography revealed a 4 cm. saccular aneurysm of the internal carotid artery (Fig. 4).

The aneurysm was subsequently excised and

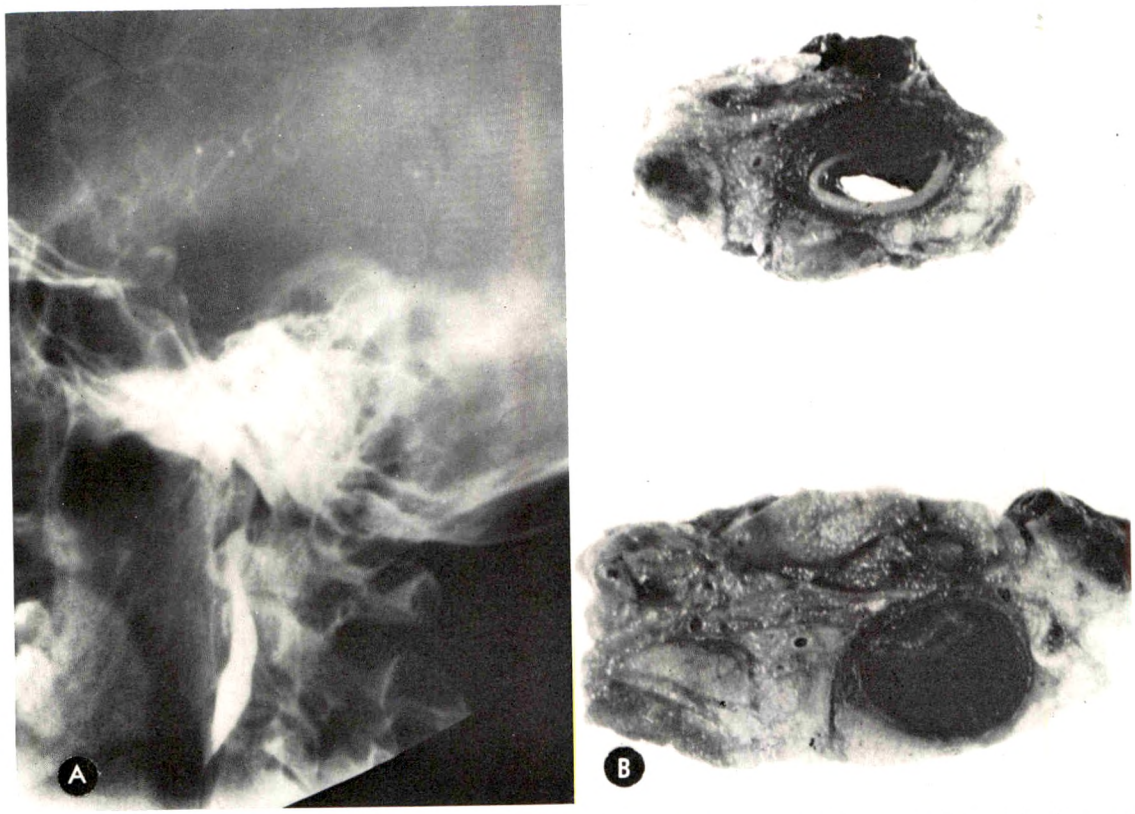


Fig. 3. Case III. (*A*) Left common carotid arteriogram demonstrates a tapered constriction of the internal carotid artery 3 cm. from its origin. (*B*) Postmortem transverse sections of internal carotid artery at site of constriction showing progressive occlusion of the vessel by hemorrhage within its wall. (Reproduced with permission of the *Journal of Neurology, Neurosurgery & Psychiatry*, 1959, *22*, 195–201.)

an end-to-end anastomosis of the internal carotid artery segments performed.

The patient experienced transient swallowing and speech difficulty, but otherwise recovered uneventfully.

Case V. The patient, a 29 year old Negro male, was admitted with the complaint of a left neck mass present for 1 month.

On physical examination the mass was seen to lie in the anterior triangle of the left neck, was pulsatile, and a bruit was detected. Serologic tests for syphilis were strongly positive.

A left common carotid arteriogram demonstrated a large aneurysm arising from the proximal portion of the left internal carotid artery (Fig. 5). The aneurysm was thought to be syphilitic in origin.

The left common carotid artery was ligated in the neck, and the patient recovered without neurologic sequelae.

DISCUSSION

In the pre-antibiotic era most cervical internal carotid artery aneurysms were of inflammatory origin. A bacterial pharyngeal or tonsillar infection, often streptococcal, resulted in an erosive lesion of the adjacent internal carotid artery. These pseudoaneurysms were difficult to detect clinically, and often the patient bled massively before the correct diagnosis was reached. At present, cervical internal carotid artery aneurysms are more frequently caused by atherosclerosis or penetrating trauma such as bullet wounds. Other traumatic causes include mandibular fractures, blunt injury, and weakness of carotid endarterectomy patch grafts. Spontaneous dissecting aneurysms and congenital aneurysms are rare.



FIG. 4. Case IV. Left common carotid arteriogram demonstrating a 3.5×4 cm. aneurysm of the internal carotid artery; most of the aneurysm lies below the angle of the jaw.

Pulsation and a bruit are not absolute indications that a cervical mass is an aneurysm since a tortuous carotid artery[12] or a carotid body tumor[6] may evince the same signs. Moreover, when a hematoma surrounds an aneurysm, no pulsation or bruit may be detected.[32]

Having found that 1 of the 5 cervical internal carotid aneurysms reported in this paper presented as a mass in the tonsillar fossa, 2 presented as neck masses, and 2 showed no palpable mass, we became interested in the anatomic explanation for this difference. Cases of cervical carotid aneurysms in which a carotid arteriogram was published with the clinical data were reviewed. Forty-seven such cases[2,4,5,7–11,13–39,41] were found in the literature, with the present 5 cases, a total of 52. When the common carotid artery bifurcates at its usual level (C_4) the proximal few centimeters of internal carotid artery lie close to the skin in the anterior triangle of the neck, and aneurysms of this portion of the internal carotid artery then project into the anterior triangle below the body of the mandible.[40] Common carotid artery aneurysms also present in the neck. Above the angle of the mandible the internal carotid artery is bounded laterally by the styloid process and associated dense aponeurotic tissue, the ramus of the mandible, and the digastric and stylohyoid muscles (Fig. 6); posteriorly the artery is delimited by the cervical vertebrae. Consequently an aneurysm arising above or projecting above the inferior margin of the mandible finds its least resistance to expansion anteromedially into the soft tissue of the lateral pharynx and tonsillar fossa[10,16,20,23,24,32,41] as in Case 1. Such an aneurysm may cause dysphagia or dyspnea and be mistaken for a peritonsillar abscess or neoplasm. In one case a large aneurysm projected both above and below the angle of the mandible and presented with masses in both the neck and pharynx.[37]

An aneurysm 2 cm. or less in width, particularly a dissecting or fusiform aneurysm, frequently fails to cause a recognizable mass, and may not be detected on palpation;[2,4,8,11,14,17,19,22,26,28,31,35,36,38] this is especially true if the aneurysm lies above the inferior margin of the mandible, as in Cases II and III. In those cases in which a mass was not clinically apparent, carotid



FIG. 5. Case V. Left common carotid arteriogram demonstrating a 3.5×4.5 cm. aneurysm arising from the internal carotid artery at its origin.

arteriography was often performed because of the symptoms of cerebral ischemia.[4,8,17,22,35,38] Most likely these symptoms were the result of embolism from the aneurysm, or constriction of the vessel lumen by dissection. In other cases cervical pseudoaneurysms were an incidental finding in patients studied primarily for traumatic brain injury,[28,31] as in Case II.

Plain roentgenograms of the neck may show a calcified rim around the periphery of the aneurysm. Separation of contrast from this rim on arteriography indicates thrombus within the aneurysm (Fig. 1*B*). Arteriography affords a means of evaluating operability, since aneurysms which extend to the skull base (Case I) are often unresectable.[1] Aneurysms lower in the neck and not confined laterally by the mandible (Case IV) are much more accessible to surgery.

SUMMARY

While aneurysms of the common carotid artery usually present as mass lesions of the neck, aneurysms of the cervical internal carotid artery may present either in the neck or the pharynx.

In the latter location they may resemble peritonsillar abscess, and many such lesions have been biopsied with tragic consequences.

Cervical carotid aneurysms which are not palpable frequently present with cerebral symptoms.

Anatomic reasons for the varied clinical presentation of these lesions are emphasized.

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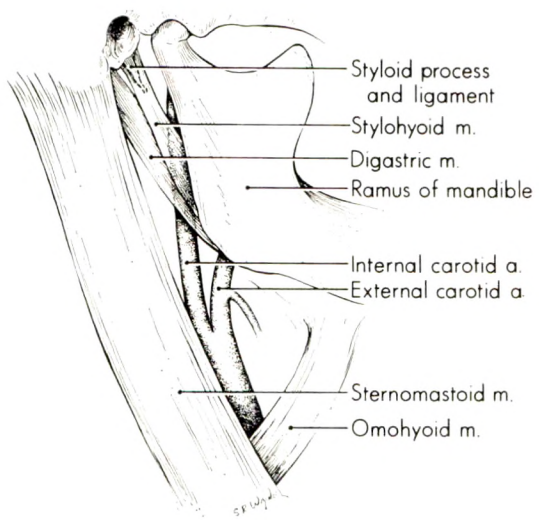


FIG. 6. Diagram showing the structures bounding the carotid artery anterolaterally in the neck.

REFERENCES

1. ALEXANDER, E., WIGGER, S. M., and DAVIS, C. H. Bilateral extracerebral aneurysms of internal carotid artery. *J. Neurosurg.*, 1966, *25*, 437–442.
2. AMBLER, Z., ULC, M., and LEDINSKY, Q. Aneurysma der A. carotis int. im extrakraniellen Verlauf. *Neurochir.*, 1967, *10*, 165–175.
3. ANDERSON, R. D., LIEBESKIND, A., SCHECHTER, M. M., and ZINGESSER, L. H. Aneurysms of internal carotid artery in carotid canal of petrous temporal bone. *Radiology*, 1972, *102*, 639–642.
4. BEAL, A. C., CRAWFORD, E. S., COOLEY, D. A., and DEBAKEY, M. E. Extracranial aneurysms of carotid artery. Cases 2, 4, 5, 6. *Postgrad. Med.*, 1962, *32*, 93–102.
5. BENVENUTO, R., DESPRES, J. P., PRIBRAM, H. F., and GALLAGHER, J. C. Excision of extracranial aneurysm of internal carotid artery employing hypothermia. *J. Thoracic & Cardiovasc. Surg.*, 1961, *2*, 165–167.
6. BERGEN, J. J., and TRIPPEL, O. H. Current concepts in surgery of extracranial carotid artery. *Surg. Clin. North America*, 1963, *43*, 277–294.
7. BOATMAN, K. K., and BRADFORD, V. A. Excision of internal carotid aneurysm during pregnancy employing hypothermia and vascular shunt. *Ann. Surg.*, 1958, *148*, 271–275.
8. BURKLAND, C. W. Spontaneous dissecting aneurysm of cervical carotid artery. Cases 1, 2. *Johns Hopkins Med. J.*, 1970, *126*, 154–159.
9. BUXTON, J. T., STEVENSON, T. B., and STALLWORTH, J. M. Arteriosclerotic aneurysm of extracranial internal carotid artery treated by excision and primary re-anastomosis under controlled hypertension. *Ann. Surg.*, 1964, *159*, 222–226.
10. CAMPICHE, R., and ZANDER, E. Anévrisme de la carotide interne dans son trajet extracranien formant une tumour pharyngienne. *Neurochir.*, 1962, *8*, 79–83.
11. DAVIS, R. H., DAROFF, R. B., and HOYT, W. F. Hemicrania, oculosympathetic paresis, and subcranial carotid aneurysm. *J. Neurosurg.*, 1968, *29*, 94–96.

12. Dieterling, R. A. Tortuous right common carotid artery simulating aneurysm. *Angiology*, 1952, *3*, 483–492.
13. Dimtza, A. Aneurysms of carotid arteries. Cases 1, 2. *Angiology*, 1956, *7*, 218–227.
14. Duplay, J., Cossa, P., and Marchal, E. Anévrismes multiples de la carotide interne dans son segment exocranien et dans son segment endocranien. *Presse méd.*, 1957, *65*, 143–144.
15. Eiseman, B., Paton, B., and Hogshead, H. Use of internal polyethylene shunt during resection of carotid aneurysm. *Am. J. Surg.*, 1961, *102*, 702–705.
16. Elliott, T. E., Sikkema, W. W., and West, J. T. Resection of extracranial internal carotid aneurysm. *Am. J. Surg.*, 1967, *113*, 812–815.
17. Finney, L. A., and David, N. J. Aneurysm of extracranial internal carotid artery. *Neurology*, 1964, *14*, 376–379.
18. Halaz, N. A., and Kennedy, J. C. Excision of arteriosclerotic aneurysms of cervical internal carotid artery. *J. Neurosurg.*, 1964, *21*, 352–357.
19. Hardin, C. A., and Snodgrass, R. G. Dissecting aneurysm of internal carotid artery treated by fenestration and graft. Cases 1, 2. *Surgery*, 1964, *55*, 207–209.
20. Isfort, A. Bemerkenswerte Aneurysmen der A. carotis interna. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1966, *105*, 851–857.
21. Jamieson, A. Aneurysm of extracranial carotid artery. *British Columbia Med. J.*, 1968, *10*, 73–74.
22. Kianouri, M. Extracranial carotid aneurysm: treatment by excision and end-to-end anastomosis. *Ann. Surg.*, 1967, *165*, 152–156.
23. Kieny, R., Baumgartner, J., Vierling, J. P., and Wertheimer, J. L'anévrisme de la carotide interne extra-cranienne. *Mém. Acad. Chir*, 1969, *95*, 411–418.
24. Kramer, W. Hyperplasie fibromusculaire et anévrisme extracranien de la carotide interne avec syndrome parapharyngien typique. *Rev. neurol.*, 1969, *120*, 239–244.
25. Lepoire, J., Montaut, J., Renard, M., Grosdidier, J., and Mathieu, P. Anévrisme sacculaire de la carotide cervicale; complication d'une angiographie carotidienne percutanée. *Neurochir.*, 1964, *10*, 275–281.
26. Liliequist, B. Roentgenologic appearance of spontaneous dissecting aneurysm of cervical internal carotid artery: report of case. *Vasc. Surg.*, 1968, *2*, 223–226.
27. Nakao, K. Extrakranielle Aneurysmen der Arteria carotis interna. *Med. Klin.*, 1968, *63*, 58–60.
28. New, P. F., and Momose, K. J. Traumatic dissection of internal carotid artery at atlantoaxial level, secondary to nonpenetrating injury. Cases 1, 2. *Radiology*, 1969, *93*, 41–49.
29. O'Hara, I., Utsumi, N., and Ouchi, H. Resection of extracranial internal carotid artery aneurysms with arterial reconstruction. Cases 1, 2. *J. Thoracic & Cardiovasc. Surg.*, 1968, *9*, 365–373.
30. Raphael, H. A., Bernatz, P. E., Spittell, J. A., and Ellis, F. H. Cervical carotid aneurysms: treatment by excision and restoration of arterial continuity. Case 2. *Am. J. Surg.*, 1963, *105*, 771–778.
31. Salmon, J. H., and Blatt, E. S. Aneurysm of internal carotid artery due to closed trauma. Case 1. *J. Thoracic & Cardiovasc. Surg.*, 1968, *56*, 28–32.
32. Silcox, L. A., and Updegrave, R. A. Extracranial aneurysm of internal carotid artery. *A.M.A. Arch. Otolaryng.*, 1958, *69*, 329–333.
33. Smith, R. B., Perdue, G. D., Collier, R. H., and Stone, H. H. Post-operative false aneurysm of carotid artery. Cases 2, 3. *Am. Surgeon*, *36*, 335–341.
34. Sperling, M., and Viehweger, G. Extrakranielle Aneurysmen der Arteria carotis interna und ihre Behandlung. *Chir.*, 1963, *34*, 369–372.
35. Thapedi, I. M., Ashenhurst, E. M., and Rozdilsky, B. Spontaneous dissecting aneurysm of internal carotid artery in neck: report of case and review of literature. *A.M.A. Arch. Neurol.*, 1970, *23*, 549–554.
36. Ullrich, D. P., and Sugar, O. Familial cerebral aneurysms including one extracranial internal carotid artery aneurysm. *Neurology*, 1960, *10*, 288–294.
37. Van Rensburg, L. C. Aneurysm of internal carotid artery presenting as peritonsillar abscess. *South African M. J.*, 1964, *38*, 567–563.
38. Webb, R. C., and Barker, W. F. Aneurysms of extracranial internal carotid artery. Cases 3, 5. *A.M.A. Arch. Surg.*, 1969, *99*, 501–505.
39. Wemple, J. B., and Smith, G. W. Extracranial carotid aneurysm. Cases 2, 5. *J. Neurosurg.*, 1966, *24*, 667–671.
40. Winslow, N. Extracranial aneurysm of internal carotid. *Arch. Surg.*, 1926, *13*, 689–729.
41. Zakrzewski, A. Spontaneous extracranial aneurysms of internal carotid artery. Cases 1, 2. *J. Laryng. & Otol.*, 1963, *77*, 342–350.

ANGIOGRAPHY IN PATIENTS WITH INTRACTABLE EPISTAXIS*

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ANGIOGRAPHY has become increasingly important in the evaluation and treatment of bleeding from various organs. It is especially useful for localizing bleeding in the gastrointestinal tract[2,4,11–14] and the central nervous system,[7,9,10,14,15] and some authors have commented upon its value in bleeding from the kidney.[5,6,9] Even if extravasation of contrast medium is not seen, a lesion known to be a cause of bleeding may be identified. Following angiographic demonstration of the bleeding site, vasoconstrictive drugs can be infused in many to attempt nonoperative control of the bleeding.[3]

Duggan and Brylski[8] have demonstrated the value of angiography in traumatic epistaxis. Spontaneous epistaxis can also be evaluated by angiography, localizing for the surgeon the source of bleeding.

We recently examined 2 patients with acute uncontrollable epistaxis. This report summarizes our experience with these patients and discusses the potential role of angiography in this area.

REPORT OF CASES

CASE 1. A 39 year old woman was admitted to the University Hospital with a 1 hour history of severe right-sided epistaxis. Prior to this episode she was in excellent health with no history of previous epistaxis, hypertension, bleeding disorders or trauma.

Physical examination was normal, except for an elevated blood pressure of 180/120 mm. Hg, and spurting blood from the right nostril. Hematocrit at the time of admission was 37 per cent.

The usual therapeutic measures including posterior nasal packs, with and without vasoconstrictors, only controlled the bleeding temporarily and ligation of the sphenopalatine artery was performed. Following surgery the hemorrhage continued and over the course of a 2 week hospitalization, 12 units of whole blood were required.

On the 14th day of hospitalization, arteriography was performed. Using a percutaneous femoral catheterization approach, the arteries were catheterized, and internal and external carotid angiograms were obtained. Extravasation of contrast medium was demonstrated from distal branches of the pterygopalatine portion of the right internal maxillary artery in the region of the posterior maxillary antrum. The remaining vessels were normal (Fig. 1; and 2, *A–D*).

Surgery was performed after the angiogra-

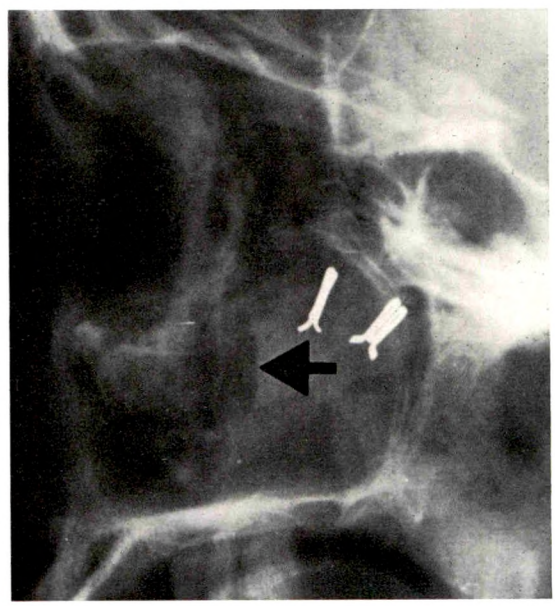


FIG. 1. Preliminary roentgenogram prior to injection of contrast material demonstrates mottled fluid density in the nasopharynx caused by blood and nasal packs. Note air-fluid level within the maxillary antrum (arrow) and Olivecrona clips secondary to previous attempts to obliterate the sphenopalatine branch of the right internal maxillary artery.

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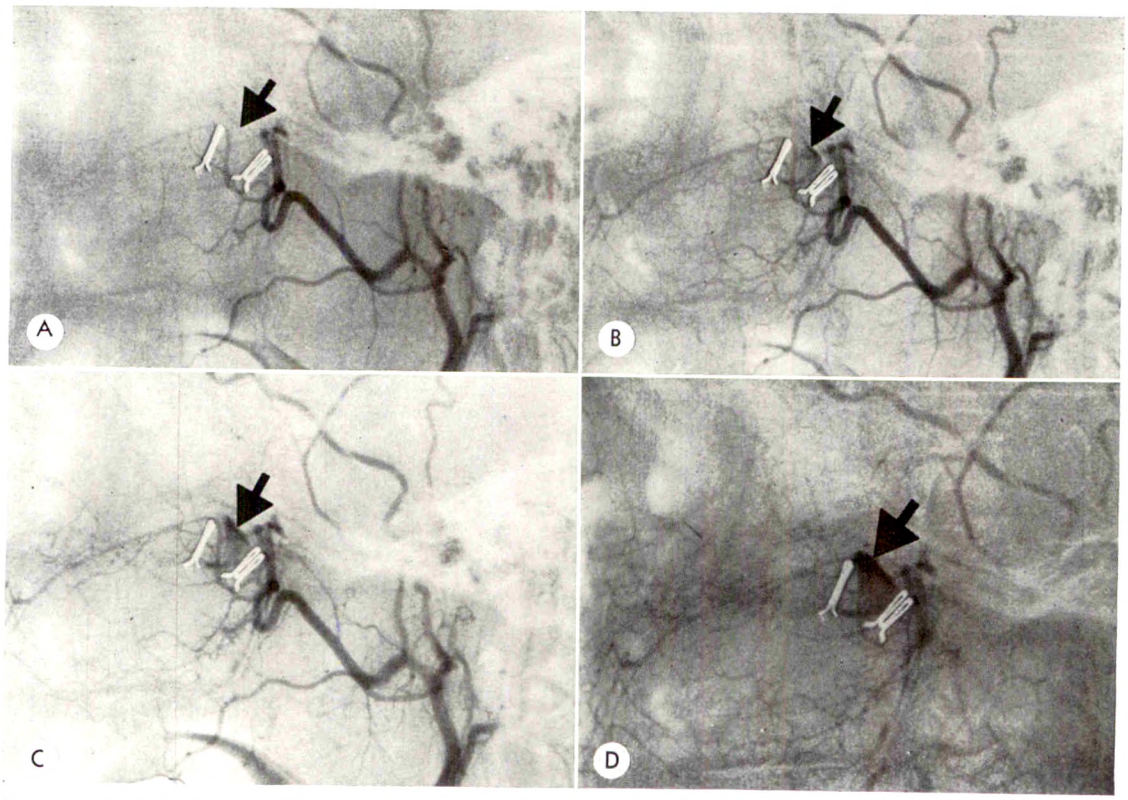


FIG. 2. (*A–D*) Serial magnification roentgenograms following injection of contrast material into the external carotid artery demonstrate extravasation of contrast material (arrow) in the vicinity of the previous surgery. Note that the posterosuperior margin of the contrast material conforms to the bony margin of the maxillary antrum. This extravasation is occurring in the vicinity of the pterygopalatine division of the internal maxillary artery.

phy. Exploration of the maxillary antrum showed hemorrhage, although a specific bleeding branch could not be identified. The internal maxillary artery was then ligated, resulting in prompt and permanent cessation of the epistaxis.

CASE II. A 36 year old male nurse with a history of rheumatic heart disease and mild essential hypertension was admitted to the University Hospital after 3 days of intermittent epistaxis. There was no history of trauma, previous epistaxis, or bleeding disorder.

Physical examination revealed a blood pressure of 150/100 mm. Hg and spurting blood from the right nostril. The hematocrit was 42 per cent.

Therapeutic measures, including anterior and posterior nasal packs, with and without vasoconstrictors only controlled the bleeding temporarily. The hematocrit dropped to 33 per cent, and 3 units of blood were required to stabilize the blood volume.

External carotid arteriography via a femoral approach was performed on the third hospital day. On a Waters' view, contrast material extravasation from the medial branches of the distal internal maxillary artery was seen. No extravasation was noted on the first injection in the lateral projection.

The patient was taken to surgery, where the sphenopalatine portion of the internal maxillary artery was clipped, resulting in immediate cessation of bleeding.

DISCUSSION

Most spontaneous epistaxis in adults arises from a sclerotic sphenopalatine artery or other internal maxillary branch in the posterior portion of the nasal cavity. Posterior ethmoidal branches of the oph-

thalmic artery can bleed following trauma. Hypertension may be associated with epistaxis, or, more rarely, bleeding diatheses, angiomas, viral infections or sudden atmospheric decompression (caisson disease). These vessels lie in thin mucosa closely applied to cartilage or bone, and retraction of the bleeding vessel is minimal. This results in vigorous nasal hemorrhage in a rather confined, inaccessible region. In children and young adults, epistaxis usually arises anteriorly from the anterior ethmoidal artery branches of the ophthalmic artery.

The early demonstration of a source of hemorrhage can prevent considerable loss of blood with its concomitant complications, and can significantly decrease the length of treatment, saving the patient considerable discomfort, time and expense. Moreover, hemorrhage in the posterior nasal cavity usually occurs in a relatively inaccessible region and the surgeon is greatly aided by knowing precisely which vessels are responsible for the bleeding. Bleeding may be from internal maxillary artery branches or from ethmoidal artery branches, and both external and internal carotid arteriograms should be obtained to localize the source. Ballenger[1] states, "The accurate location of the exact site of bleeding is important not only for the accurate placement of cautery, for other forms of local therapy, but also for identifying the trunk blood vessels supplying this point."

With modern angiographic techniques, selective external and internal carotid angiographies are relatively easily performed.

Angiography may define the site of bleeding in many instances of uncontrolled epistaxis and may prove to be very useful in the therapy of this disease. Water-soluble contrast media produce peripheral vasodilatation, and allow for increased blood loss. As in our Case II, a second injection may be necessary to see extravasation. It is conceivable that the infusion of vasoconstrictive drugs into the parent artery may result in cessation of bleeding and the avoidance of surgery.

CONCLUSIONS

This report documents the role of angiography in cases of uncontrolled epistaxis. In 2 patients extravasation of contrast medium was noted from distal branches of the right internal maxillary artery. In each case following angiography, the vessel was ligated and the bleeding was arrested.

Angiography should prove useful in evaluating patients with uncontrolled epistaxis by determining the source of hemorrhage.

It is anticipated that following demonstration of bleeding from the external carotid branches, the addition of vasoconstrictor drugs could be infused at the time of angiography to control the hemorrhage and possibly decrease the need for surgery in some patients.

SUMMARY

Internal and external carotid angiography was used to precisely localize the site of hemorrhage in 2 patients with severe, uncontrollable epistaxis.

Following angiography, ligation of the internal maxillary artery in each case resulted in prompt and permanent cessation of the bleeding.

The potential role of angiography in patients with intractable epistaxis is discussed and the possible use of vasoconstrictor drugs infused at the time of angiography in an attempt to control the hemorrhage is considered.

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REFERENCES

1. Ballenger, J. H. Diseases of the Nose, Throat and Ear. Lea & Febiger, Philadelphia, 1969.
2. Baum, S., Nusbaum, M., Blakemore, W. S., and Finklestein, A. K. Preoperative radiographic demonstration of intra-abdominal bleeding from undetermined sites by percu-

taneous selective celiac and superior mesenteric arteriography. *Surgery*, 1965, *58*, 797–805.

3. BAUM, S., and NUSBAUM, M. Control of gastrointestinal hemorrhage by selective mesenteric arterial infusion of vasopressin. *Radiology*, 1971, *98*, 497–505.
4. BOIJSEN, E., and REUTER, S. R. Angiography in diagnosis of chronic unexplained melena. *Radiology*, 1967, *89*, 413–419.
5. CHIHAK, R. J., RAINES, R. B., POLLARD, J. J., and FLEISCHLI, D. J. Spontaneous renal bleeding in hydronephrotic solitary kidney: report of case with angiographic demonstration of bleeding site. *Radiology*, 1969, *93*, 1305–1307.
6. CORNELL, S. Angiographic demonstration of acute renal hemorrhage. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *109*, 549–552.
7. DOPPMAN, J., DICHIRO, G., and OMMAYA, A. K. Selective Arteriography of the Spinal Cord. W. H. Green, St. Louis, 1969.
8. DUGGAN, C., and BRYLSKI, J. Angiographic demonstration of bleeding in intractable traumatic epistaxis. *Radiology*, 1970, *97*, 605–606.
9. IMMERGUT, M., and PLOTKIN, A. Hemorrhage following needle biopsy of kidney: diagnosis by renal arteriography. *J.A.M.A.*, 1970, *211*, 827–828.
10. JANON, E. Arteriographic demonstration of spontaneous spinal subarachnoid hemorrhage. *Radiology*, 1970, *97*, 385–386.
11. KITTREDGE, R. D., COLAIACE, W. M., KANICK, V., and FINBY, N. Angiography of hemorrhage. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1969, *107*, 181–190.
12. MARGULIS, A. R., HEINBECKER, P., and BERNARD, H. R. Operative mesenteric arteriography in search for site of bleeding in unexplained gastrointestinal hemorrhage. *Surgery*, 1960, *48*, 534–539.
13. REUTER, S. R., FRY, W. J., and BOOKSTEIN, J. J. Mesenteric artery branch aneurysms. *A.M.A. Arch. Surg.*, 1968, *97*, 497–499.
14. REUTER, S. R., and BOOKSTEIN, J. J. Angiographic localization of gastrointestinal bleeding. *Gastroenterology*, 1968, *54*, 876–883.
15. SAHS, A. L., PERRET, G. E., LOCKSLEY, H. B., and NISHIOKA, H. Intracranial Aneurysms and Subarachnoid Hemorrhage; Cooperative Study. J. B. Lippincott Company, Philadelphia, 1969.

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ANGIOGRAPHIC DEMONSTRATION OF IMPORTANT ANATOMIC VARIATIONS OF THE POSTERIOR DESCENDING CORONARY ARTERY*

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DIAPHRAGMATIC myocardial infarction is one of the more common consequences of coronary artery disease. It can occur as a result of interruption of the blood supply to the posterior diaphragmatic portion of the interventricular septum and/or the diaphragmatic surface of the left ventricle. Previous publications on coronary angiography have paid relatively little attention to the fact that there is considerable variation in the arterial supply of this important region of the heart. Understanding these variations is essential in diagnosing obstructions of these arteries and planning surgical therapy.

NORMAL ANATOMY

The concept of right coronary artery (RCA) or left coronary artery (LCA) preponderance was first proposed by Schlesinger in 1940.[4] The preponderant, or "dominant," artery is the one which supplies the posterior diaphragmatic portion of the interventricular septum and the diaphragmatic surface of the left ventricle.

Between 77 and 90 per cent of human subjects have dominant right coronary arteries,[1,2] an anatomic arrangement illustrated in Figure 1, *A* and *B*. In these individuals, the RCA extends all the way around the right atrioventricular sulcus, passes the crux of the heart (the junction of the right and left atrioventricular sulci and the posterior interventricular sulcus) and then continues toward the diaphragmatic surface of the left ventricle. During its passage along the right atrioventricular sulcus the RCA may give off, among other branches, one or more acute marginal (AM) arteries and one or more posterior right ventricular (PRV) arteries. At or shortly before the crux, a large posterior descending (PD) artery arises and passes forward along the interventricular sulcus for a variable distance toward the apex of the heart. This vessel is the chief source of blood to the posterior diaphragmatic portion of the septum. After passing the crux, the dominant RCA continues a short distance before it terminates by giving rise to one or more large posterior left ventricular (PLV) arteries. The latter vessels, which generally parallel the PD artery, are the chief source of blood to the diaphragmatic surface of the left ventricle. Individuals with right coronary dominance have small left circumflex arteries which do not reach the crux of the heart.

A small percentage of subjects have dominant left coronary arteries. In these individuals, it is the RCA which is small and does not reach the crux. The left circumflex artery is much larger and passes completely around the left atrioventricular sulcus. Along the diaphragmatic surface of the heart, it gives rise to the PLV arteries and then turns towards the apex to continue as the PD artery. This anatomic arrangement is illustrated in Figure 2, *A* and *B*.

Finally, there is another small group of subjects who have neither right nor left dominance, but who instead have a "balanced" coronary circulation. Here the RCA reaches the area of the crux where it turns towards the apex and continues as the PD artery. It does not, however, pass beyond the crux and does not give rise to the PLV arteries. The latter originate instead from the distal portion of the left circumflex artery.

Thus, in right coronary dominance, the

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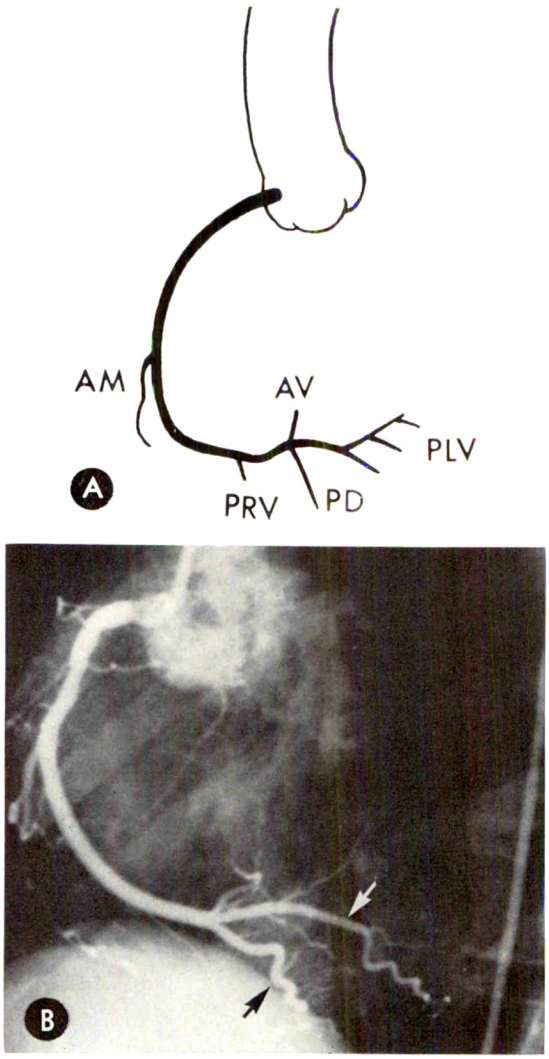


FIG. 1. (*A*) Diagram of a dominant right coronary artery (RCA); left anterior oblique projection. AM = acute marginal artery; AV = atrioventricular node artery; PRV = posterior right ventricular artery; PD = posterior descending artery; PLV = posterior left ventricular arteries. (*B*) Left anterior oblique view of a dominant RCA. The black arrow points to the PD artery and the white arrow to a large PLV artery.

RCA supplies blood to both the posterior diaphragmatic portion of the interventricular septum and the diaphragmatic surface of the left ventricle. In left coronary dominance, both these areas are supplied by the LCA. In a balanced circulation, the posterior diaphragmatic portion of the septum is supplied by the RCA, while the diaphragmatic surface of the left ventricle is supplied by the LCA.

ANATOMIC VARIATIONS

As indicated above, the PLV arteries arise from either the RCA or the LCA. There are generally 1 to 4 of these branches

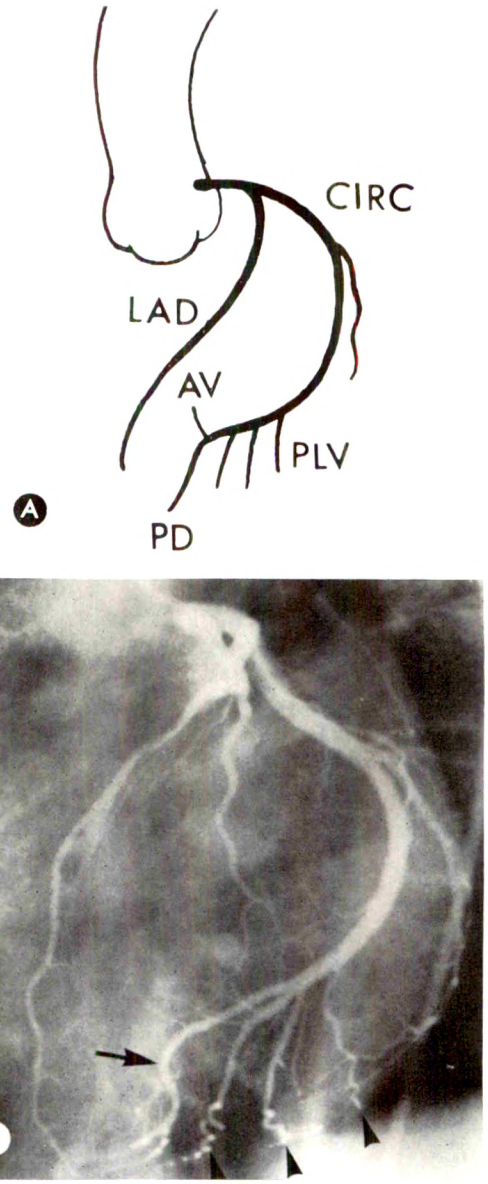


FIG. 2. (*A*) Diagram of a dominant left coronary artery (LCA); left anterior oblique projection. LAD = left anterior descending artery. (*B*) Left anterior oblique view of a dominant left coronary artery. The black arrow points to the PD artery and the black arrowheads to 3 PLV arteries.

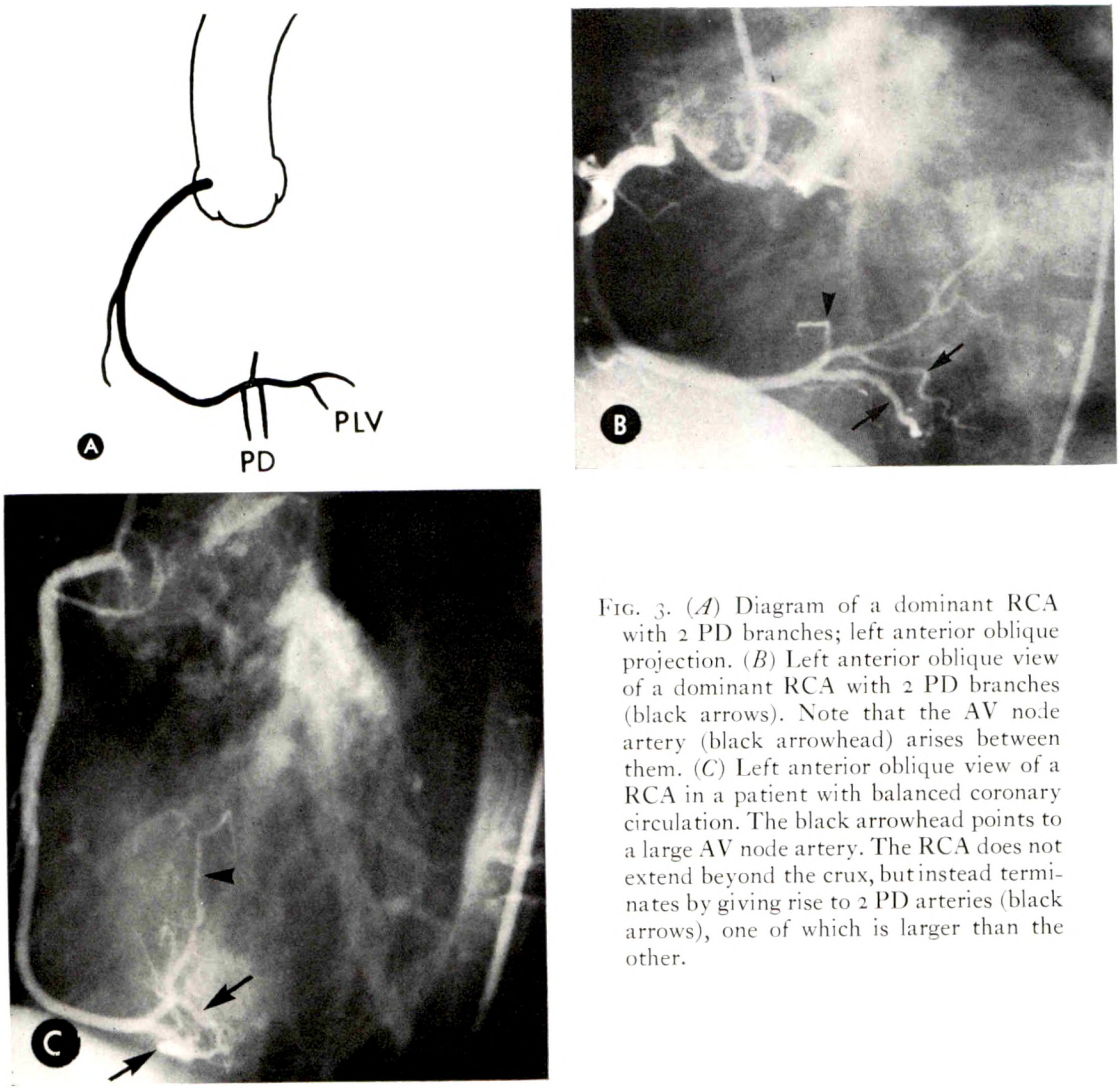


Fig. 3. (*A*) Diagram of a dominant RCA with 2 PD branches; left anterior oblique projection. (*B*) Left anterior oblique view of a dominant RCA with 2 PD branches (black arrows). Note that the AV node artery (black arrowhead) arises between them. (*C*) Left anterior oblique view of a RCA in a patient with balanced coronary circulation. The black arrowhead points to a large AV node artery. The RCA does not extend beyond the crux, but instead terminates by giving rise to 2 PD arteries (black arrows), one of which is larger than the other.

present. Variations of these left ventricular arteries are otherwise rare.

On the other hand, we have found significant anatomic variations in the origin, course and distribution of the PD artery in 23 per cent of 200 recently reviewed coronary angiograms. These variations are discussed below and the incidence of each is indicated. The individual percentage figures total slightly in excess of 23 per cent, because several cases demonstrated a combination of variations.

A. *Double posterior descending artery* (6 per cent). The PD artery is commonly thought of as a single large trunk occupying the posterior interventricular sulcus. On occasion, however, two parallel PD branches may be present—one on either side of the sulcus. These vessels may be of the same or different size and both will supply branches to the posterior septum. Figure 3, *A–C*, shows examples of double PD arteries. At times it may be difficult to decide whether an artery in this area is a PD or a nearby PLV branch since, as indicated earlier, these vessels generally pursue a parallel course and are often quite close to each other.

B. *Early origin of the posterior descending artery* (5 per cent). Instead of arising at or

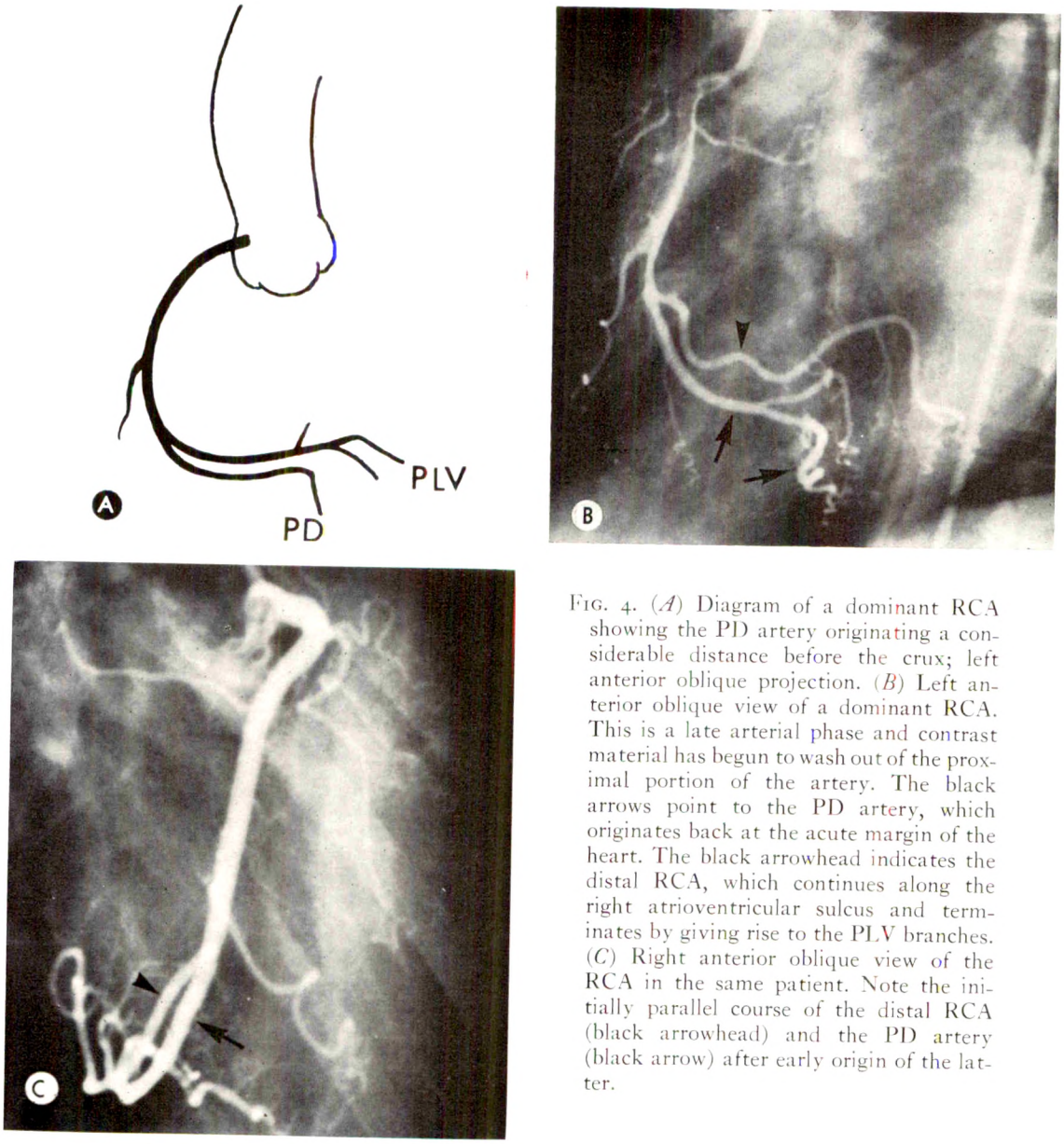


FIG. 4. (*A*) Diagram of a dominant RCA showing the PD artery originating a considerable distance before the crux; left anterior oblique projection. (*B*) Left anterior oblique view of a dominant RCA. This is a late arterial phase and contrast material has begun to wash out of the proximal portion of the artery. The black arrows point to the PD artery, which originates back at the acute margin of the heart. The black arrowhead indicates the distal RCA, which continues along the right atrioventricular sulcus and terminates by giving rise to the PLV branches. (*C*) Right anterior oblique view of the RCA in the same patient. Note the initially parallel course of the distal RCA (black arrowhead) and the PD artery (black arrow) after early origin of the latter.

just before the crux of the heart, the PD artery may arise from the RCA 3 or 4 cm. proximal to the crux. It then continues parallel to the RCA just in front of the right atrioventricular suclus until it reaches the interventricular sulcus. At that point, it turns 90° and thereafter passes forward toward the cardiac apex in the usual manner. Figure 4, *A–C* illustrates this variant.

C. *Partial blood supply of the diaphragmatic portion of the interventricular septum by a posterior right ventricular branch* (8 per cent). The PRV branches of the RCA are inconstant and may not be visualized at all in some patients. On occasion, however, as shown in Figure 5, *A* and *B*, a large PRV branch may arise from the RCA and pass obliquely across the inferior surface of the right ventricle to supply the mid-diaphragmatic portion of the interventricular sep-

tum. In some of these cases, the PD artery itself may be small and only pass a short distance along the posterior interventricular sulcus. The PRV artery is in effect supplying a portion of the septum ordinarily supplied by the PD artery.

D. *Partial blood supply of the diaphragmatic portion of the interventricular septum by an acute marginal artery* (7 per cent). In this variation, shown in Figure 6, *A–F*, the PD artery again can be relatively small and extend only a short distance forward from the crux. The mid-diaphragmatic portion of the interventricular septum is supplied by a large acute marginal (AM) branch. The latter originates from the RCA at the right, or acute, margin of the heart. Initially it passes anteriorly along the lateral right ventricular wall, then turns obliquely beneath the inferior surface of the right ventricle and finally terminates along the septum.

E. *Origin of the posterior descending from the left anterior descending artery*. An example of this rare variant has been demonstrated by Baroldi and Scomazzoni.[1] Here, the left anterior descending artery passes around the cardiac apex and continues back along the posterior interventricular sulcus all the way to the crux. In effect, the entire posterior septal circulation is thus derived from the left anterior descending artery. This variation is diagramed in Figure 7. We have not seen cases of this type in our material.

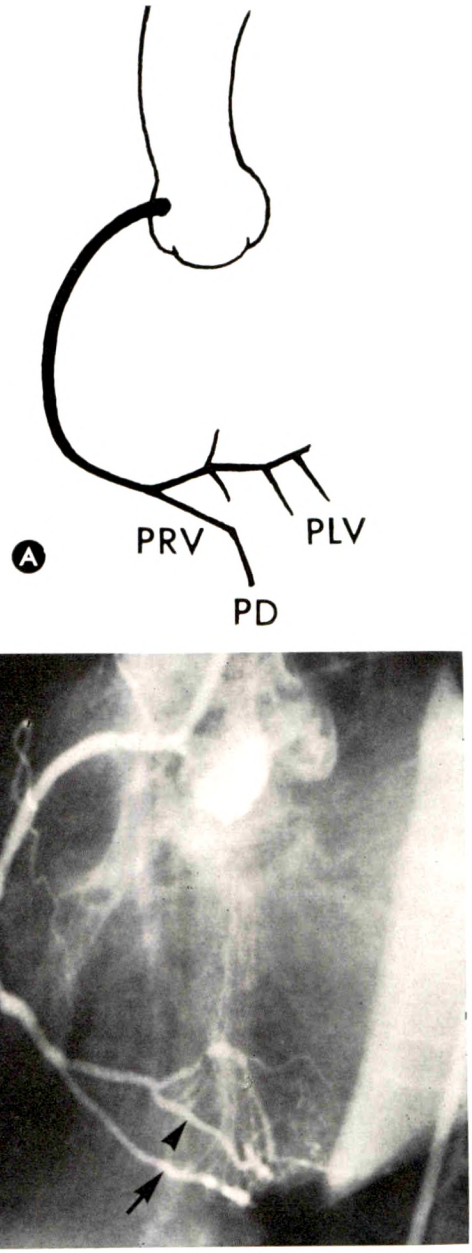
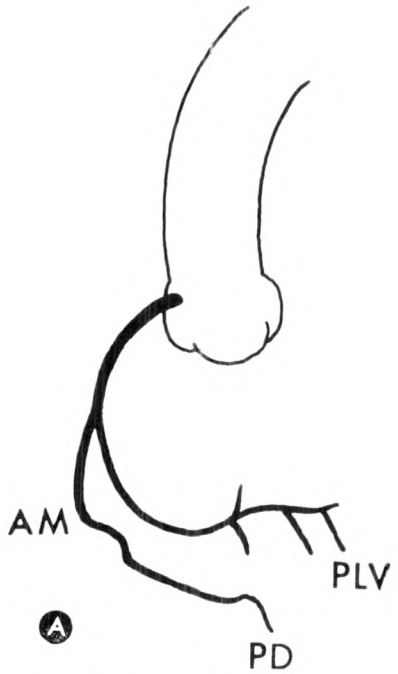
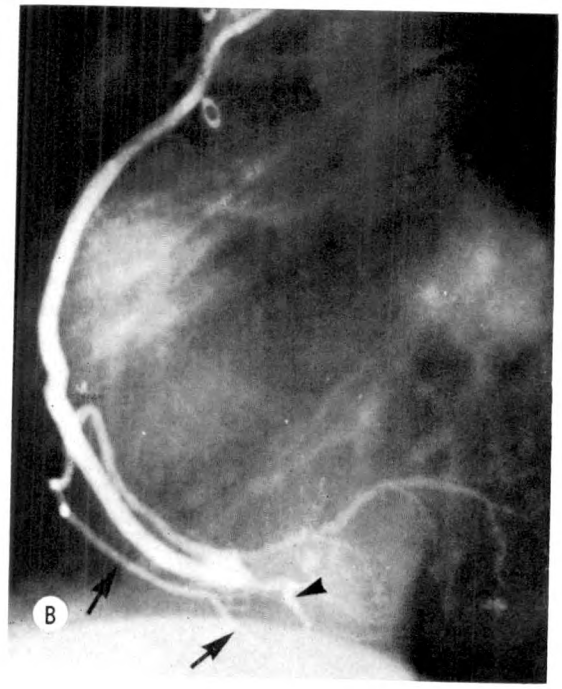


Fig. 5. (*A*) Diagram of a dominant RCA; left anterior oblique projection. There is a small PD branch arising in the usual position and a larger PD branch arising from a PRV artery. (*B*) Left anterior oblique view of a dominant RCA. The black arrowhead points to a PD artery arising in the usual position. The black arrow points to a PRV artery which passes beneath the right ventricle and ends by supplying the mid-diaphragmatic portion of the interventricular septum. Small septal branches are seen arising from both these vessels.

DISCUSSION

Proper interpretation of coronary arteriograms requires a thorough understanding of these important variations in the blood supply of the posterior diaphragmatic portion of the interventricular septum. Although obstructions of a large PD artery or the distal RCA generally are easily discerned through familiarity with the normal anatomy, complete occlusion at the origin of AM, PRV or accessory PD branches which supply this area may be easily overlooked. It is sometimes necessary to rely





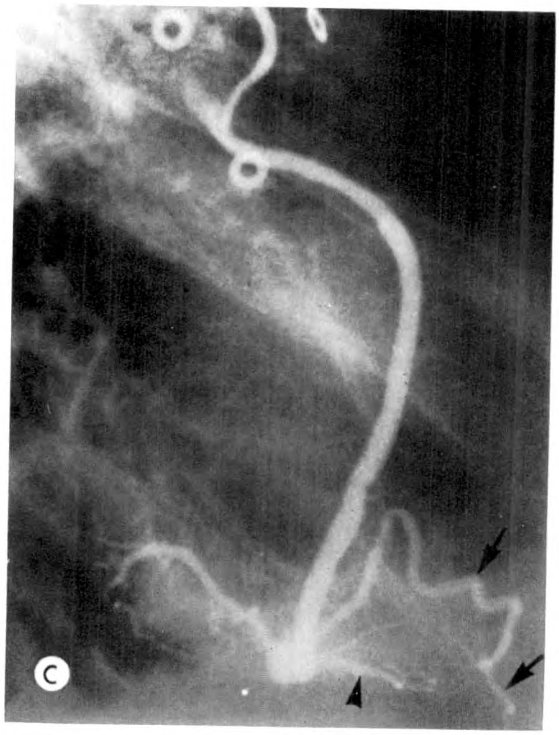


FIG. 6. (*A*) Diagram of a dominant RCA; left anterior oblique projection. There is a small PD branch arising in the usual position and a larger PD branch arising from the AM artery. (*B*) Left anterior oblique view and (*C*) right anterior oblique view of an aorto-right coronary bypass in a patient with occlusion of the proximal RCA. There is a large AM branch (black arrows) which first passes anteriorly, then turns under the right ventricle to supply the septal region. The black arrowhead points to a relatively short PD branch arising in the normal position.

upon visualization of a small stump at the origin, late filling of the vessel through collaterals, or the presence of a localized ventricular aneurysm to signify an obstruction of one of these variant arteries. By the same token, one should not overdiagnose obstruction of a coronary artery where no lesion actually exists. Lack of visualization of a large PD branch arising from the distal RCA at the crux of the heart does not

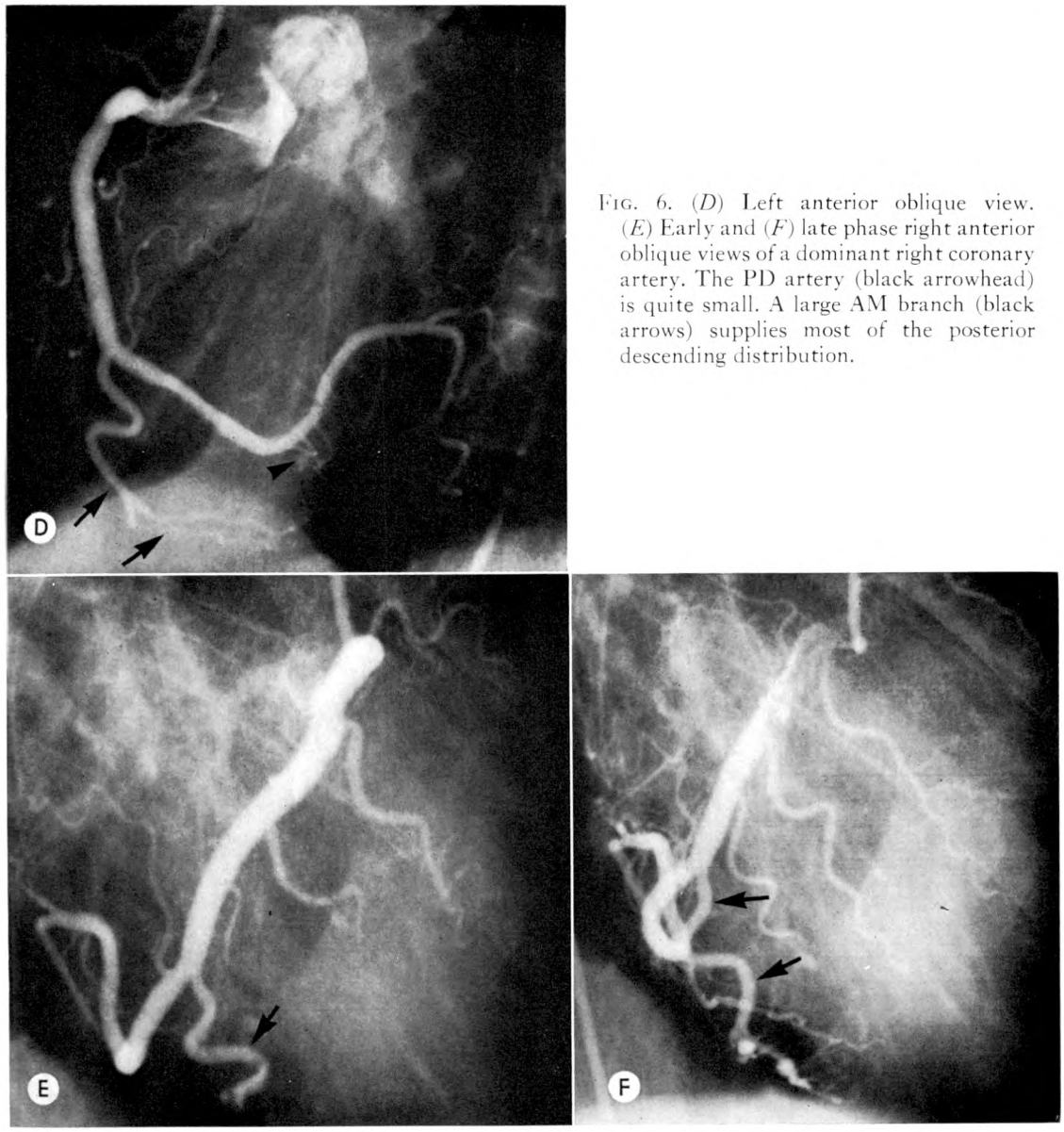


Fig. 6. (*D*) Left anterior oblique view. (*E*) Early and (*F*) late phase right anterior oblique views of a dominant right coronary artery. The PD artery (black arrowhead) is quite small. A large AM branch (black arrows) supplies most of the posterior descending distribution.

necessarily indicate an obstructing lesion. The PD branch may originate some distance proximal to the crux or may in reality be a very small vessel with most of the posterior septal supply coming from a PRV or AM artery.

In cases where patients are being considered for right coronary bypasses, it is particularly important to call the attention of the surgeon to the presence of any of these variations. It is often difficult at the operating table to properly identify arteries at the base of the heart, even under the best of circumstances. Where these vessels do not conform to classical anatomic conceptions, it may be impossible, unless one is forewarned as to the atypical configuration. In addition, these variations affect the decision as to the nature of the bypasses to be constructed. It may be of little value, for instance, to bypass a lesion of the distal RCA or PD artery if most of the posterior interventricular septum is supplied by an AM or PRV branch which originates prox-

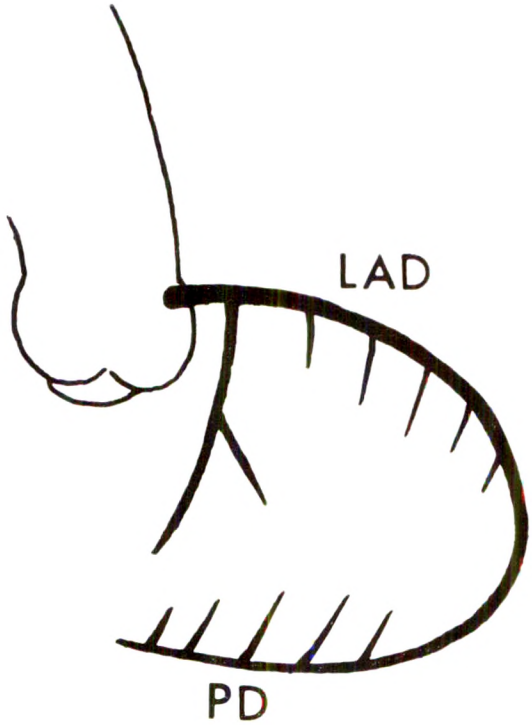


FIG. 7. Diagram of a LCA in which the PD artery arises from the LAD artery; right anterior oblique projection.

imal to the obstruction. This situation is exemplified by Figure 8, *A* and *B*. In other cases, where there is a dual supply of the posterior interventricular septum, multiple stenoses might necessitate construction of 2 different bypasses to provide adequate revascularization. An example of such a case is shown in Figure 9.

SUMMARY

One of the most important branches of the right coronary artery in most patients is the posterior descending artery, which

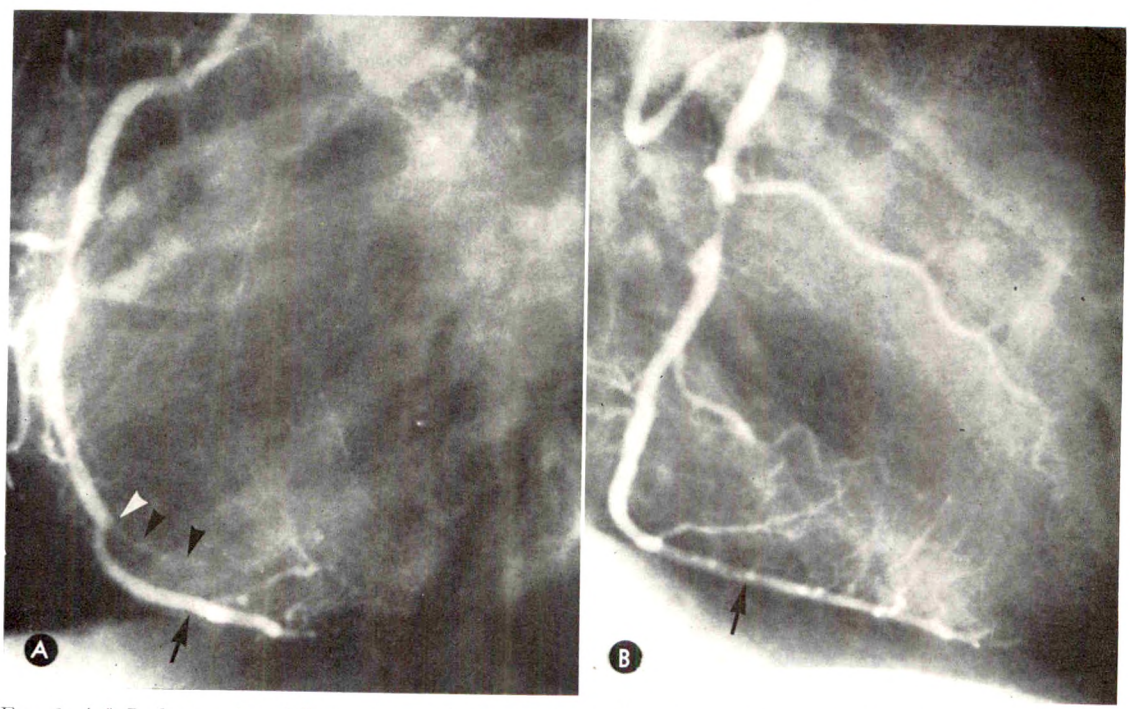


FIG. 8. (*A*) Left anterior oblique view and (*B*) right anterior oblique view of the RCA in a patient with balanced coronary circulation. There is stenosis proximally and nearly complete obstruction distally (white arrowhead). Beyond this point the RCA itself (black arrowheads) is severely narrowed. Fortunately for the patient, the posterior interventricular septum is supplied by a PRV branch (black arrows) which originates just before the distal obstruction. This obviated the necessity of revascularizing the severely diseased distal RCA. A bypass was attached instead to the PRV branch along the inferior aspect of the right ventricle.

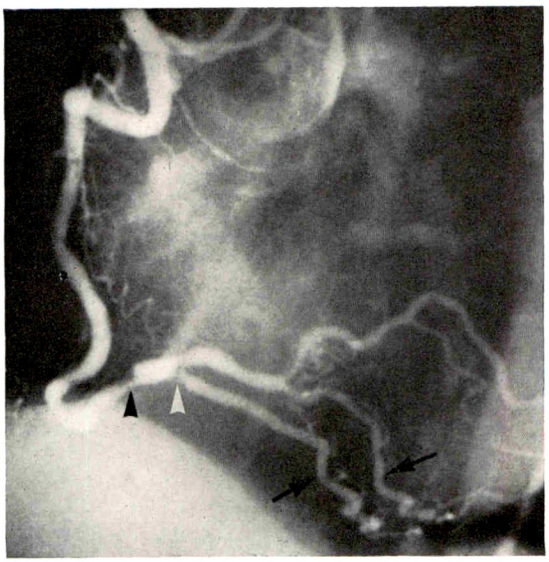


Fig. 9. Left anterior oblique view of a dominant RCA. This patient exhibits a combination of variations. There are 2 PD arteries (black arrows), the first of which has an early origin 3 cm. proximal to the crux. There is a severe stenosis of the RCA (black arrowhead) and also of the first PD artery (white arrowhead). To completely revascularize the posterior interventricular septum in this case, 2 separate bypasses would have to be constructed —one attached to the first PD branch and the other to either the distal RCA or the second PD branch.

supplies blood to the posterior diaphragmatic portion of the interventricular septum.

Significant anatomic variations were found in the origin, course or distribution of this vessel in 23 per cent of a series of 200 patients.

These included double posterior descending branches, early origin of the posterior descending artery and partial supply of the diaphragmatic portion of the septum by acute marginal or posterior right ventricular branches. These variations are described and illustrated angiographically.

Their importance in proper interpretation of coronary arteriograms and planning of bypass procedures is stressed.

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REFERENCES

1. Baroldi, G., and Scomazzoni, G. Coronary Circulation in the Normal and the Pathologic Heart. Office of Surgeon General, Washington, D.C., 1967.
2. James, T. N. Anatomy of the Coronary Arteries. Paul B. Hoeber, Inc., New York, 1961.
3. Paulin, S. Coronary angiography: technical, anatomic and clinical study. *Acta radiol.*, 1964, Suppl. 233, 5–215.
4. Schlesinger, M. J. Relation of anatomic pattern to pathologic conditions of coronary arteries. *Arch. Path.*, 1940, *30*, 403–415.

RADIOGRAPHIC ANATOMY OF THE CORONARY COLLATERAL CIRCULATION*

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THE purpose of this study is to describe the preferential pathways of the *in vivo* coronary collateral circulation by high resolution large film coronary angiography and cineangiography.

The topographic anatomy of the coronary arteries (and their anastomoses) has been extensively investigated in postmortem injection and dissection studies, and the physiologic importance of collateral flow has been evaluated with various techniques. There is general agreement that anastomoses between the coronary arteries are present in normal hearts[1–3,6,10–12,15,17–19,21] and their size has been calculated to be from 20–350 μ. Vastesaeger *et al.*[21] showed that the smallest vessel demonstrable by optimal technique in postmortem studies has a diameter of 50 μ.

The coronary circulation has been studied radiographically with special emphasis on the collateral channels by Paulin,[16] Gensini and da Costa,[8] Helfant *et al.*,[9] and Cosbey *et al.*,[5] using various techniques and equipment. Paulin's studies were done with injection of contrast material into the aortic root, using large films and stereo x-ray tubes. Gensini and Buonanno,[7] and Helfant *et al.*[9] used Sones' selective injection technique with 35 mm. cineangiography and 16 mm. cineangiography, respectively. According to Gensini and Buonanno,[7] and Gensini and da Costa[8] vessels as small as 100 μ are demonstrable by cineradiographic techniques.

Because the power of resolution of the screen film system is 4 times[14] that of the available image intensifiers, we believe that with a rapid serial film angiographic technique it is possible to visualize vessels 100 μ in diameter.

METHOD OF EXAMINATION

One hundred coronary arteriograms were studied. The examinations were selected in chronologic order and only 10 had to be discarded because of suboptimal technique. All coronary arteriograms were obtained with the Judkins[14] selective technique, using the percutaneous femoral artery approach, and occasionally the percutaneous brachial approach.

The end hole, preformed Judkins catheter assures a consistent truly selective coronary injection allowing better opacification of the coronary artery and increasing the possibility of visualization of small collateral channels. Both cine and direct rapid filming was routinely performed in every patient.

Cineangiography was done in the left anterior oblique and right anterior oblique projections with 35 mm. double X-Kodak film. Large films were exposed in the vertical plane of an Elema roll film changer using the horizontal x-ray tube with a 1.2 mm. focal spot, exposure time 4 to 9 msec., 1,000 ma. and 80 to 95 kvp. Examinations were made in the left anterior oblique, right anterior oblique, and lateral positions. To diminish the secondary radiation in addition to the collimator (Collimaster II) a cone and a parallel Lysholm grid (10 to 1) were used. To visualize small collateral vessels we employed 2 film-screen combinations: one, a medium speed high contrast film (Cronex 4) with a fast screen (DuPont High Plus) gives the best resolution; and another, a medium speed film with a wide latitude of contrast film (Cronex 6) combined with an ultrafast screen (Lightning Plus DuPont) allowed reduction of exposure time by 50 per cent.[22]

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Using all three projections of the large film series, the degree of vessel narrowing was measured in the most stenotic portion. The percentage was calculated in relation to the normal width immediately above the stenosis.

Only those collateral vessels were listed as present which could be definitely identified; indirect evidence was not accepted.

MATERIAL AND RESULTS

One hundred consecutive arteriograms of patients with coronary artery disease were reviewed. In 17 cases there were abnormalities of only 1 major coronary artery; in 36 cases 2 major coronary arteries were abnormal. The left anterior descending artery (LAD) and the left circumflex artery (Cx) were involved in 5 patients, the LAD and right coronary artery (RCA) were diseased in 18 cases, and the RCA and the Cx were abnormal in 13 cases. In 47 of the total 100 cases, all 3 major coronary arteries showed stenotic areas. In 9 of these, the left marginal artery of the obtuse angle was involved (Table 1).

1. LEFT ANTERIOR DESCENDING ARTERY (LAD)

Stenosis of the LAD was identified in 11 cases of 1-artery disease, in 23 cases of 2-artery disease, and in 46 cases of 3-artery disease. A single site of obstruction was present in 9 of the cases with 1-artery, in 18 with 2-artery, and in 24 with 3-artery disease. In 8 patients with 1-artery disease, the degree of stenosis exceeded 50 per cent. In 5 of these, there was a complete occlusion. Of the cases with 2-artery disease, there were 21 with a stenosis over 50 per cent and 11 with complete occlusion. In 38 of the 3-artery disease group, the stenosis exceeded 50 per cent and in 15 complete occlusions were observed.

(a) *Septal collaterals:* Examples of collateral flow through the septum from the posterior descending artery to the left anterior descending artery are illustrated in Figure 4, *A–D*, and the distribution of the 37 cases having septal anastomoses is detailed in Table 1.

(b) *Apical collaterals:* Anastomoses of the epicardial branches of the posterior descending artery over the apex of the heart to the LAD could be demonstrated in 28 cases.

(c) *Left marginal anastomoses to the diagonal branches:* Marginal branches of the left circumflex artery were seen to anastomose with the diagonal branches of the left anterior descending artery in 16 cases.

(d) *Anastomoses* from the right marginal of the acute angle with the left anterior descending artery were ob-

TABLE I

ANASTOMOSES TO THE LEFT ANTERIOR DESCENDING ARTERY

	1-Artery Disease	2-Artery Disease	3-Artery Disease	Total
Number of cases	11	23	47	81
Number of cases with more than 50 per cent occlusion	8	21	38	67
Number of cases with complete occlusion	5	11	15	31
Septal anastomoses	7	14	16	37
Apical anastomoses	6	9	13	28
Anastomoses of the left marginal circumflex to LAD	3	5	8	16
Anastomoses of the right marginal circumflex to LAD	0	2	2	4
Vieussen's circle	2	1	8	11
Intracoronary anastomoses across the occlusion	0	1	1	2

LAD = left anterior descending artery.

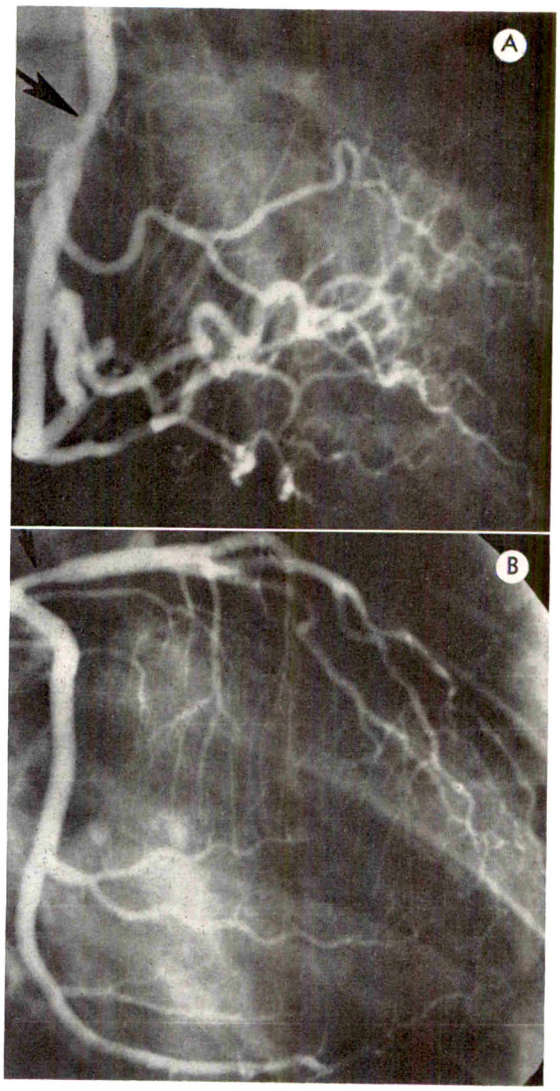


FIG. 1. (*A*) Normal septal branches from the right posterior descending artery in a patient without lesions of the left coronary system. Stenosis of the right coronary artery (arrow). (*B*) Normal septal branches from the left anterior descending artery in a patient without lesions of the right coronary system. Stenosis of the left anterior descending coronary artery (arrow, left upper corner).

served in 4 cases (Fig. 2, *A–D*; and 3, *A–D*).

(e) *Vieussen's circle (VC):* Anastomotic channels from the conus artery supplied the post-stenotic area of the left anterior descending artery in 11 cases (Fig. 2, *A–D*; 3, *A–D*; and 5, *A* and *B*).

(f) *Anastomoses* between diagonal branches and the post-stenotic left anterior descending artery were identified in 5 cases.

(g) *Intracoronary collaterals* effectively bridging the stenosis were noted in 2 cases (Fig. 5, *A* and *B*; and 6, *A–D*).

2. RIGHT CORONARY ARTERY (RCA)

Stenosis of the RCA was identified in 84 cases: 6 cases of 1-artery disease; 31 cases of 2-artery disease; and in 47 cases of 3-artery disease. A single site of stenosis was present in 4 cases of 1-artery disease, in 24 cases of 2-artery, and in 23 cases of 3-artery disease. The RCA was occluded in 47 of the 68 cases having greater than 50 per cent stenosis.

(a) *Septal anastomoses:* Collateral blood flow through the septum from the LAD to the RCA was detected in 53 cases.

(b) *Apical anastomoses* from the LAD to the posterior descending artery over the apex were present in 45 cases (Fig. 8, *A* and *B*).

(c) *Left circumflex artery* anastomoses with the distal RCA in the atrioventricular sulcus were observed in 27 cases. Anastomoses of a branch of the atrial circumflex artery with the distal RCA could be identified in 11 cases (Fig. 6, *A–D*; and 8, *A* and *B*).

(d) *Anastomoses of the left marginal* with the posterior descending artery occurred in 5 cases. These anastomoses extended over the free ventricular wall but they were mainly seen to run over the apex of the heart in a similar way as those from the LAD.

(e) *Anastomoses of the sinus node artery with the RCA* over the posterior wall of the atria in the region of the crux were present in 12 cases.

(f) *Kugel's arteria anastomotica auricularia magna:* This vessel, extensively described by Kugel in 1928 and by James[11] could be demonstrated in 7 cases. By means of an experimental model we made sure that the artery

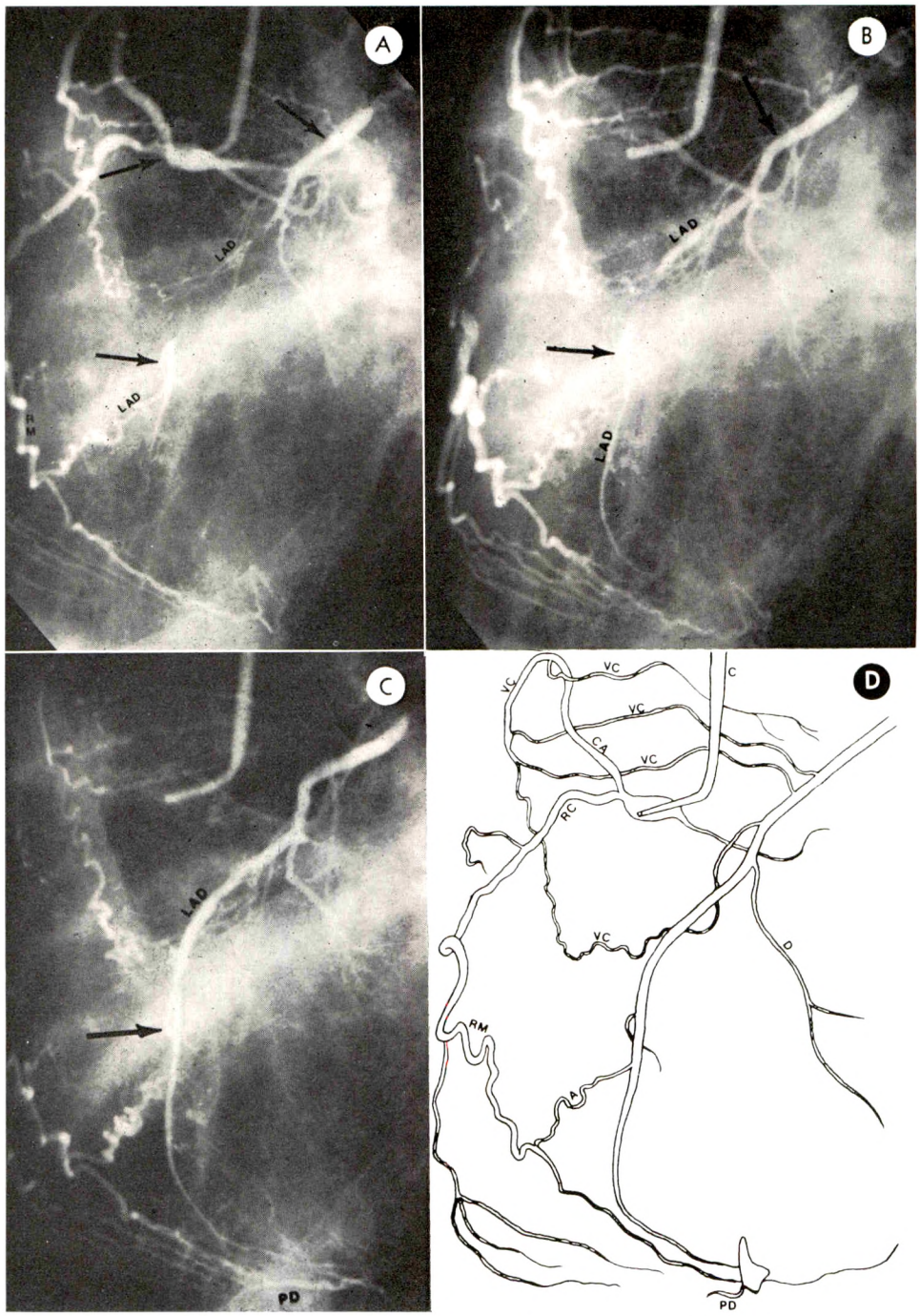


FIG. 2. Right coronary injection to demonstrate collateral channels in occlusion of the left anterior descending coronary artery. (*A*) Early filling by retrograde flow of proximal and middle segments of left anterior descending artery. (*B* and *C*) Progressive filling with complete visualization of the post-stenotic left anterior descending artery. (*D*) Schematic drawing.

Key: C = catheter; VC = Vieussen's arterial circle; CA = conus artery; RC = right coronary; RM = right marginal; PD = posterior descending; LAD = left anterior descending; and D = diagonal branch. The same abbreviations will be used in the following figures.

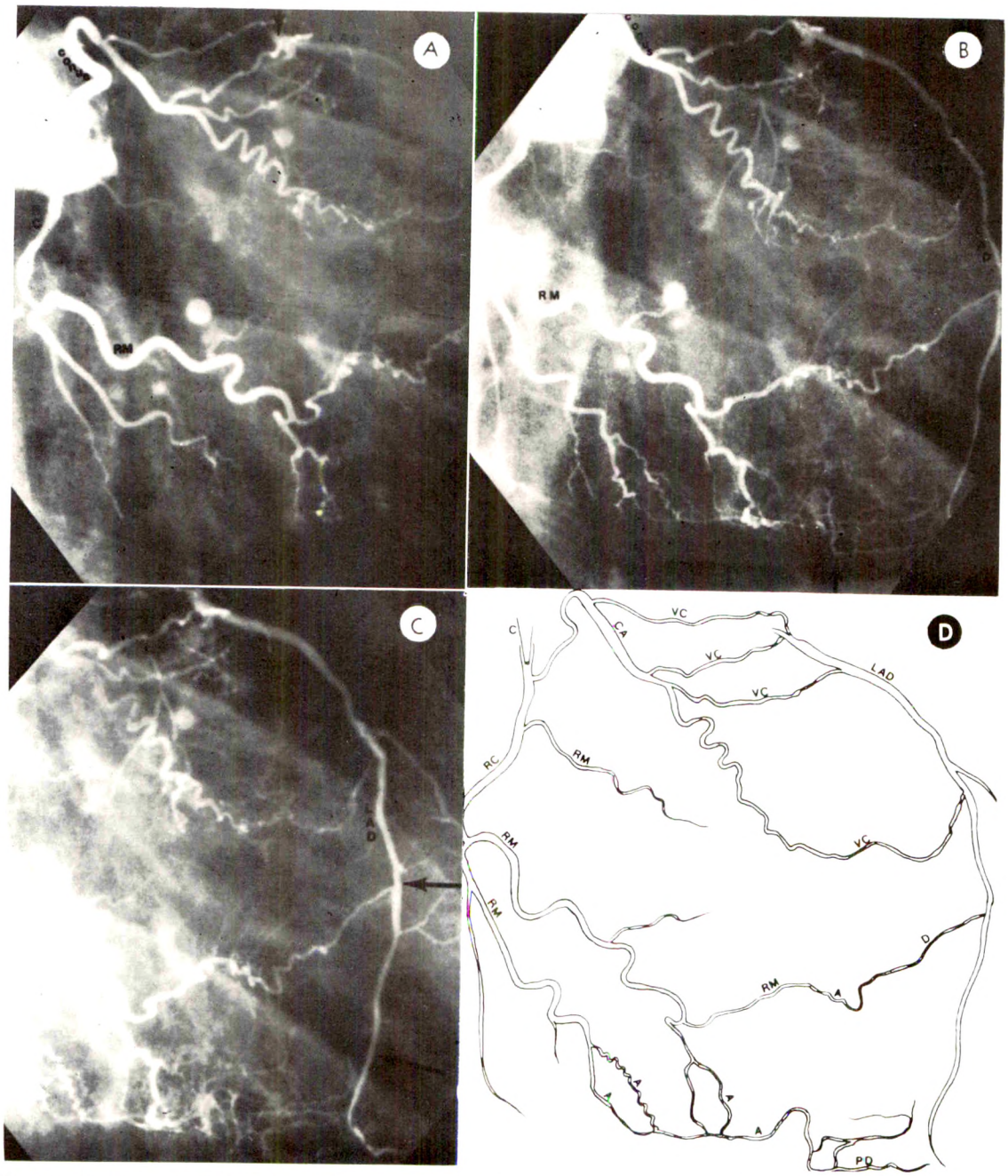


FIG. 3. Right coronary injection of the same case as in Figure 2 in right anterior oblique projection. (*A*) Anastomosis between left and right coronary arteries in the ventricular wall. Arrows in *A* and *C* point to the filling of the left anterior descending, and in *B* to the posterior descending artery. (*D*) Schematic drawing.

identified was indeed the artery described by Kugel, connecting the proximal RCA with the distal RCA.[13]

(g) *Conus artery anastomoses to the right marginal branches:* Intracoronary connections between the ventricular branches of the conus artery and the marginal branches of the RCA on the right ventricle could be seen in 5 cases.

(h) *Intracoronary anastomoses* of the

right coronary artery were identified in 18 cases.

3. LEFT CIRCUMFLEX ARTERY

In none of the 17 cases with 1-artery disease was the left circumflex artery involved. However, there was stenosis in this vessel in 65 cases: in 18 cases of 2-artery disease; and in 47 cases of 3-artery disease. A single site of stenosis was present in 17

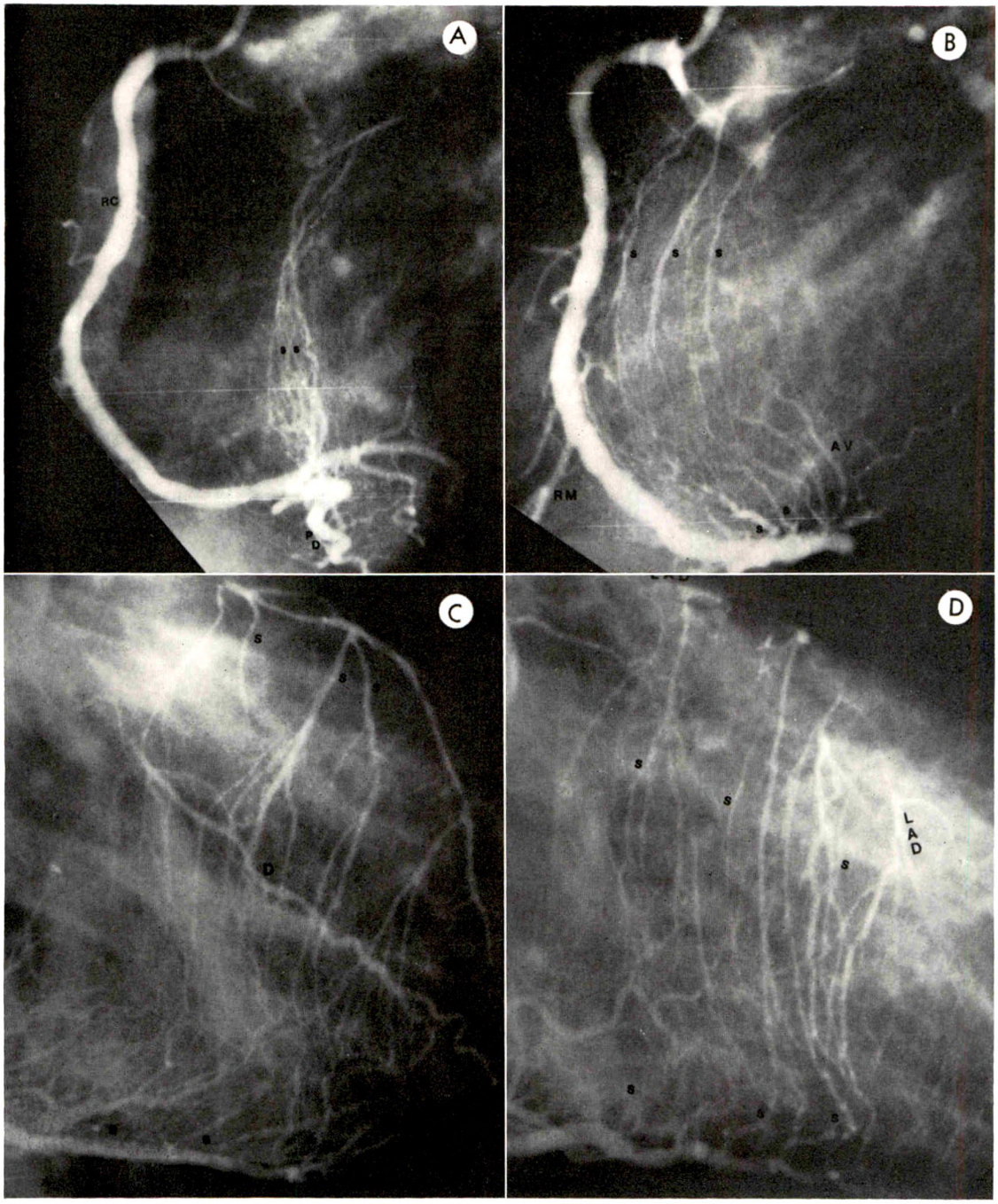


FIG. 4. Septal collaterals (S) in occlusion of the left anterior descending artery in left anterior oblique (*A*), lateral (*B*) and right anterior oblique (*C*) projections. Right anterior oblique projection (*D*) in a different patient.

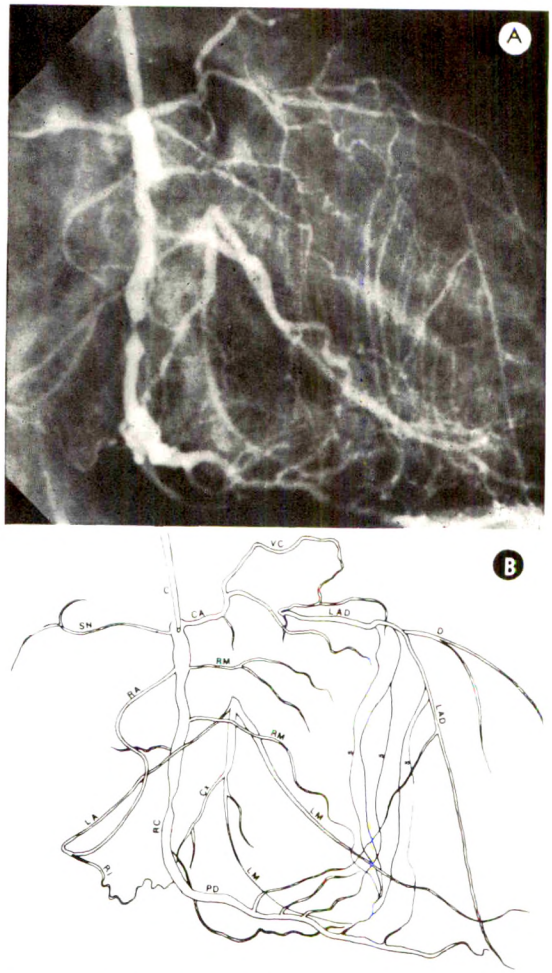


FIG. 5. (*A* and *B*) Right coronary artery injection with visualization of the post-stenotic left circumflex artery by atrial anastomoses and opacification of the left anterior descending artery by Vieussen's circle and septal branches.

Key: SN = sinus node artery; RA = right atrial branch; LA = left atrial branch from the left circumflex artery; RI = right intermediate artery; LM = left marginal artery.

cases with 2-artery, and in 26 cases with 3-artery disease. The Cx was occluded in 29 cases and in 46 cases the stenosis was greater than 50 per cent.

(a) *Distal RCA anastomosis to the left circumflex artery:* Retrograde filling of the left circumflex artery through anastomoses from the distal RCA occurred in 7 cases.

(b) *A direct communication of a branch of the left atrial circumflex artery* could be observed in 23 cases (Fig. 6, *A–D*).

(c) *Anastomoses from the diagonal branches of the LAD to the left circumflex artery:* Epicardial branches connecting the diagonal ventricular branches from the LAD with the marginal branches of the left circumflex artery and supplying this vessel by retrograde flow could be seen in 5 cases.

(d) *Intracoronary anastomoses*, bridging the occlusion of the left circumflex artery, were identified in 3 cases.

DISCUSSION

All collateral pathways detected in postmortem studies could be observed in our coronary angiograms. Some were more frequently visualized than others. With the radiographic technique used we were able to observe normal septal branches from the left anterior descending and right posterior descending arteries without ultimate anastomosis between left and right systems (Fig. 1, *A* and *B*). The fact that collateral channels are not seen in normal hearts is in contrast to the pathologic anatomic findings of many investigators. No explanation is known, unless one accepts the theory that these collaterals are indeed nonfunctioning in the normal heart.[9,18,24]

Collateral circulation is usually not identified radiologically, even in arteries affected with stenosis of less than 75 per cent. Only in patients with multiple stenoses or diffuse atherosclerotic involvement will anastomosis be seen, and there is a definite correlation between the degree of the stenosis, the number of arteries involved, and the number and location of the collateral channels.[5,8,9,16] Bidirectional flow was found in 6 cases with marked stenosis of the LAD and RCA without occlusion. With an injection into the left coronary artery, the posterior descending artery showed retrograde filling and with an injection into the RCA, retrograde flow occurred in the LAD. Such a mechanism should be able to prevent the acute changes otherwise seen in complete occlusion of 1 of the 2 vessels involved. The major importance of the septal anastomoses for the collateral flow between the left and right coronary artery is well

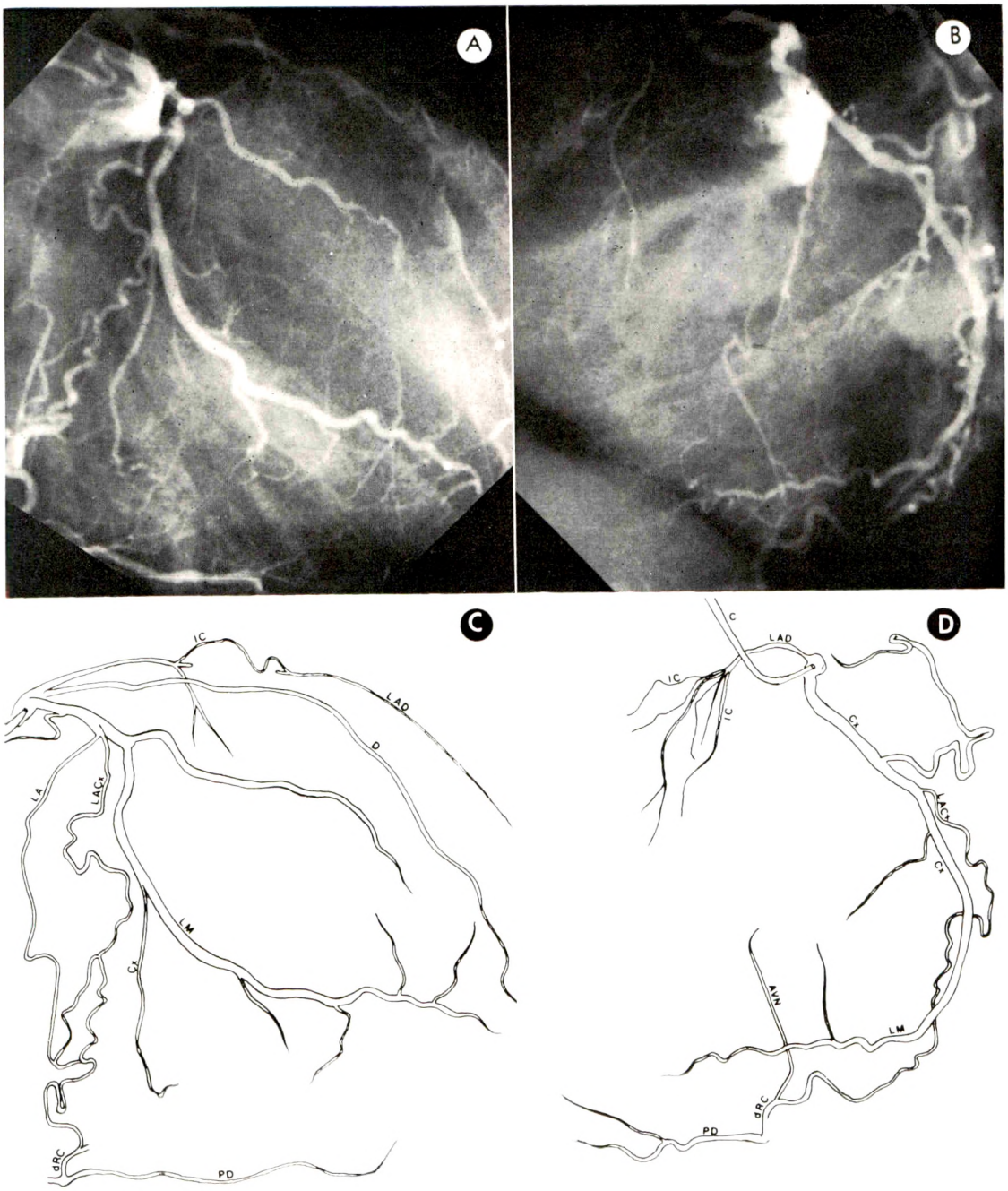


Fig. 6. (*A–D*) Left coronary artery injection with retrograde visualization of the distal right and posterior descending artery from the left atrial circumflex artery.

Key: LA = left atrial branch; LACx = left atrial circumflex artery; dRC = distal right coronary artery; AVN = atrioventricular node artery; IC = intracoronary anastomoses of the occluded left anterior descending artery. (*A* and *C*) Right anterior oblique projection. (*B* and *D*) Left anterior oblique projection.

known.[15,19] We found in the 3-artery disease group a greater tendency of the collateral flow to occur from the left anterior descending to the posterior descending artery than in the opposite direction (Tables II and III). This may be due to the fact that the septal branches of the right coronary artery occur distally, whereas the septal branches from

TABLE II

ANASTOMOSES TO THE RIGHT CORONARY ARTERY

	1-Artery Disease	2-Artery Disease	3-Artery Disease	Total
Number of cases	6	31	47	84
Number of cases with more than 50 per cent occlusion	3	25	40	68
Number of cases with complete occlusion	2	19	26	47
Septal anastomoses	2	19	32	53
Apical anastomoses	2	13	30	45
Left circumflex anastomoses with distal RCA	1	10	16	27
Atrial circumflex anastomoses with distal RCA	0	3	8	11
Right marginal anastomoses with posterior descending	0	2	11	13
Left marginal anastomoses with posterior descending	0	2	3	5
Sinus node artery anastomoses with distal RCA	1	2	9	12
Kugel's artery	0	2	5	7
Conus artery anastomoses with the right marginal branches	0	1	4	5
Intercoronary anastomoses across occlusion	0	6	12	18

RCA = right coronary artery.

the left anterior descending artery originate more proximally. In cases of complete occlusion of 1 major artery, septal and epicardial anastomoses over the apex are invariably seen. The frequency of visualization decreased with a lesser degree of stenosis. Our data suggest that when septal anastomoses are present, collateral flow over the apex will be present also. The septal and apical collaterals are the most common and most important connection between the left and right coronary arteries (Fig. 2, *A–D*; 5, *A* and *B*; 6, *A–D*; and 9). Vieussen's arterial ring[11–20] and anastomoses between the right marginal branches and the left anterior descending artery are less frequent (Fig. 2, *A–D*; 3, *A–D*; and 5, *A* and *B*). Intracoronary collaterals which are said to be derived from adventitial arteries or vasa vasorum[12] are not often seen on the left anterior descending artery (Fig. 6, *A–D*; and 7, *A* and *B*) and left circumflex artery. They are common in the right coronary artery. When they are located in the proximal portion of this vessel, they usually are effective in opacifying the post-stenotic area.

The preferential collateral pathways to each artery are diagrammatically shown in Figure 9. In stenosis of the LAD (Table 1), septal and apical collaterals are the most frequent. Anastomoses of the left marginal

TABLE III

ANASTOMOSES TO THE LEFT CIRCUMFLEX ARTERY

	1-Artery Disease	2-Artery Disease	3-Artery Disease	Total
Number of cases	0	18	47	61
Number of cases with more than 50 per cent occlusion	0	12	34	36
Number of cases with complete occlusion	0	8	21	29
Distal RCA anastomoses with left circumflex	0	3	4	7
Atrial circumflex anastomoses with left circumflex	0	7	16	23
Anastomoses between left diagonal and left marginal branches	0	1	4	5
Intercoronary anastomoses across occlusion	0	0	3	3

branches of the left circumflex artery are a common pathway also. Vieussen's arterial circle is present almost as frequently. The right marginal branches infrequently supply blood to the LAD.

In stenosis of the right coronary artery (Table II) again the septal and apical anastomoses are the most frequent. Less common but probably of equal importance are the anastomoses to the distal right coronary artery from the left circumflex artery and its atrial branches (Fig. 8, *A* and *B*). These branches either supply blood directly to the distal right coronary artery or fill the distal left circumflex artery and the blood flows retrograde through the distal right and into the posterior descending artery.

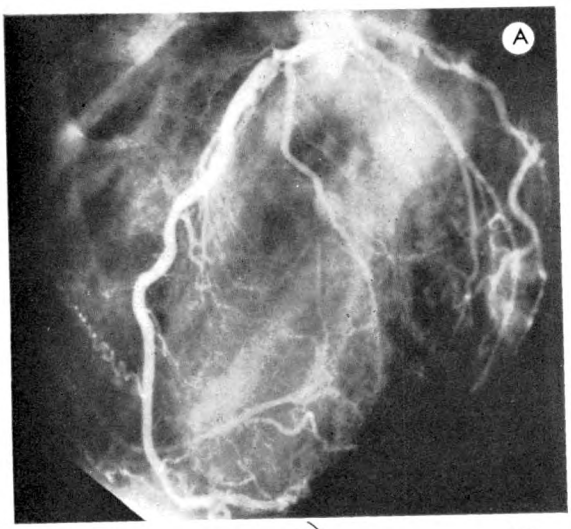


Fig. 7. (*A* and *B*) Left coronary injection (lateral projection) with filling of the right coronary artery by anastomoses through the right ventricular wall.

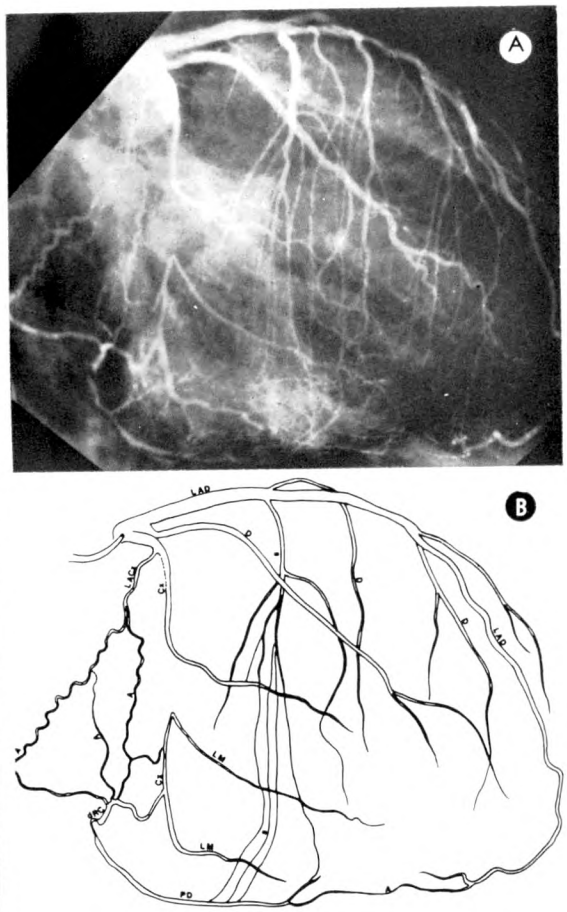


Fig. 8. (*A* and *B*) Left coronary injection in right anterior oblique projection. Occlusion of the left circumflex artery with anastomoses from the left atrial circumflex artery filling the post-stenotic circumflex and distal right coronary arteries is seen. The posterior descending artery is filled through the distal right and from the left anterior descending artery via septal and apical collaterals.

Relatively often seen are the collaterals of the sinus node artery to the distal right coronary artery over the posterior surface of the atria. Kugel's artery,[3,24] although rarely seen, can be very important in the blood supply of the atrioventricular node. The most frequently used pathway of collateral flow to the left circumflex artery is from the RCA through the left atrial circumflex branch.



Fig. 9. Anastomoses of the coronary arteries in order of frequency.

I. Left Anterior Descending Artery: 1. Septal anastomoses; 2. Anastomoses over the apex; 3. Left marginal anastomoses to the LAD; 4. Vieussen's circle; 5. Right marginal anastomoses to the LAD; 6. Intracoronary anastomoses.

II. Right Coronary Artery: 1. Septal anastomoses; 2. Apical anastomoses; 3. Left circumflex anastomoses with distal RC; 4. Atrial circumflex anastomoses with distal RC; 5. Right marginal anastomoses with posterior descending; 6. Left marginal anastomoses with posterior descending; 7. Sinus node artery anastomoses with distal RC; 8. Kugel's artery; 9. Conus artery anastomoses with the right marginal branches; 10. Intracoronary anastomoses across occlusion.

III. Left Circumflex Artery: 1. Anastomoses from the left atrial circumflex to the circumflex; 2. Anasto-

SUMMARY

The selective coronary arteriograms of 100 patients with coronary disease were studied with large films and 35 mm. cinefilming.

The examinations were analyzed for the presence of anastomoses and the preferential pathways of the collateral flow.

We found that all anastomoses described in postmortem studies could be demonstrated with selective coronary arteriography, if a significant stenosis is present, and if proper equipment is used.

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REFERENCES

1. Baroldi, G., Mantero, O., and Scomozoni, G. Collaterals of coronary arteries in normal and pathologic conditions. *Circulation Res.*, 1956, *4*, 223–229.
2. Bellmann, S., and Frank, H. A. Intercoronary collaterals in normal hearts. *J. Thoracic Surg.*, 1958, *36*, 584–603.
3. Bloor, C. M., and Liebow, A. A. Coronary collateral circulation. *Am. J. Cardiol.*, 1965, *16*, 238–252.
4. Bookstein, J. J., and Kahn, D. R. Appraisal of coronary arteriography in evaluating hemodynamic significance of experimental coronary artery stenosis. *Radiology*, 1967, *88*, 672–679.
5. Cosbey, R. S., Giddings, J. A., and See, R. Coronary collateral circulation. Abstracts of 44th Scientific Session of American Heart Association, Anaheim, Calif., November, 1971. *Circulation*, 1971, *44*, II–123.
6. Fulton, W. F. M. The Coronary Arteries. Charles C Thomas, Publisher, Springfield, Ill., 1965.
7. Gensini, G. G., and Buonanno, C. Coronary arteriography. *Dis. Chest*, 1968, *54*, 10–19.
8. Gensini, G. G., and da Costa, C. Coronary collateral circulation in living man. *Am. J. Cardiol.*, 1969, *24*, 393–400.
9. Helfant, R. H., Vokonas, P. S., and Gorlin, R. Functional importance of human coronary collateral circulation. *New England J. Med.*, 1971, *284*, 1277–1281.
10. Hugnet, J., Luccioni, R., Navarro, B., and Collonna, F. Les anastomoses coronariennes. *Ann. radiol. diag.*, 1970, *13*, 651–666.
11. James, T. M. Anatomy of Coronary Arteries. Paul B. Hoeber, Inc., New York, 1961.
12. James, T. M. Delivery and distribution of coronary collateral circulation. *Dis. Chest*, 1970, *58*, 183–203.
13. Jochem, W., Soto, B., Karp, R. B., and Barcia, A. Radiographic anatomy of arteria anastomotica auricularis magna (Kugel's artery). To be published.
14. Judkins, M. P. Percutaneous transfemoral selective coronary arteriography. *Radiol. Clin. North America*, 1968, *6*, 467–492.
15. Laubry, Ch., Soulier, P., and Thys, H. Les anastomoses septales. *Arch. mal. coeur*, 1948, *41*, 1–24.
16. Paulin, S. Interarterial coronary anastomoses in relation to arterial obstruction demonstrated in coronary arteriography. *Invest. Radiol.*, 1967, *2*, 147–159.
17. Prinzmetal, M., Simkin, B., Bergman, H. C., and Kruger, H. E. Studies on collateral circulation: II. Collateral circulation of normal human heart by coronary perfusion with radioactive erythrocytes and glass spheres. *Am. Heart J.*, 1947, *33*, 420–442.
18. Russel, R. Myocardial collateral circulation. *Brit. Heart J.*, 1969, *31*, 1–4.
19. Schlesinger, M. J. Injection plus dissection study of coronary artery occlusions and anastomoses. *Am. Heart J.*, 1938, *15*, 528–568.
20. Schlesinger, M. J., Zoll, P. M., and Wessler, S. Conus artery: third coronary artery. *Am. Heart J.*, 1949, *38*, 823–836.
21. Vastesaeger, M. M., van der Straeten, P. P., Friart, J., Candaele, A., Ghys, R., and Bernard, R. M. Les anastomoses intercoronariennes telles qu'elles apparaissent à la coronariography post-mortem. *Acta cardiol.*, 1957, *12*, 365–401.
22. Wayrynen, R. E. Personal communication.
23. Wexler, L. Simplified serial coronary arteriography. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *111*, 780–781.
24. Wilson, W. J., Lee, G. B., and Amplatz, K. Biplane selective coronary arteriography via percutaneous transfemoral approach. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *100*, 332–340.
25. Zoll, R. M., Wessler, S., and Schlesinger, M. J. Interarterial coronary anastomoses in human heart with particular reference to anemia and relative cardiac anoxia. *Circulation*, 1951, *4*, 797–815.

moses from the right coronary artery to the left circumflex; 3. Diagonals over left marginals to the left circumflex; 4. Intracoronary anastomoses bridging the occlusion.

Key: LC = left main coronary artery; Cx = left circumflex artery; RC = right coronary artery; AVN = atrioventricular node artery; LAD = left anterior descending artery; LM = left marginal artery of the obtuse angle; RM = right marginal artery of the acute angle; SN = sinus node artery; CA = conus artery.

DETERMINATION OF THE TRUE SIZE OF THE CORONARY ARTERY IN CORONARY ARTERIOGRAPHY*

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THE true size of the lumen of the coronary artery and the actual degree of narrowing in the coronary artery caused by an atherosclerotic plaque are often of crucial interest to the radiologist, the cardiologist, and most important to the cardiac surgeon. Because cine as well as film coronary arteriography magnify the opacified coronary artery, the true size of the lumen of the opacified coronary artery can only be estimated.

It is possible to measure the true size of the lumen of the coronary artery precisely, if the angiogram contains the image of an object of known size. The object of known size is measured on the angiogram, and a magnification factor is determined. As the catheter is present on all films and in the same plane with the catheterized coronary artery, its image may be used to compute the magnification factor. An attempt should be made to take the measurement of the catheter in the plane in which the coronary artery is approximately parallel with the horizontal plane.

In order to facilitate this determination, tables have been computed. Table I applies when a No. 7 French catheter is used (O.D. = 2.34 mm.). Table II applies when a No. 8 French catheter is used (O.D. = 2.64 mm.) for selective catheterization.

Table I is used in the following manner: (1) select the row in the left hand column corresponding to the size of the image of the coronary catheter; (2) select the column corresponding to the size of the measured lumen of the coronary artery; and (3) the number at the intersection of this row and column is the actual size of the lumen of the coronary artery.

DISCUSSION

At the University of Minnesota Heart Hospital, coronary arteriography has been performed for the past 16 years. Phasic root injections,[8] acetylcholine arrest with atrial pacing, and more recently transfemoral selective coronary arteriography have been employed.[1,9] The latter technique has proved to be the simplest and most informative, particularly if magnification coronary arteriography is employed.[7]

Among the various techniques for grading of coronary arteriograms,[2–6] a simple grading system proved helpful ranging from 1+ to 4+ depending on the degree of narrowing as estimated from less narrowed segments of the coronary artery, with 1+ referring to 1–25 per cent narrowing, 2+ to 26–50 per cent narrowing, 3+ to 51–99 per cent narrowing, and 4+ to complete occlusion. This classification, also used by many pathologists, has the obvious disadvantage that narrowed segments are compared to other more or less narrowed arterial segments. Therefore, on occasion, a diffusely narrowed but severely diseased coronary artery without localized stenosis may be classified as 1+ or minimal involvement in spite of severe advanced coronary artery disease. The pathologist, on the other hand, grades the degree of stenosis by comparing the remaining lumen to the outside diameter of the artery at postmortem examination. Consequently, he may classify the same artery which was graded as 1+ angiographically as 3+ pathologically (Fig. 1). Furthermore, the pathologist is examining a specimen lacking arterial distention by blood pressure, and there may even be contraction by fixation. Pathologists as well

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as radiologists are puzzled by lacking correlation of these findings.

The discrepancy between the interpretation of the coronary arteriogram and the findings at postmortem examination has stimulated us to devise a method to measure the actual diameter of the true lumen of coronary arteries as well as the percentage of stenosis. Since cine as well as roentgenographic coronary arteriography have a varying degree of magnification, such absolute measurements are inaccurate due to the magnification factor. If an object of known external dimensions, such as the selective catheter, can be used as a reference, the magnification factor can be easily calculated. Although the catheter may not be in the exact plane as the distal opacified coronary artery, the error of measurement is negligible. In any event, the catheter tip lies exactly in the same plane as the catheterized coronary artery ostium. Film coronary angiography lends itself particularly well for such measurements.

By measuring the size of the catheter on the roentgenogram, an exact magnification factor can be calculated which allows computation of the actual coronary artery stenosis using the above described tables. This allows one to determine the exact diameter of the coronary artery distal to an area of stenosis which is of particular interest to the operating surgeon considering a bypass procedure. One must be aware, however, that on occasion the diameter of the coronary artery distal to an area of severe stenosis or occlusion may appear smaller on the angiogram than is actually found at time of surgery. This discrepancy is apparently due to the low perfusion pres-

TABLE I

DETERMINATION OF THE DIAMETER OF CORONARY ARTERIES

These values apply for a No. 7 French catheter (2.34 mm. O.D.)

X (mm.)	Y (mm.)				
	3.0	3.5	4.0	4.5	5.0
0.5	0.4	0.3	0.3	0.3	0.2
1.0	0.8	0.7	0.6	0.5	0.5
1.5	1.2	1.0	0.9	0.8	0.7
2.0	1.6	1.3	1.2	1.0	0.9
2.5	2.0	1.7	1.5	1.3	1.2
3.0	2.3	2.0	1.8	1.6	1.4
3.5	2.7	2.3	2.1	1.8	1.6
4.0	3.1	2.7	2.3	2.0	1.9
4.5	3.5	3.0	2.6	2.3	2.1
5.0	3.9	3.3	2.9	2.6	2.3
5.5	4.3	3.7	3.2	2.9	2.6
6.0	4.7	4.0	3.5	3.1	2.8
6.5	5.1	4.3	3.8	3.4	3.0
7.0	5.5	4.7	4.1	3.6	3.3
7.5	5.8	5.0	4.4	3.9	3.5
8.0	6.2	5.4	4.7	4.2	3.7

The size of the diameter of the image of the coronary artery, X, and the diameter of the image of the catheter, Y, are measured on the roentgenogram. If the true diameter of the catheter is 2.34 mm., then X and Y are used to locate the true diameter Z, of the coronary artery in the Table. Z = 2.34 (X/Y).

X = outside diameter of the image of the artery, as measured on the film (mm.). Y = outside diameter of the image of the catheter, as measured on the film (mm.). Z = true diameter of the artery at that point (mm.).

TABLE II

DETERMINATION OF THE DIAMETER OF CORONARY ARTERIES

These values apply for a No. 8 French catheter (2.64 mm. O.D.)

X (mm.)	Y (mm.)				
	3.0	3.5	4.0	4.5	5.0
0.5	0.4	0.4	0.3	0.3	0.3
1.0	0.9	0.8	0.7	0.6	0.5
1.5	1.3	1.1	1.0	0.9	0.8
2.0	1.8	1.5	1.3	1.2	1.1
2.5	2.2	1.9	1.6	1.5	1.3
3.0	2.6	2.3	2.0	1.8	1.6
3.5	3.1	2.6	2.3	2.1	1.8
4.0	3.5	3.0	2.6	2.4	2.1
4.5	4.0	3.4	3.0	2.6	2.4
5.0	4.4	3.8	3.3	2.9	2.6
5.5	4.8	4.1	3.6	3.2	2.9
6.0	5.3	4.5	4.0	3.5	3.2
6.5	5.7	4.9	4.3	3.8	3.4
7.0	6.2	5.3	4.6	4.1	3.7
7.5	6.6	5.7	4.9	4.4	4.0
8.0	7.0	6.0	5.3	4.7	4.2

The size of the diameter of the image of the coronary artery, X, and the diameter of the image of the catheter, Y, are measured on the roentgenogram. If the true diameter of the catheter is 2.64 mm., then X and Y are used to locate the true diameter Z, of the coronary artery in the Table. Z = 2.64 (X/Y).

X = outside diameter of the image of the artery, as measured on the film (mm.). Y = outside diameter of the image of the catheter, as measured on the film (mm.). Z = true diameter of the artery at that point (mm.).

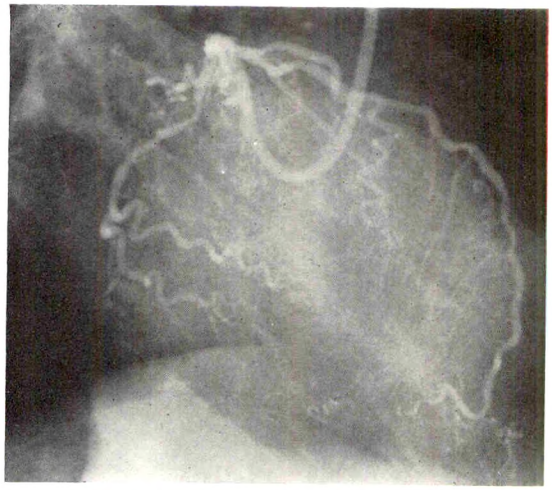


FIG. 1. Only minimal caliber changes of the left anterior descending artery are well seen on magnification coronary arteriogram. Absolute measurement, derived from catheter size, shows a 1 mm. diameter, indicating severe, diffuse narrowing of the left anterior descending artery. By the other technique the disease process would have been classified as 1+ only.

sure and distention of poststenotic arterial segments. In spite of the fact that such arteries may appear to be extremely small or not opacified on the angiogram, surgery is not necessarily contraindicated.

Absolute measurements of stenotic coronary arteries are particularly important when comparison is made with already diffusely narrowed coronary arteries. A mild, angiographically nonimpressive stenosis of 50 per cent in a 1 mm. coronary artery may reduce the lumen to 0.5 mm., which would make the stenosis certainly hemodynamically constricting. On the other hand a 5 mm. artery would only be narrowed to 2.5 mm., which may not reduce blood flow. The absolute measurement, therefore, of the stenosis, not only in diameter but also in length, becomes of great clinical importance. Admittedly the coronary angiogram cannot give definite hemodynamic information, unless collaterals or delayed opacification behind stenotic arterial branches are present. It is important to note, however, that delayed filling of one of the major coronary artery branches may also be due to such technical factors as partial occlusion by catheter tip or penetration of the catheter beyond a major coronary artery bifurcation.

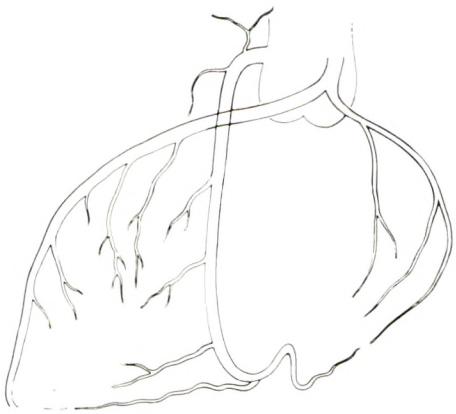
Since coronary arteriography is no longer a purely diagnostic test, but has become the most important information for the operating surgeon, the following grading procedure is presently used at the University of Minnesota Hospitals: (1) classification of 1 to 4+ narrowing is abandoned since this system is particularly inaccurate in the 3+ range which varies from 51 to 99 per cent. This represents the degree of disease which may be surgically important; (2) the degrees of stenosis are expressed by actually measuring the pre- and poststenotic segment of the opacified artery and calculating the degree of narrowing from the projection showing maximal arterial constriction; (3) the site of stenosis, percentage of narrowing, and collateral pathways are diagrammatically recorded on a drawing of the coronary arteries (Fig. 2) which is pasted on the x-ray jacket and the patient's chart; (4) the true diameter of the prestenotic as well as poststenotic coronary artery segment as well as the diameter of coronary artery stenosis and length are computed by the above mentioned tables; and (5) additional physiologic parameters, as end systolic volume, end diastolic volume, ejection fraction, and diastolic pressures prior to and post left ventriculography, etc., are recorded.

Since the muscle mass of the heart shows only minor variations in healthy individuals, it stands to reason to assume that coronary artery size will also vary to a minor degree. As soon as normal standards are available for various age groups from pathologic specimens, the absolute measurements on the coronary arteriogram will become of even greater importance.

CONCLUSION AND SUMMARY

The hemodynamic assessment of coronary artery stenoses on coronary arteriograms is difficult unless delayed opacification or collaterals are demonstrated.

Expressing the degree of narrowing in



	DIAMETER		STENOSES		END DIAS PRES	
	1	2	mm I %	mm II %	1	2
LM						
LAD					L.V. FUNCTION	
LC						
MAR						
RC					COLLATERALS	
PD						

Fig. 2. This diagram is placed in the chart of each patient following coronary arteriography. The areas of stenoses are graphically depicted on the diagram of the coronary arteries. The true diameter of the coronary artery proximal (1) and distal (2) to the stenosis is recorded, and the percentage of stenosis is calculated. The end diastolic pressure of the left ventricle pre (1) and post (2) left ventricular cine angiogram is recorded. LM = left main; LAD = left anterior descending; LC = left circumflex; MAR = marginal branch; RC = right coronary; PD = posterior descending; End Dias Pres = end diastolic pressure; L.V. Function = left ventricular function.

percentage may be disadvantageous, since disease segments are compared with more or less stenotic segments. This system is particularly inaccurate in the 3+ category (51 to 99 per cent) which is of particular interest to the cardiologists and operating surgeon.

Exact measurement of the coronary artery proximal and distal to the stenosis as well as measurement of the stenosis in width and length may be more hemodynamically meaningful. A simple conversion table allows computation of actual coronary artery size with high accuracy, taking into consideration various magnification factors. These tables are particularly useful in film recording of coronary arteriograms, since cine coronary arteriography lacks definition and does not lend itself to measurements as readily.*

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REFERENCES

1. Bilgutay, A. M., Wingrove, R. C., and Lillehei, C. W. Double contrast left cardioangiography utilizing carbon dioxide: safe method for direct vision of luminal and valvular lesions employing new device for gas injection. *Surgery*, 1962, *52*, 77–84.
2. Gensini, G. G., and Bjonanno, C. Coronary arteriography: study of 100 cases with angiographically proved coronary artery disease. *Dis. Chest*, 1968, *54*, 90–94.
3. Hultgren, H., Calciano, A., Platt, F., and Abrams, H. Clinical evaluation of coronary arteriography. *Am. J. Med.*, 1967, *42*, 228–231.
4. Licoff, W., Segal, B. L., and Kasparian, H. Paradox of normal selective coronary arteriograms in patients considered to have unmistakeable heart disease. *New England J. Med.*, 1967, *276*, 1063–1064.
5. Paulin, S. Coronary angiography. *Acta radiol.*, 1964, Suppl. 233.
6. Proudfit, W. L., Shirey, E. K., and Sones, F. M. Distribution of arterial lesions demonstrated by selective cine coronary arteriography. *Circulation*, 1967, *36*, 54–62.
7. Randall, P. A., and Amplatz, K. Magnification coronary arteriography. *Radiology*, 1971, *101*, 51–56.
8. Thal, A. P., Lester, R. G., Richards, L. S., and Murray, M. J. Coronary arteriography in atherosclerotic disease of heart. *Surg., Gynec. & Obst.*, 1957, *105*, 457–464.
9. Wilson, W. J., Lee, G. B., and Amplatz, K. Biplane selective coronary arteriography via percutaneous transfemoral approach. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *100*, 232–340.

* This same technique has been found useful as a method of determining the exact size of other vascular structures such as the aortic root and pulmonary arteries.

ANGIOGRAPHIC DETERMINATION OF OPERABILITY IN CANDIDATES FOR AORTO-CORONARY BYPASS*

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WITHIN the last 2 decades a variety of direct and indirect myocardial revascularization operations have been devised. Serious doubts have been raised as to the efficacy of some,[10] however, while others were plagued by high mortality rates.[9] In this setting, the introduction by Johnson *et al.*,[6] and Favaloro[4] of the aorto-coronary bypass using a saphenous vein autograft has been greeted enthusiastically by most of those who feel surgery is the treatment of choice for coronary artery disease. In addition to relatively low mortality rates,[9,12] dramatic and unequivocal angiographic evidence of improved coronary blood flow can be obtained postoperatively. Bypass patency rates approximating 90 per cent have been noted in a few short term follow-up series.[6,9]

The many recent articles discussing the operation have dealt primarily with matters such as the clinical indications for it,[5,12] the specific surgical techniques to be used,[4,6,7,11] the subjective benefits to the patient,[7,9,12] and the short term follow-up results.[6,10] One aspect which has not yet been properly investigated is the correlation between the preoperative angiographic flow pattern seen in a diseased coronary artery and its suitability for venous bypass. This important correlation is the subject of the present study.

Arteriography is widely used in peripheral arterial occlusive disease to determine the feasibility of bypass surgery. If a leg arteriogram shows only minimal or absent "run-off" distal to a superficial femoral or popliteal artery occlusion, bypass surgery is generally obviated. The question arises as to whether the same principle applies in patients with coronary artery disease; *i.e.*, might a stenotic or occluded coronary artery with poor distal "run-off" be similarly unsuited for bypass? Should the use of the operation be restricted only to individuals whose coronary arteries can be arteriographically shown to be patent distally, or can the surgeon proceed under the assumption that they are *probably* patent distally even though not demonstrable on the arteriogram?

MATERIAL AND METHOD

In an attempt to answer these questions we have studied 57 venous bypasses in 30 patients who have had angiography both preoperatively and within 2–20 weeks postoperatively. Each artery which was later bypassed was assigned to 1 of 2 categories based upon the angiographically-demonstrated caliber of its distal segment beyond the stenotic lesion.

Arteries assigned to Group I had distal lumina of more than 1 mm. diameter after correction for magnification. Those arteries assigned to Group II either completely lacked distal flow or had very minimal distal flow with an opacified lumen of only 1 mm. diameter or less. No differentiation was made between antegrade filling past a partial obstruction or retrograde filling via collaterals in cases of complete or nearly complete obstruction.

The corresponding postoperative venous bypass angiograms were then reviewed. Finally, comparison was made of the bypass patency rates for the 2 groups.

The roentgenologic studies were carried out utilizing the percutaneous femoral approach and the polyurethane Ducor catheters manufactured by Cordis. Both the angiographic techniques[1,2,8] and the surgical procedures[4,6] have been described in detail elsewhere.

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RESULTS

Forty-seven of the bypassed arteries had a distal lumen diameter of greater than 1 mm. at arteriography and thus met the criterion for Group I preoperatively. Forty-three of these bypasses were found to be patent postoperatively, representing an early success rate of 91 per cent.

Ten of the arteries had distal lumen

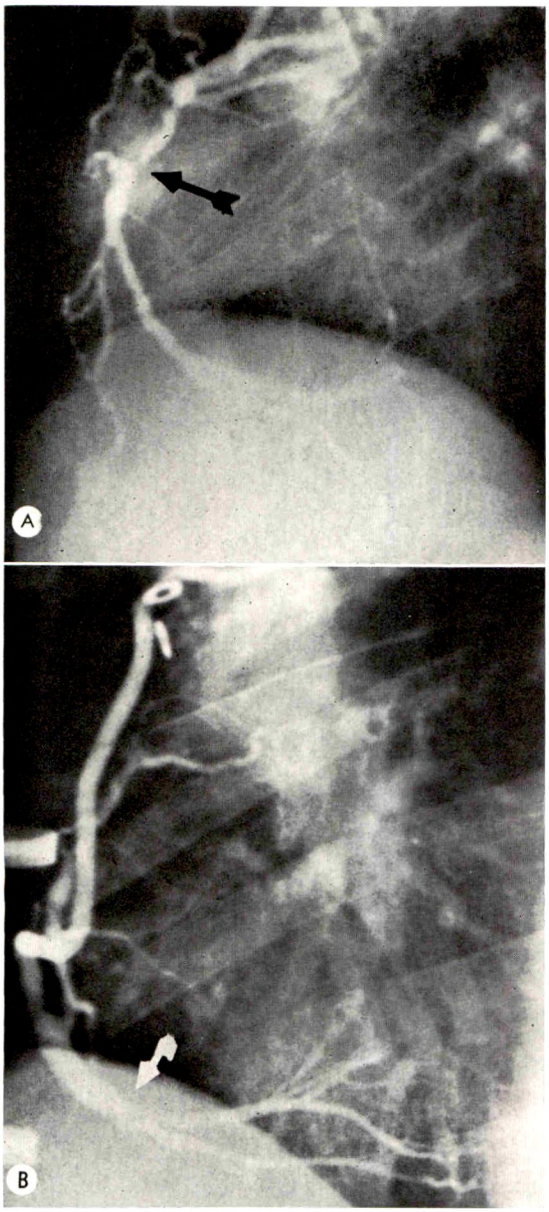


FIG. 1. (*A*) Left anterior oblique (LAO) view of a Group I right coronary artery. There is severe stenosis (black arrow) of the proximal portion of this dominant artery. Distally it is widely patent with good flow into the posterior descending and posterior left ventricular branches. (*B*) Postoperative LAO view of the patent venous bypass. The white arrow points to the anastomosis of the vein with the artery.

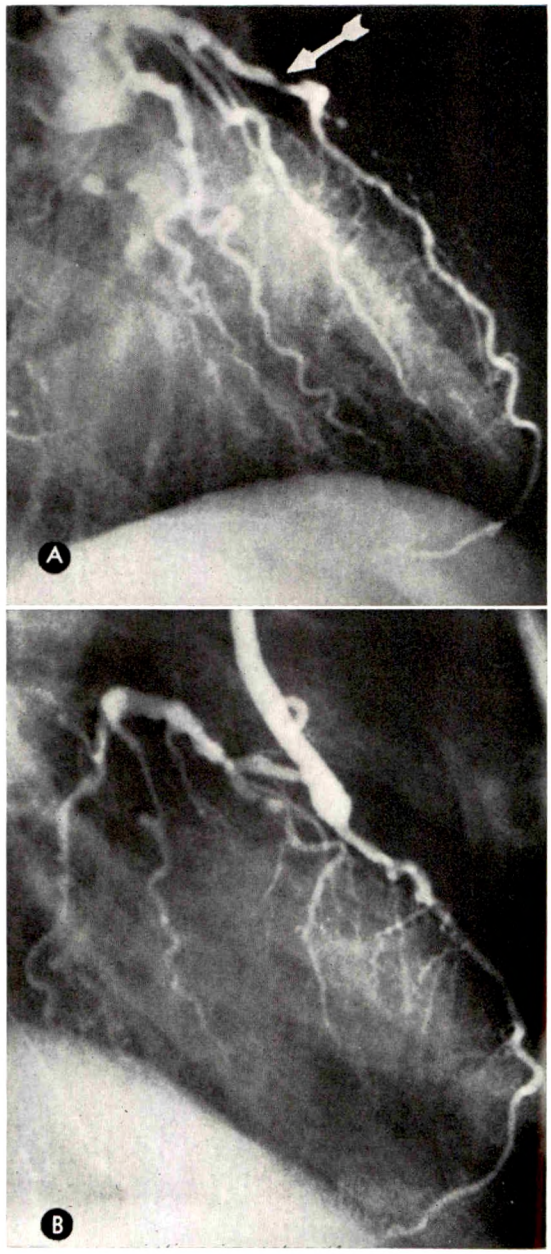


FIG. 2. (*A*) Right anterior oblique (RAO) view of a Group I left anterior descending artery. There is stenosis proximally (white arrow) with a normal caliber distally. (*B*) Postoperative lateral view of the patent venous bypass.

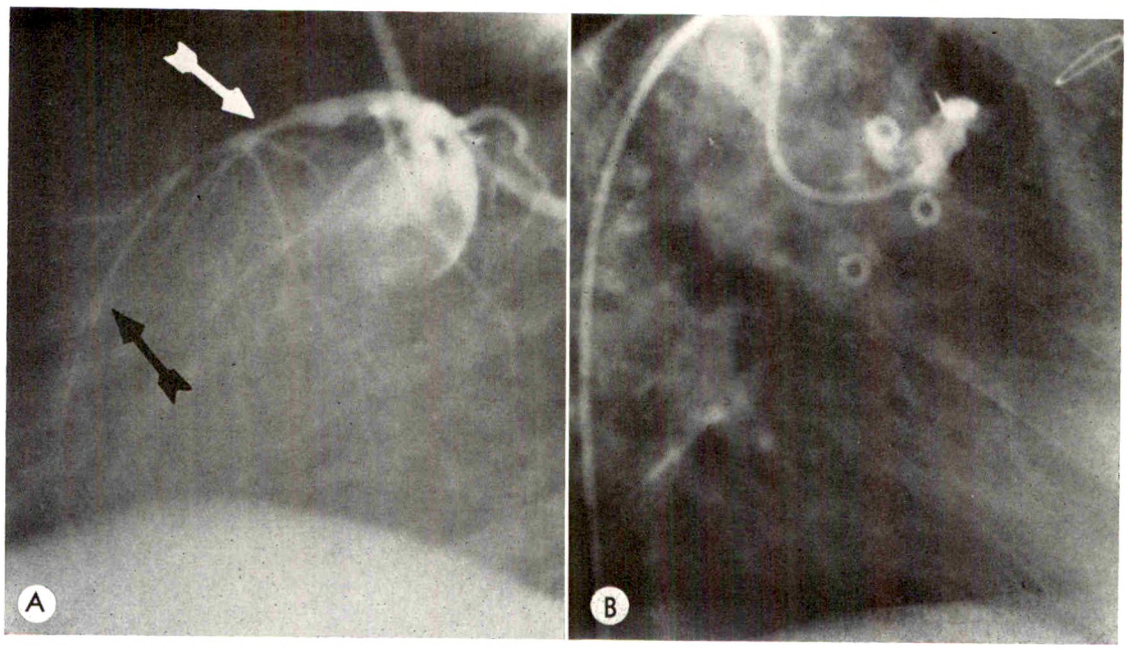


FIG. 3. (*A*) LAO view of a Group II left anterior descending artery. There is severe proximal stenosis (white arrow). The distal segment of this artery (black arrow) is of very narrow caliber, measuring less than 1 mm. in diameter. There was no collateral flow from the right side. (*B*) Postoperative study showing complete occlusion of the venous bypass.

diameters of 1 mm. or less at arteriography and thus fell into Group II preoperatively. There were 3 patent venous bypasses among this group, representing a success rate of only 30 per cent.

Figures 1 and 2 are examples of Group I coronary arteries which were successfully bypassed. Figures 3–5 are examples of Group II cases in which the bypasses were found to be occluded. Figures 6 and 7 show 2 of the 3 Group II arteries which were successfully bypassed.

DISCUSSION

Many living patients with severe proximal obstructions of the major coronary arteries have rather substantial blood flow within the distal segments of these arteries. Blood enters these segments in an antegrade direction if the obstruction is partial or where recanalization of a thrombus has occurred. It enters in retrograde fashion from collaterals where the obstruction is total or nearly so. In either event this flow can be visualized angiographically and means that the distal portion of the artery remains patent. Our demonstration of 91 per cent early graft patency among Group I coronary arteries indicates that where a distal lumen of greater than 1 mm. angiographic diameter has been preserved, bypass can be carried out with a high expectation of success.

The outlook is considerably less favorable for Group II cases, where the distal coronary artery segments either fail to opacify or are only 1 mm. or less in diameter. In this group, only a 30 per cent early graft patency rate could be achieved.

When an arteriogram reveals the coronary tree distal to a stenosis to be of extremely narrow caliber (*i.e.*, Group II), it is generally unclear whether this indicates diffuse atheromatous disease or merely collapse due to lack of high pressure flow in an otherwise patent channel. The surgeon who bypasses such an artery proceeds on the latter assumption, and the 3 successful bypasses in Group II patients (Fig. 6, *A–C*; and 7, *A* and *B*) are instances in which this

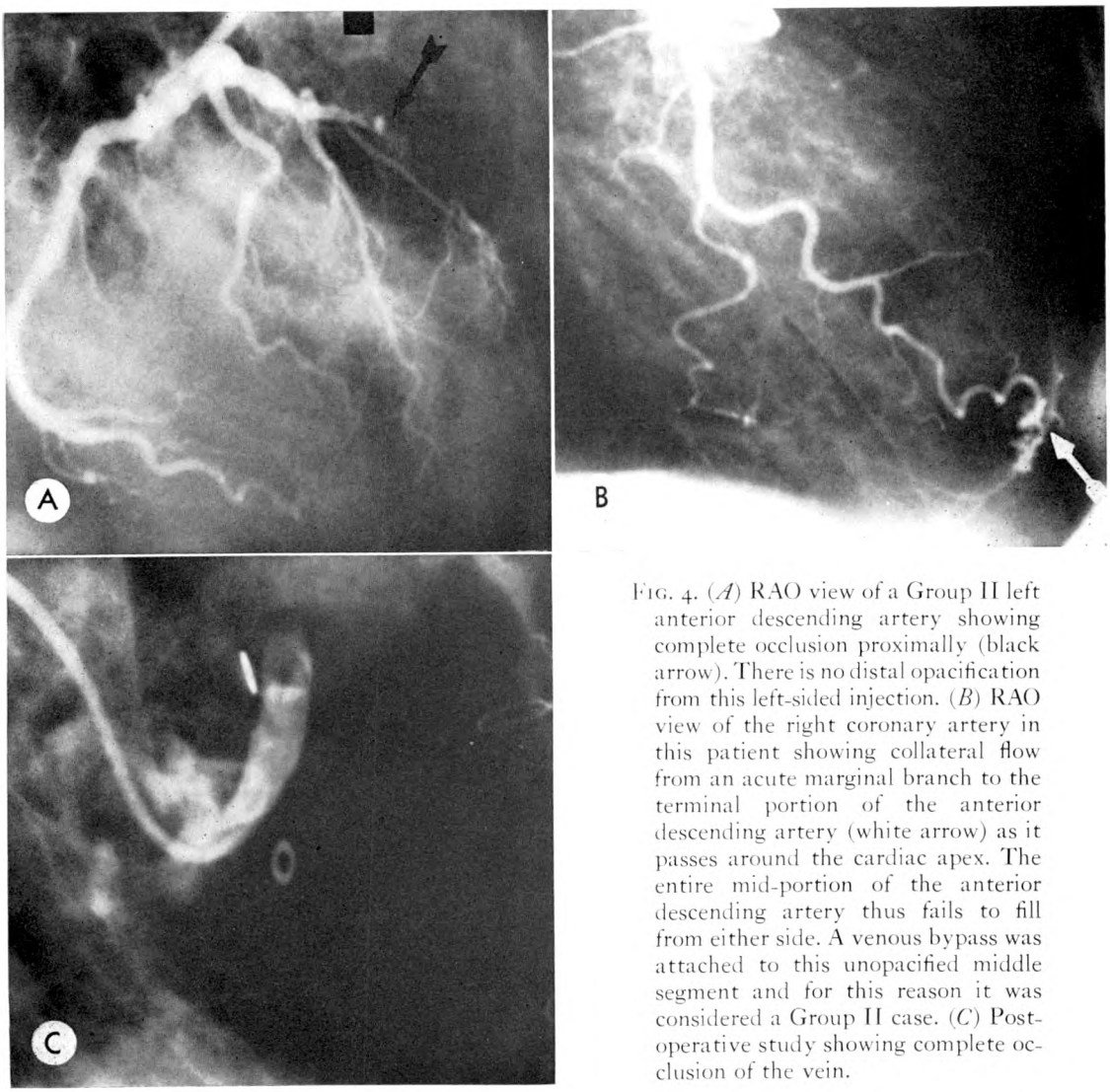


Fig. 4. (*A*) RAO view of a Group II left anterior descending artery showing complete occlusion proximally (black arrow). There is no distal opacification from this left-sided injection. (*B*) RAO view of the right coronary artery in this patient showing collateral flow from an acute marginal branch to the terminal portion of the anterior descending artery (white arrow) as it passes around the cardiac apex. The entire mid-portion of the anterior descending artery thus fails to fill from either side. A venous bypass was attached to this unopacified middle segment and for this reason it was considered a Group II case. (*C*) Postoperative study showing complete occlusion of the vein.

judgment was correct. Preoperatively the distal segment diameters of these arteries measured 1 mm. arteriographically, but they were seen to be larger postoperatively. Their narrow preoperative calibers were obviously due to poor perfusion, since dilatation occurred once increased flow was supplied by the venous bypasses. In such cases the coronary arteriogram was deceptive in that a normally patent distal vessel was made to appear very narrow because of a small volume of flow at low pressure.

However, there is no escaping the fact that most Group II arteries in this series could not be successfully bypassed. The reason was generally felt to be the presence of extensive atherosclerosis along the entire distal course of the artery or the associated technical problems. This diffuse disease makes construction of the anastomosis quite difficult and produces high resistance to flow through the vein, leading ultimately to stasis and thrombosis. In most of these cases then, the arteriogram gave a true indication of severe diffuse atherosclerotic narrowing, rendering these vessels unsuitable for venous bypass. Lesions of this type may be more amenable to coronary endarterectomy or an indirect revascularization procedure such as the Vine-

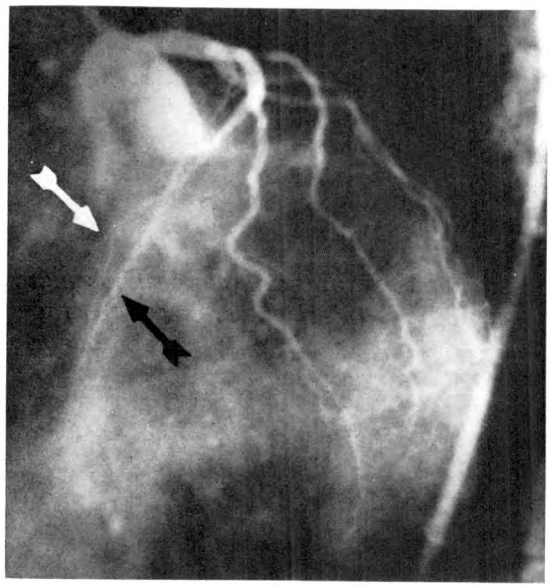
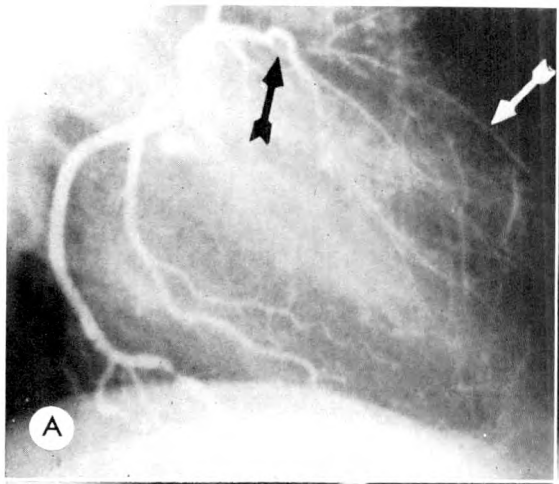
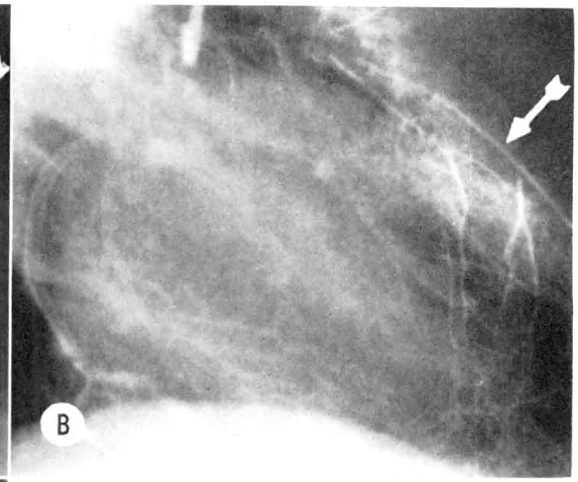


FIG. 5. LAO view of a Group II left anterior descending artery (white arrow) which tapers to a complete obstruction. No distal filling was seen from either side. The black arrow points to a large septal branch. A venous bypass was attached to the distal anterior descending artery, but postoperative angiography revealed it to be occluded.

berg operation.[13] Figures 8 and 9 are additional examples of patients with narrow, poorly opacified poststenotic distal coronary artery segments who were considered unfavorable candidates for venous bypass. The first patient (Fig. 8, *A–C*) underwent successful gas endarterectomy of his distal





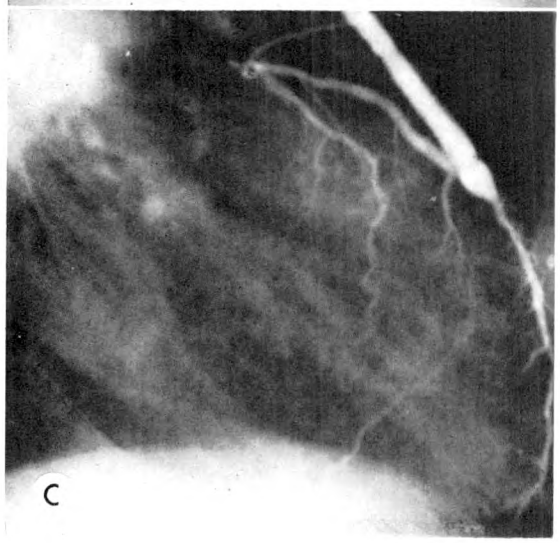


FIG. 6. (*A*) Early and (*B*) late phase RAO views of a Group II left anterior descending artery. There is complete occlusion proximally (black arrow) just beyond the origin of a diagonal branch. The distal segment (white arrows) fills via collaterals, but is of narrow caliber, measuring 1 mm. in diameter. (*C*) In spite of this, the vein bypass was later seen to be functioning well in this lateral view. Note that the caliber of the artery has increased due to improved flow.

right coronary artery with removal of a thick atheromatous core. He has not been studied postoperatively, but has done well clinically.

Fortunately only a minority of patients with significant coronary atherosclerosis have distal arteries of such narrow caliber that they are unfavorable candidates for bypass. This is borne out by the fact that there were 47 Group I arteries and only 10 Group II arteries in this series, as well as by our much wider general experience in patients with this disease.

In a recent article, Spencer *et al.*[12] expressed the belief that inability to demonstrate coronary artery patency beyond an obstruction does not contraindicate bypass surgery, since arteries seemingly 1 mm. or less in diameter at angiography could frequently be dilated to 1.5 or 2.0 mm. at the operating table. This same thing was found in approximately half of our Group II patients. However, postoperative angiograms were only done in a relatively small percentage of the total series of Spencer *et al.*[12] and no indication at all is given of how many vein bypasses to these small distal coronary arteries remained patent. The ability to insert a 2.0 mm. probe into a diseased artery does not necessarily mean that the lumen is actually that large. The probe may spread atheromatous material, fibrin and thrombi apart as it passes through, only to have them reobstruct the artery when it is withdrawn. Dotter and Judkins[3] showed clearly that a probe could quite readily be passed through a totally occluded atherosclerotic vessel.

The results of our study tend to refute the notion that failure of good opacification of the distal segment of a stenotic coronary artery can be disregarded by the surgeon. Instead they suggest that there is indeed strong correlation between the caliber of this distal segment, as demonstrated angiographically, and the suitability of the artery for venous bypass. Early graft patency rates of over 90 per cent should be achieved where the poststenotic distal segment of the artery is seen to have a diameter of more than 1 mm. The chance of success is considerably less where the diameter

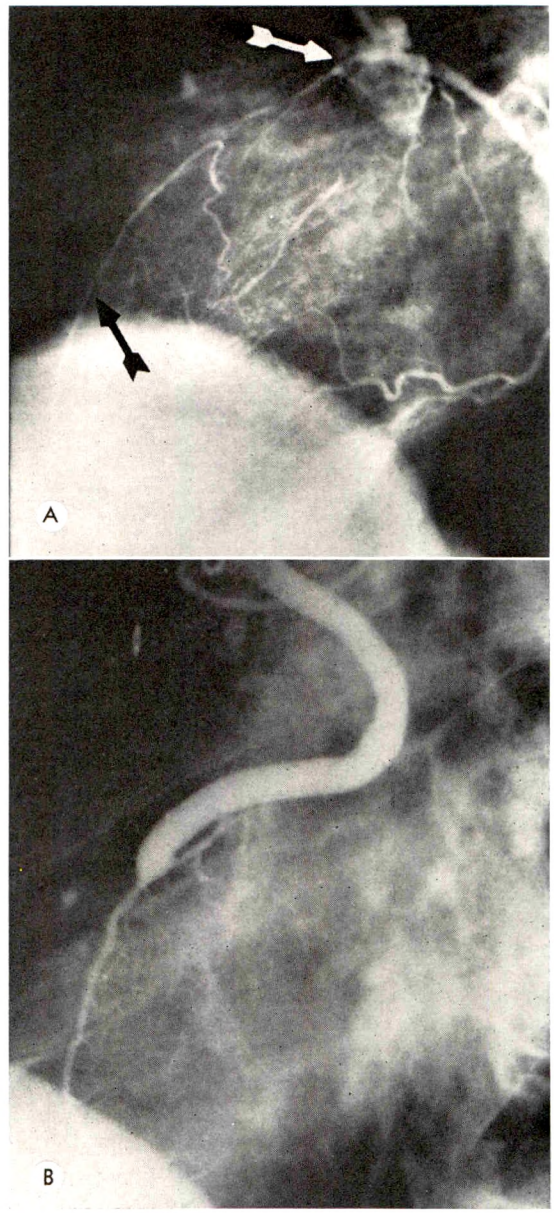


Fig. 7. (*A*) LAO view of a Group II left anterior descending artery showing severe stenosis proximally (white arrow). Collateral flow from the obtuse marginal branch of the circumflex artery to the diagonal branch opacifies the distal anterior descending artery (black arrow), but its lumen measures only 1 mm. in diameter. (*B*) Nevertheless, the LAO view of the bypass angiogram demonstrates a successful result. Again note that the caliber of the artery has enlarged postoperatively.

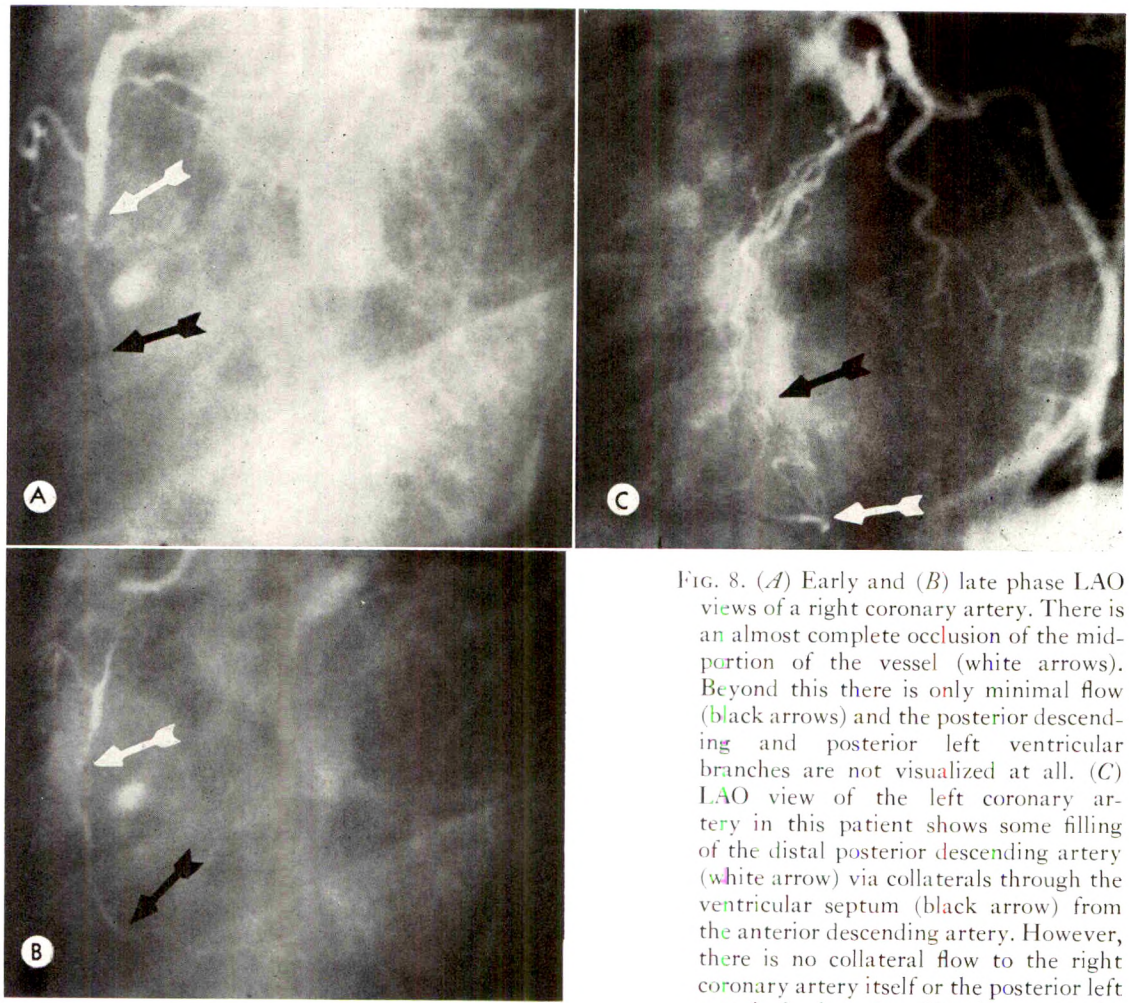


FIG. 8. (*A*) Early and (*B*) late phase LAO views of a right coronary artery. There is an almost complete occlusion of the mid-portion of the vessel (white arrows). Beyond this there is only minimal flow (black arrows) and the posterior descending and posterior left ventricular branches are not visualized at all. (*C*) LAO view of the left coronary artery in this patient shows some filling of the distal posterior descending artery (white arrow) via collaterals through the ventricular septum (black arrow) from the anterior descending artery. However, there is no collateral flow to the right coronary artery itself or the posterior left ventricular branches. For this reason the patient was considered an unfavorable candidate for venous bypass and underwent right coronary gas endarterectomy.

appears smaller than 1 mm. or where there is no demonstrable distal flow at all. Coronary arteriography thus not only identifies the location and severity of an obstructing lesion, but also assumes an important role in determining the advisability of bypass surgery.

SUMMARY

The results of venous bypass operations upon 57 diseased coronary arteries have been studied, using pre and postoperative angiography.

An attempt was made to determine if the angiographically demonstrated caliber of the poststenotic distal segment of the artery was of any significance in deciding which patients were suitable candidates for bypass surgery.

Among the 47 arteries having a distal lumen diameter of greater than 1 mm., a 91 per cent graft patency rate was achieved. Among the 10 arteries having a distal lumen diameter of 1 mm. or less, only a 30 per cent graft patency rate could be achieved.

Thus it would appear that the coronary arteriogram gives an accurate indication of the actual caliber of the distal segment and that where this is 1 mm. or less, diffuse

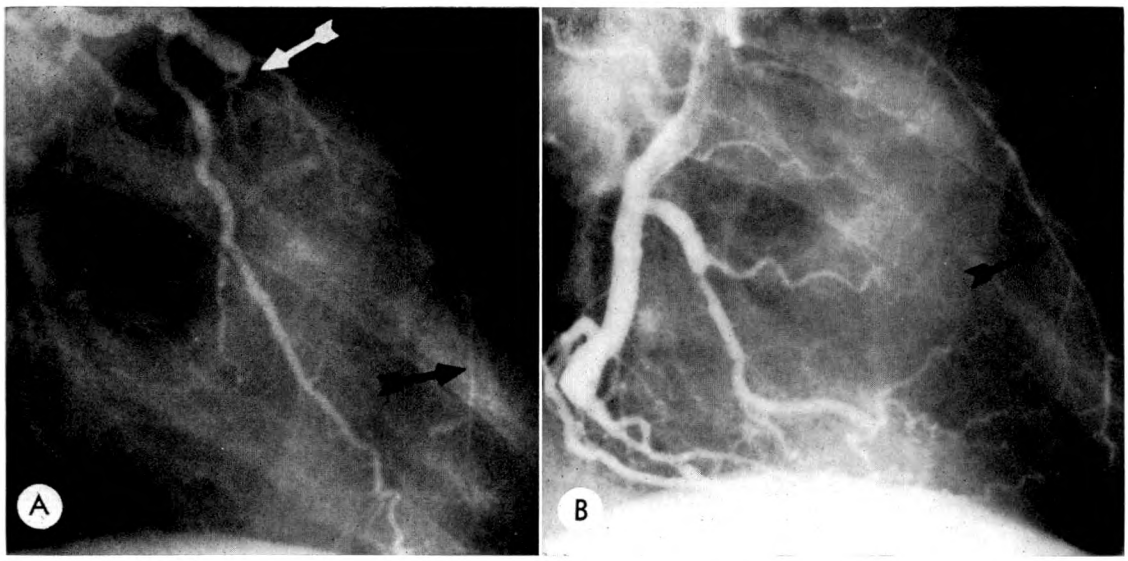


Fig. 9. (*A*) RAO view of the left coronary artery showing total occlusion of the anterior descending artery (white arrow). There is faint opacification of a distal segment (black arrow). (*B*) RAO view of the right coronary artery in the same patient. There is collateral flow from acute marginal branches to the distal anterior descending artery (black arrow) which measures less than 1 mm. in diameter. This patient would therefore be considered an unfavorable candidate for venous bypass.

atherosclerosis is likely to be present, making the outlook for a bypass operation rather unfavorable.

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REFERENCES

1. Amplatz, K., Formanek, G., Stanger, P., and Wilson, W. Mechanics of selective coronary artery catheterizations via femoral approach. *Radiology*, 1967, *89*, 1040–1047.
2. Baltaxe, H. A., Carlson, R. G., and Lillehei, C. W. Roentgenographic appearance of aorto-coronary artery bypass using reversed saphenous vein. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *110*, 734–738.
3. Dotter, C. T., and Judkins, M. P. Percutaneous transluminal treatment of arteriosclerotic obstruction. *Radiology*, 1965, *84*, 631–643.
4. Favaloro, R. G. Saphenous vein graft in surgical treatment of coronary artery disease: operative technique. *J. Thoracic Surg.*, 1969, *58*, 178–185.
5. Johnson, W. D., Flemma, R. J., and Lepley, D. Direct coronary surgery utilizing multiple-vein bypass grafts. *Ann. Thoracic Surg.*, 1970, *9*, 436–444.
6. Johnson, W. D., Flemma, R. J., Lepley, D., and Ellison, E. H. Extended treatment of severe coronary artery disease: total surgical approach. *Ann. Surg.*, 1969, *170*, 460–470.
7. Johnson, W. D., and Lepley, D. Aggressive surgical approach to coronary disease. *J. Thoracic & Cardiovasc. Surg.*, 1970, *59*, 128–138.
8. Judkins, M. P. Selective coronary arteriography. *Radiology*, 1967, *89*, 815–824.
9. Sheldon, W. C., Favaloro, R. G., Sones, F. M., and Effler, D. B. Reconstructive coronary artery surgery: venous autograft technique. *J.A.M.A.*, 1970, *213*, 78–82.
10. Sheldon, W. C., Sones, F. M., Shirey, E. K., Ferguson, D. J., and Effler, D. B. Reconstructive coronary artery surgery: postoperative assessment. *Circulation*, 1969, *39*, Suppl. 1, 61–66.
11. Spencer, F. C. Venous bypass grafts for occlusive disease of coronary arteries. *Am. Heart J.*, 1970, *79*, 568–571.
12. Spencer, F. C., Green, G. E., Tice, D. A., and Glassman, E. Bypass grafting for occlusive disease of coronary arteries: report of experience with 195 patients. *Ann. Surg.*, 1971, *173*, 1029–1044.
13. Vineberg, A. M. Development of anastomosis between coronary vessels and transplanted internal mammary artery. *Canad. M. A. J.*, 1946, *55*, 117–119.

LATE CHANGES IN AORTOCORONARY VEIN GRAFTS: ANGIOGRAPHIC FEATURES*

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EARLY postoperative angiographic evaluation of patients with aortocoronary vein grafts may yield valuable information.[3,5,11,21] This information may help the cardiologist in the treatment of patients with persisting or recurring symptoms. It may also, in case of occlusion or stenosis of vein grafts, lead to early surgical correction of these conditions.[9] When conducted systematically in all patients in the early and late postoperative period, these angiographic studies may provide a clear estimate of the attrition rate of aortocoronary vein grafts and help to improve the angiographic selection of patients, and the surgical techniques.[1,10,15]

The purpose of this communication is to describe the angiographic changes noted in a consecutive series of patients with aortocoronary vein grafts who underwent early and late postoperative angiographic evaluation.

MATERIAL AND METHOD

Early and late postoperative angiographic studies were carried out in 96 patients. The first angiographic study was performed 2 to 3 weeks after operation, the second 12 to 18 months later. These 96 patients were among the first 138 patients to be submitted to aortocoronary vein grafts at the Montreal Heart Institute.

The 42 patients not included in the present study comprise those who died early or late after operation (14 patients), those who declined the initial study (5 patients) or the late study (14 patients), and those who had thrombosis of a single graft (9 patients) on the initial angiogram.

The 96 patients had a total of 144 vein grafts. Eleven grafts were occluded on the initial evaluation. Thirty of the remaining 133 grafts were occluded 1 year later, allowing a comparative study in 103 of the initial 144 grafts. Thus the over-all patency rate after 12 to 18 months was 72 per cent.

The quality of the distal coronary arterial run-off was assessed on the preoperative arteriogram.[15] Blood flow through the graft was also measured at operation.[11] Postoperative selective angiography of vein grafts was performed with specially designed polyethylene catheters.[2] The caliber of vein grafts was measured on the frontal plane angiogram projected on a Tage-Arno screen. The uncorrected value is utilized throughout this presentation. Late changes in size or diameter of vein grafts were expressed in percentage of the initial value obtained.

RESULTS

A decrease in size of vein grafts was noted on the second angiographic study in almost all cases. Narrowing or stenosis of vein grafts was either diffuse or segmental and was further classified as mild, moderate or severe.

SEGMENTAL NARROWING OF VEIN GRAFTS (TABLE I)

Thirty-six of the 103 grafts showed localized narrowing on the late study. This narrowing was mild (<30 per cent) in 19 grafts (Fig. 1), moderate (40 to 60 per cent) in 14 grafts (Fig. 2, *A* and *B*) and

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severe (>80 per cent) in 3 grafts (Fig. 3, *A* and *B*).

DIFFUSE NARROWING OF VEIN GRAFTS (FIG. 4)

As stated above, on the second angiogram, most grafts displayed, in addition to localized narrowing, some reduction in caliber throughout their entire length (mean reduction of 30 per cent). This diffuse reduction in caliber was negligible (0 to 20 per cent) in 33 grafts (Fig. 5, *A–C*), mild (20 to 40 per cent) in 38 grafts, moderate (40 to 60 per cent) in 29 grafts (Fig. 6, *A* and *B*) and severe (>80 per cent) in 3 grafts (Fig. 7, *A* and *B*).

FATE OF VEIN GRAFTS WITH MODERATE TO SEVERE DIFFUSE OR LOCALIZED NARROWING

Two patients have thus far been submitted to a third angiographic study 2 years after operation. In one instance, severe segmental narrowing of a vein graft progressed to complete occlusion. In the other patient, the second angiographic study had shown both types of narrowing—diffuse and segmental—of a vein graft (Fig. 8, *A–C*). This graft was occluded on the third angiographic study. At reoperation, a second vein graft was fashioned on the left anterior descending artery. Histologic sections of the occluded vein graft showed marked intimal fibrous proliferation (Fig. 8*D*).

INITIAL DIAMETER OF GRAFT AND DEGREE OF SUBSEQUENT REDUCTION IN SIZE

Vein grafts were divided into 3 groups according to their initial diameter. Large vein grafts underwent a proportionately greater reduction in size than grafts with a smaller diameter. As a consequence, all grafts in the 3 groups on the second angiogram tended to be of similar size and, as a rule, their size tended to approximate that of large coronary vessels (3 to 4 mm.).

TABLE I

SEGMENTAL STENOSES OF VEIN GRAFTS (103 grafts)

Mild	19
Moderate	14
Severe	3
	36
Stenoses at proximal third	26
Stenoses at distal third	8
Cases with residual lumen <2 mm.	14

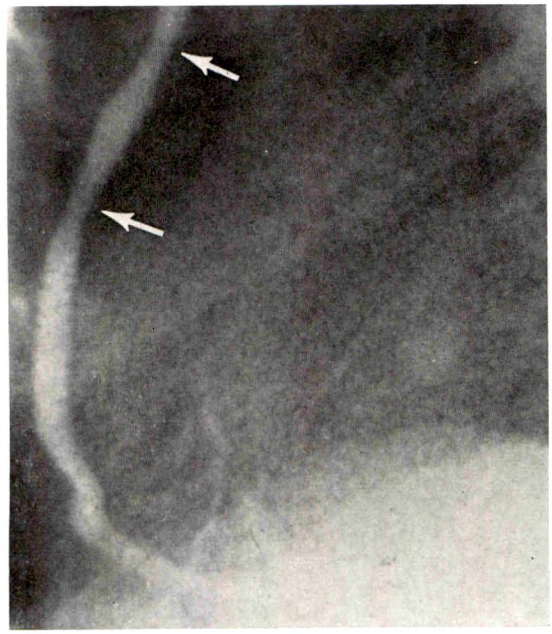


FIG. 1. Right aortocoronary vein graft showing mild segmental stenoses at the proximal and middle portion of the graft (arrows).

DISTAL ARTERIAL RUN-OFF, BLOOD FLOW MEASUREMENTS, AND DIFFUSE OR SEGMENTAL NARROWING OF VEIN GRAFTS

The distal arterial run-off, as assessed on the preoperative coronary arteriogram was judged to be adequate in all 17 grafts showing moderate to severe segmental narrowing, and in 23 of the 29 grafts showing moderate diffuse narrowing. Run-off was poor in 2 of the 3 cases displaying severe diffuse narrowing (Fig. 9).

There was likewise no correlation between intraoperative blood flow measurements and late stenosis of vein grafts.

DISCUSSION

The attrition rate of aortocoronary vein grafts can best be assessed by multiple serial angiographic evaluation conducted in

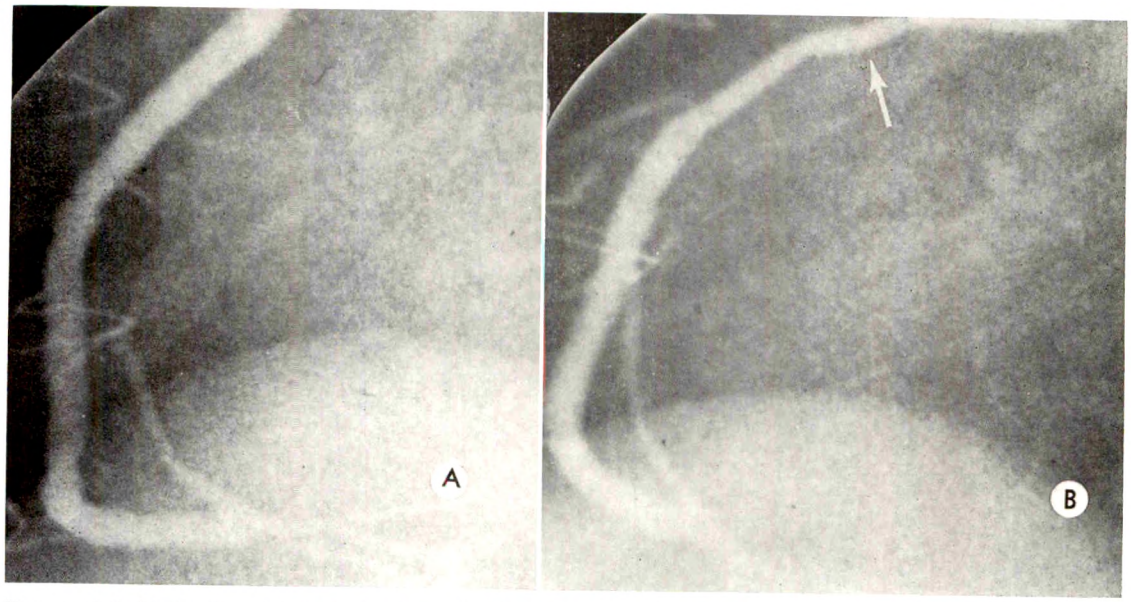


FIG. 2. (*A*) Early (2 weeks) angiogram of right aortocoronary vein graft. Normal angiographic aspect. (*B*) Selective angiogram 1 year after operation showing moderate segmental narrowing (50 per cent) of proximal portion of vein graft (arrow).

a large and consecutive series of patients. As was the case for internal mammary artery implantation, clinical symptoms are not a reliable method of assessing patency of aortocoronary vein grafts.

Sheldon and Grinfeld[22] reported an overall patency rate of 85 per cent up to 3 years after aortocoronary vein grafts. In the study of Johnson and co-workers[14] performed 2 weeks to 14 months after opera-

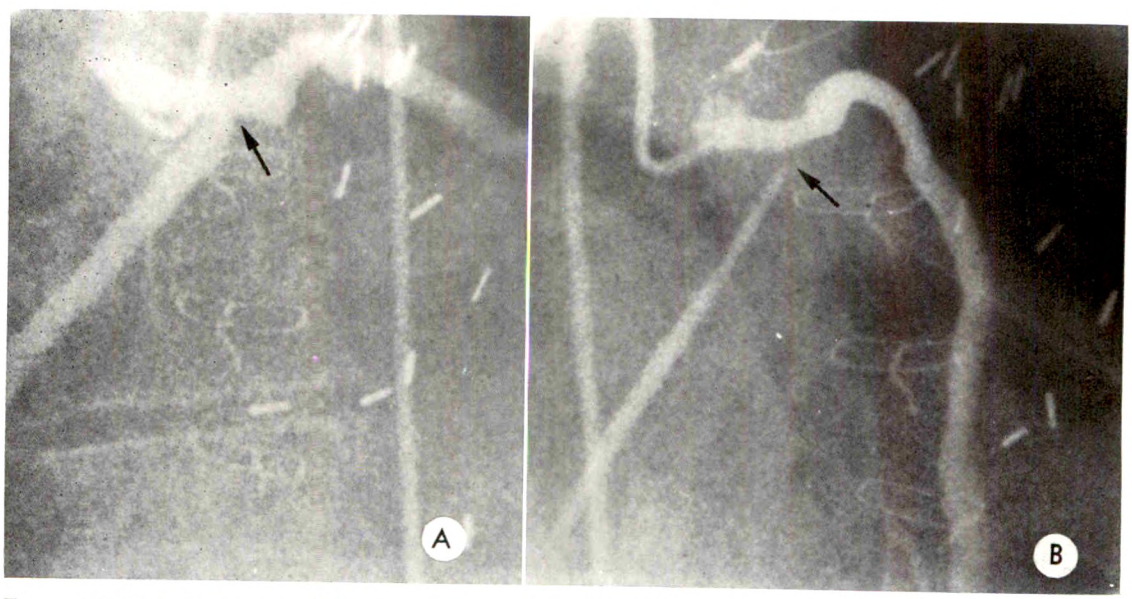


FIG. 3. (*A*) Early angiogram of a Y-shaped vein graft to circumflex and right coronary arteries. Both branches form a wide angle. There may be a mild stenosis at the origin of the branch to the right coronary artery (arrow). (*B*) Late evaluation 1 year after operation shows marked segmental narrowing (80–90 per cent) of proximal portion (arrow) of the graft to the right coronary artery. The remaining portion of the graft is also diffusely narrowed.

tion, patency rate was 90 per cent in 300 patients. Both studies consisted of single postoperative angiographic evaluation.

Patency rate may vary with the timing of the postoperative angiographic study. Thus, in the case of single evaluation of a large series of patients, if most patients are studied in the early postoperative period when patency rate is expected to be high (>90 per cent), late evaluation of the remaining patients in that series will hardly affect the over-all patency rate which

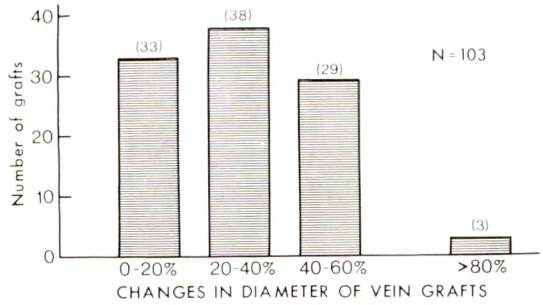


FIG. 4. Diffuse reduction of diameter of all grafts after 1 year. Reduction is expressed in percentage of the initial diameter.

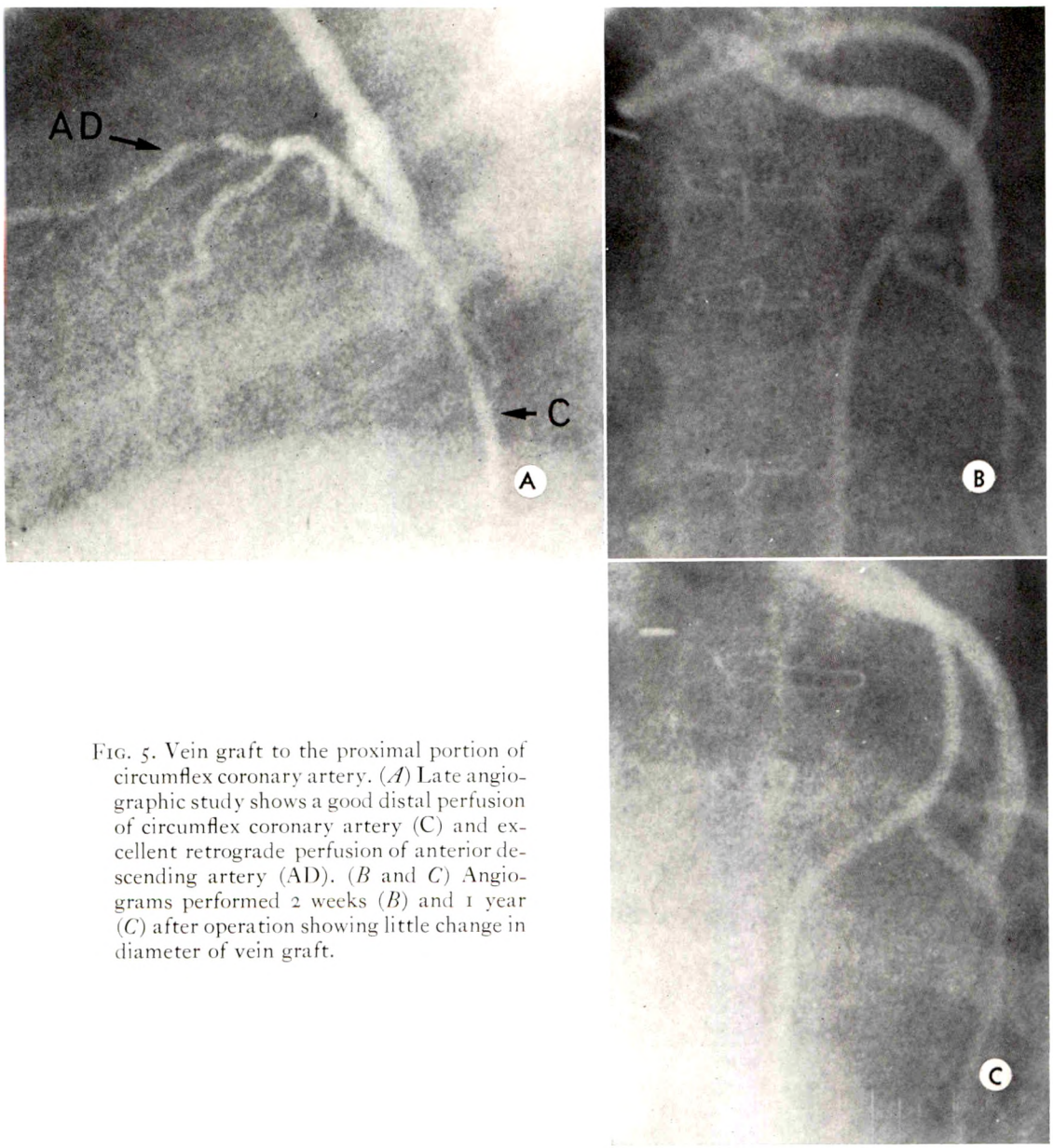


FIG. 5. Vein graft to the proximal portion of circumflex coronary artery. (*A*) Late angiographic study shows a good distal perfusion of circumflex coronary artery (C) and excellent retrograde perfusion of anterior descending artery (AD). (*B* and *C*) Angiograms performed 2 weeks (*B*) and 1 year (*C*) after operation showing little change in diameter of vein graft.

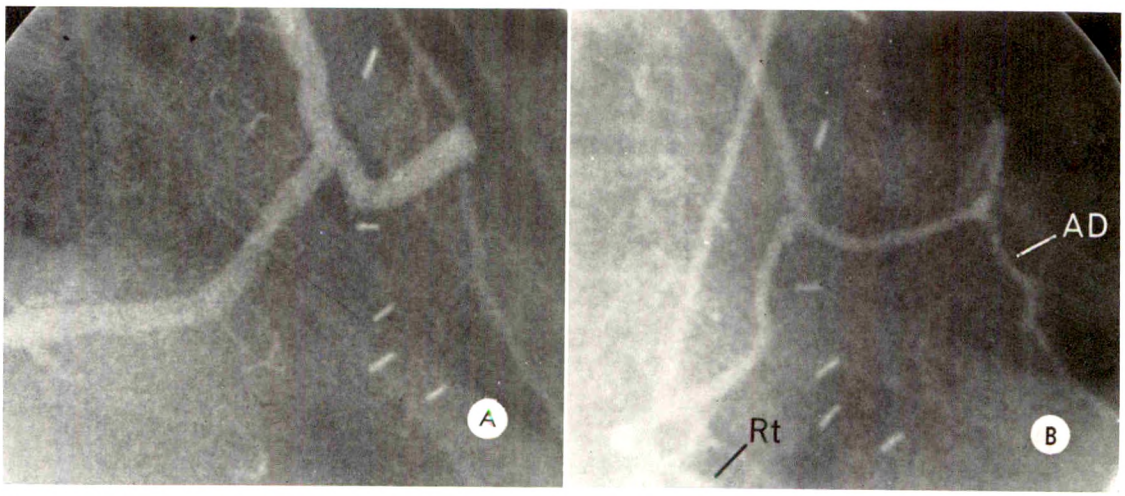


FIG. 6. (*A*) Early angiographic evaluation (2 weeks): Y graft to anterior descending and distal right coronary arteries. (*B*) Diffuse narrowing involving the main trunk of graft and its divisions to anterior descending (AD) and right coronary arteries (Rt). The residual caliber of the graft nearly equals that of the coronary arteries grafted.

would then in fact reflect the early patency rate. Similarly, the incidence of late stenosis or thrombosis of vein grafts may be unusually high when angiographic studies are performed only in symptomatic patients.

Although localized or diffuse narrowing of aortocoronary vein grafts have been previously observed by several authors,[12,13,16,17,20] the cause and incidence of graft stenosis are unknown. Johnson and his group first described 15 cases of narrowing of vein grafts in their early series.[13]

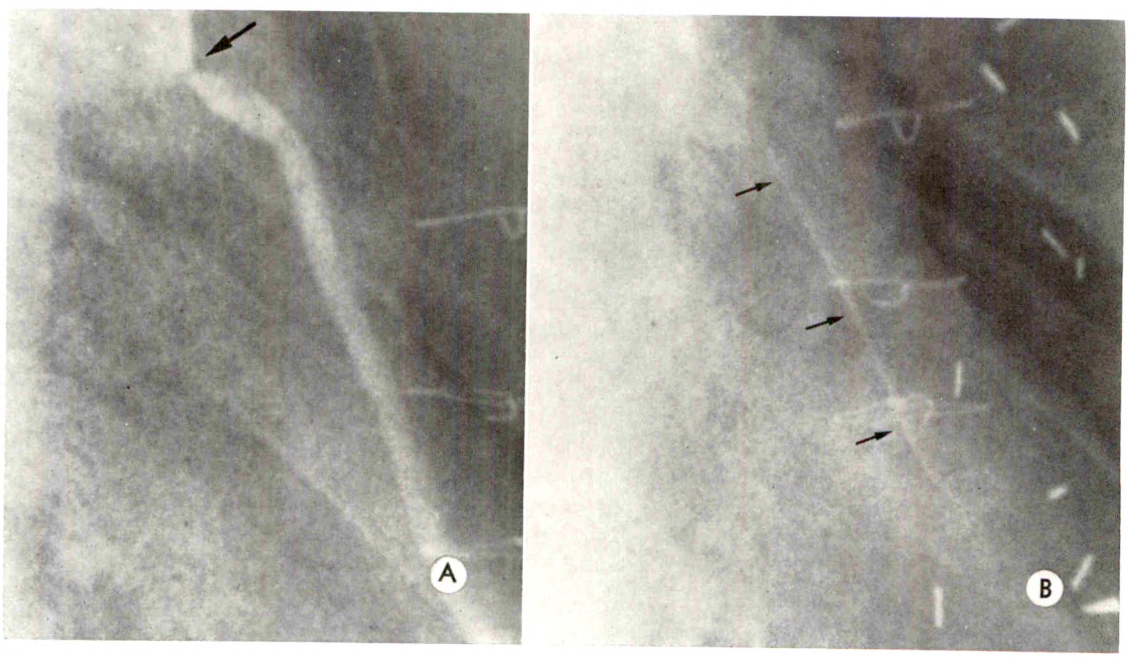


FIG. 7. (*A*) Graft to the anterior descending artery. Early angiographic evaluation shows good caliber of graft. There is a severe narrowing (arrow) of aortic anastomosis. The tip of the catheter could not be introduced into the graft. (*B*) One year after operation, the vein graft shows marked diffuse narrowing: approximately 1 mm. in diameter (arrows).

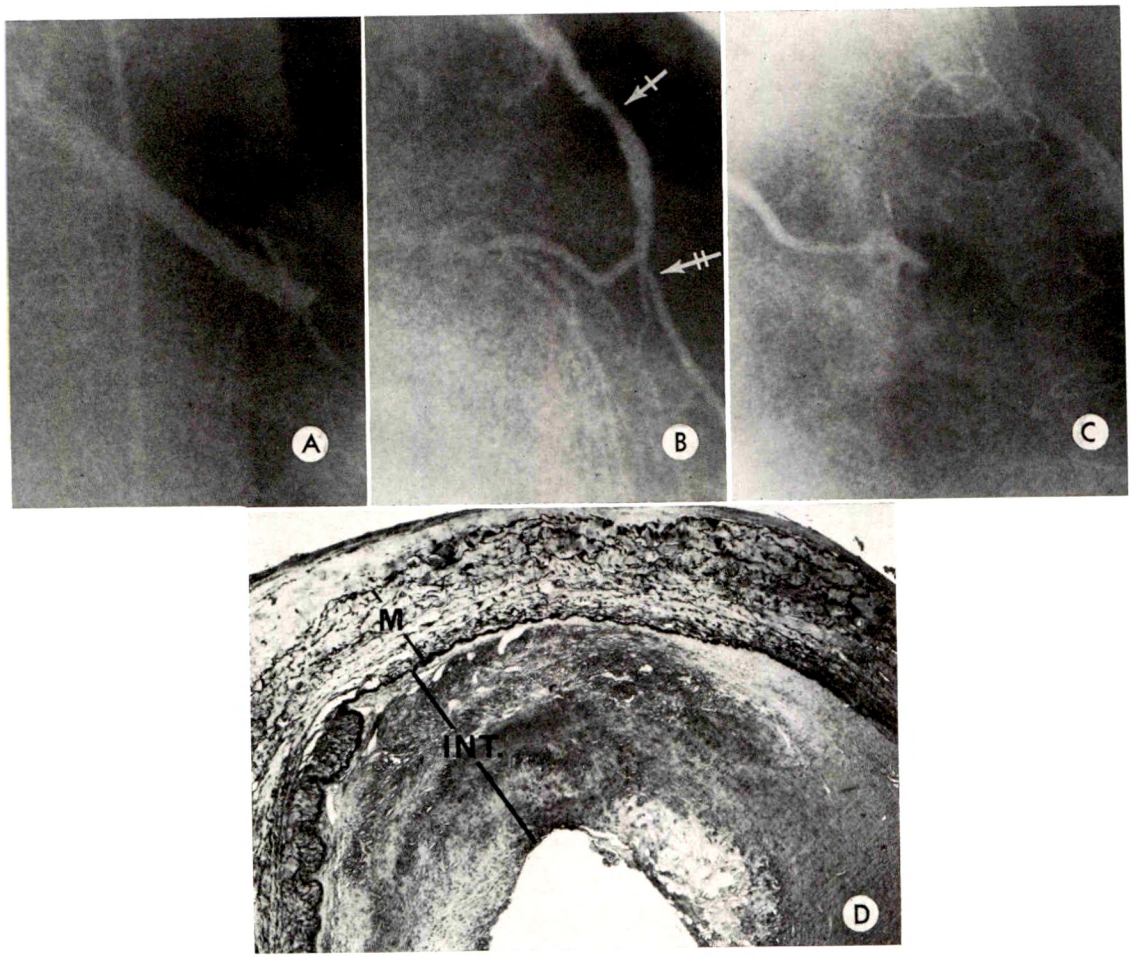


FIG. 8. (*A*) Early postoperative angiogram showing normal aspect of vein graft. (*B*) One year after operation, the angiogram demonstrates diffuse moderate narrowing (50 to 60 per cent) and segmental stenosis (75 per cent) in the middle portion of the vein graft (single crossed arrow). Note moderate traction of graft on anterior descending artery (double crossed arrow). (*C*) Two years after operation, the vein graft is completely occluded. Note opacification of proximal stump. (*D*) Histologic section of the vein graft showing marked intimal fibrous hyperplasia (INT.). M = medial layer. Weigert stain (×35).

Conversely, Morris and colleagues reported only 2 instances of fibrosis of vein graft in a large series of patients who were submitted to angiographic studies 1 to 30 months after operation.[19] Contracture at the site of anastomosis was believed responsible for late failure in the report of Sheldon and Grinfeld.[22]

Localized or diffuse narrowing of vein graft has also been described in femoropopliteal vein grafts.[6,7,18] Several factors have been invoked. Venous valves and the presence of twists or kinks in the vein graft may be responsible for segmental stenosis. Ischemia of the endothelial and medial layers may play a role in diffuse narrowing of vein grafts.[4]

In the present series, more than 70 per cent (26 of 36) of segmental stenoses were located in the proximal portion of vein grafts. Turbulence at the site of aortic anastomosis or the jet effect of a narrowed aortic orifice may explain the high incidence of proximal stenosis.

Distal run-off as assessed on the preoperative coronary arteriogram and by intraoperative blood flow measurements did not appear to be influential in the present series except in the case of severe diffuse stenoses where the so-called water-

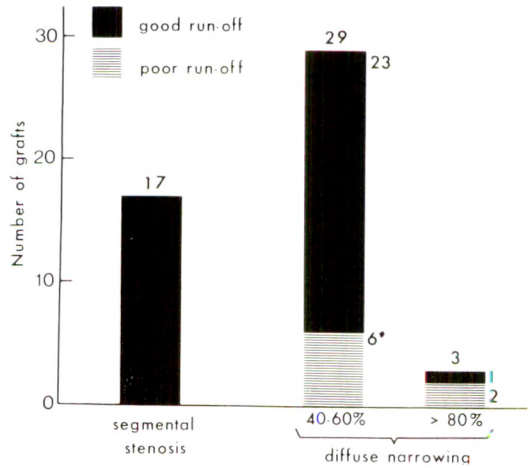


FIG. 9. There is no correlation between the distal coronary run-off and the presence of segmental or diffuse narrowing, except perhaps in the group with severe (>80 per cent) diffuse narrowing.

hammer effect of a restricted run-off may have been instrumental.[23] Severe stenoses at proximal aortic anastomosis may also impede blood flow and lead to progressive diffuse narrowing of vein graft (Fig. 7, *A* and *B*).

All aortocoronary vein grafts in place for more than 1 month in our own series[8] and for more than 2 months in the series of Vlodaver and Edwards[23] showed some degree of intimal fibrous proliferation.

At the present time, it is not known whether this process is self limited or is progressive. Longer follow-up studies are needed. The present study has nevertheless prompted several changes in the surgical technique which should prevent the occurrence of stenosis at the aortic orifice and which were aimed at minimizing ischemia and trauma to the vein graft.

SUMMARY

Two separate angiographic studies were carried out in 96 patients early (2 weeks) and late (12–18 months) after aortocoronary vein grafting. Eleven of the initial 144 grafts were found to be occluded on the early study. Thirty of the remaining 133 grafts were occluded on the late study —a combined patency rate of 72 per cent.

Most grafts displayed some reduction in size on the second study. Mean reduction was 30 per cent. This reduction in caliber was negligible to mild in 71 grafts, moderate (40 to 60 per cent reduction) in 29 grafts and was severe (>80 per cent) in 3 grafts.

In addition to the diffuse decrease in size, there were 36 instances of localized narrowing on the late study. This narrowing was mild in 19 grafts, moderate in 14 grafts and severe in 3 grafts.

It is concluded that, although some degree of narrowing is to be expected in aortocoronary vein grafts after 1 year, certain factors are likely responsible for the more severe narrowing and for late thrombosis of vein grafts.

Early and late angiographic studies can help the cardiologist and surgeon in the detection and prevention of the factors which, in some cases, may be responsible for late occlusion or stenosis of aortocoronary vein grafts.

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REFERENCES

1. BOURASSA, M. G., LESPÉRANCE, J., CAMPEAU, L., and SIMARD, P. Factors influencing patency of aortocoronary vein grafts. *Circulation*, 1972, *45–46* (Suppl. I), I-79, I–85.
2. BOURASSA, M. G. Preshaped polyethylene catheters for percutaneous hemodynamic and angiographic studies. *Angiology*, 1971, *22*, 320–331.
3. BOURASSA, M. G., LESPÉRANCE, J., SALTIEL, J., CASTONGUAY, Y. M., and GRONDIN, P. Changes in left ventricular dynamics and functions following successful aorto-coronary bypass surgery. (Abstr.) *Circulation*, 1970, *41–42* (Suppl. III), III–13.
4. BRODY, W. R., ANGELL, W. W., and KOSEK, J. C. Histologic fate of venous coronary artery bypass in dogs. *Am. J. Path.*, 1972, *66*, 111–130.
5. CAMPEAU, L., ALONZO, F., ELIAS, G., and BOURASSA, M. G. Left ventricular performance during exercise before and after aorto-coronary vein graft surgery. (Abstr.) *Circulation*, 1971, *43–44* (Suppl. III), III-148
6. DE WEESE, J. A., and ROB, C. G. Autogenous

venous bypass grafts five years later. *Ann. Surg.*, 1971, *174*, 346–356.

7. Downs, A. R. Repair of late vein graft occlusions. *Arch. Surg.*, 1971, *103*, 639–643.
8. Grondin, C. M., Bourassa, M. G., Lespérance, J., Simard, P., Castonguay, Y. R., Campeau, L., and Grondin, P. Late stenosis and pathologic changes in aorto-coronary vein grafts: incidence and histological features. To be published.
9. Grondin, C. M., Castonguay, Y. R., Lepage, G., Meere, C., and Grondin, P. Aortocoronary bypass grafts: early post-operation angiographic evaluation and reexploration for stenosis or thrombosis of vein graft. *Arch. Surg.*, 1971, *103*, 535–538.
10. Grondin, C. M., Castonguay, Y. R., Lespérance, J., Bourassa, M. G., Campeau, L., and Grondin, P. Attrition rate of aorta-to-coronary saphenous vein grafts after one year: study in consecutive series of 96 patients. To be published.
11. Grondin, C. M., Lepage, G., Castonguay, Y. R., Meere, C., and Grondin, P. Aorto-coronary bypass graft: initial blood flow through graft and early post-operative patency. *Circulation*, 1971, *44*, 815–819.
12. Grondin, C. M., Meere, C., Castonguay, Y. R., Lepage, G., and Grondin, P. Progressive and late obstruction of aorto-coronary venous bypass graft. *Circulation*, 1971, *43*, 698–702.
13. Johnson, W. D., Auer, J. E., and Tector, A. J. (Abstr.). Late changes in coronary vein grafts. (Abstr.) *Am. J. Cardiol.*, 1970, *26*, 640.
14. Johnson, W. D., Flemma, R. J., Harding, H. W., Cooper, G. N., and Lepley, D., Jr. Surgical principles in direct reconstruction of left coronary flow. *Ann. Thoracic Surg.*, 1970, *10*, 141–151.
15. Lespérance, J., Bourassa, M. G., Biron, P., Campeau, L., and Saltiel, J. Aorta-to-coronary saphenous vein grafts: preoperative angiographic criteria for successful surgery. To be published.
16. Levin, D. C., Baltaxe, H. A., and Carlson, R. G. Bidirectional coronary artery blood flow. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *113*, 554–561.
17. Marti, M. C., Bouchardy, B., and Cox, J. N. Aorto-coronary bypass with autogenous vein grafts: histopathological aspects. *Virchows Arch. path. Anat.*, 1971, *352*, 255–266.
18. McNamara, J., Darling, R. C., and Linton, R. R. Segmental stenosis of saphenous vein autografts. *New England J. Med.*, 1967, *277*, 290.
19. Morris, G. C., Reul, G. J., Howell, J. F., Crawford, S. E., Chapman, D. W., Beazley, H. L., Winters, W. L., Peterson, P. K., and Lewis, J. M. Follow-up results of distal coronary artery bypass for ischemic heart disease. *Am. J. Cardiol.*, 1972, *29*, 180–185.
20. Rösch, J., Judkins, M. P., Green, G. S., and Kidd, H. Aorto-coronary venous bypass grafts: angiographic study of 84 cases. *Radiology*, 1972, *102*, 567–573.
21. Saltiel, J., Lespérance, J., Bourassa, M. G., Castonguay, Y. R., Campeau, L., and Grondin, P. Reversibility of left ventricular dysfunction following aorto-coronary bypass grafts. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *110*, 739–746.
22. Sheldon, W. C., and Grinfeld, L. Direct myocardial revascularization: venous autograft technique; post-operative assessment. *Surg. Clin. North America*, 1971, *51*, 1043–1049.
23. Vlodaver, Z., and Edwards, J. E. Pathologic changes in aorto-coronary arterial saphenous vein grafts. *Circulation*, 1971, *44*, 719–728.

CORONARY ARTERIOVENOUS FISTULA*

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IN 1958, only 22 cases of coronary arteriovenous fistula were found in the literature.[14] Now, it is no longer an oddity and more and more cases are being reported.[1–11,15,16] Congestive heart failure and death due to coronary arteriovenous fistula have been reported;[14] thus it is important to establish the diagnosis early and determine if surgery is indicated.

The definitive diagnosis of coronary arteriovenous fistula is made by aortography and coronary arteriography. Since these techniques are now readily available and are without serious untoward effects, every patient with an atypical continuous chest murmur should have these tests routinely. Indeed, coronary arteriovenous fistulas were encountered in many instances during coronary arteriography done because of angina.[3] Review of the literature has shown that a large number of patients have been alleviated of their murmurs and had effective obliteration of the fistula, especially with bypass technique.[3,10]

REPORT OF CASES

CASE I. *Left circumflex coronary artery—right ventricular fistula.* A 3 year old male was admitted in May 1963, because of a cardiac murmur heard soon after birth. He had always been asymptomatic, but increase in size of the right heart and plethoric lung fields became evident (Fig. 1*A*).

Physical examination showed a continuous murmur over the left lower end of the sternum. The blood pressure was 110/140 mm. Hg. The electrocardiogram was normal.

Cardiac catheterization on March 6, 1962, showed a left-to-right shunt into the right ventricle.

Retrograde aortography demonstrated a coronary arteriovenous fistula between the left coronary artery and the right ventricle (Fig. 1, *B* and *C*).

On May 7, 1963, ligation of the left circumflex coronary-right ventricular fistula was made; and the patient has remained well for the past 8 years.

CASE II. *Left coronary artery—coronary sinus arteriovenous fistula.* A 32 year old woman was admitted on October 3, 1965, because of a heart murmur since childhood. She had had palpitation, increasing dyspnea, and ankle edema for 2 years. She had been digitalized and the palpations had decreased.

On physical examination, a continuous murmur (Grade 3) was heard throughout the cardiac cycle at the right second intercostal space and along the left sternal border. The blood pressure was 140/40 mm. Hg. The electrocardiogram showed incomplete atrioventricular dissociation with sinus arrhythmia.

Roentgenogram of the chest revealed cardiac enlargement and plethoric lung fields (Fig. 2*A*).

Cardiac catheterization demonstrated a left-to-right shunt at the level of the right atrium with pulmonary flow of 8.3 l./min., while the systemic flow was 4.1 l./min. The left ventricular pressure was 93/12, and the aortic was 97/49 mm. Hg.

Intravenous angiocardiography showed enlargement of the cardiac chambers, plethoric lungs, a left-to-right shunt with a left coronary artery fistula (Fig. 2, *B–E*).

Aortography revealed a large coronary artery—right heart fistula (Fig. 2, *F* and *G*).

Operation on October 13, 1965, showed some increased pericardial fluid with a large tortuous left coronary artery arising from the left side of the aorta (measuring 15 to 18 mm. in diameter) and coursing into the right atrium. Ligation of the coronary artery proximal to the arteriovenous fistula was performed without change in the electrocardiogram. She did well after surgery, but after a day began having paroxysmal supraventricular tachycardia followed by nodal and atrial premature contractions, which persisted despite procaine-amid therapy. She improved and was discharged on November 15, 1965.

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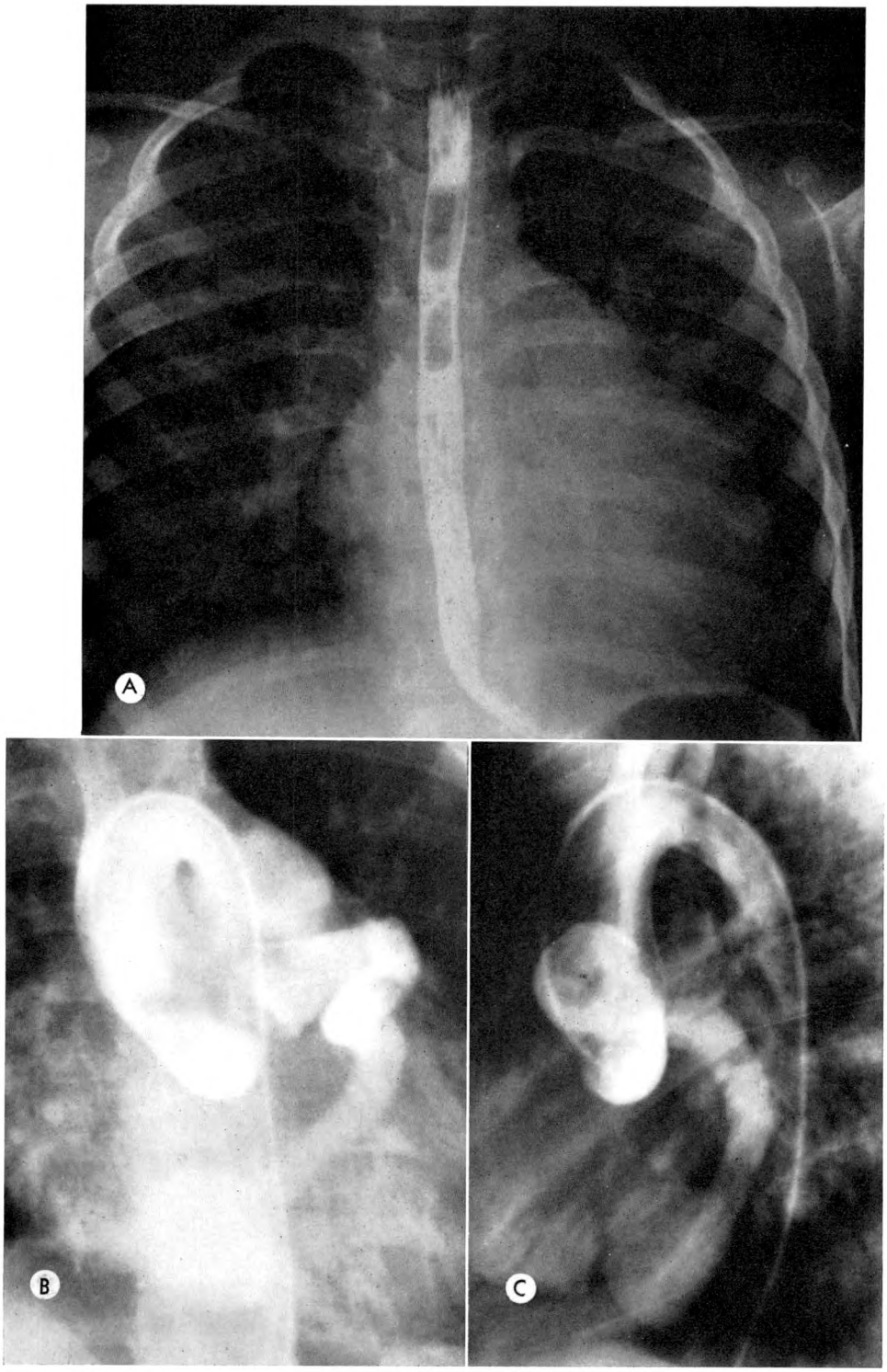


Fig. 1. Case 1. (*A*) Frontal esophagogram shows enlargement of the heart and plethoric lung fields. (*B*) Frontal aortogram shows the huge left coronary fistula draining into the right ventricle. (*C*) Lateral biplane view of *B* shows the left coronary artery—right ventricular fistula.

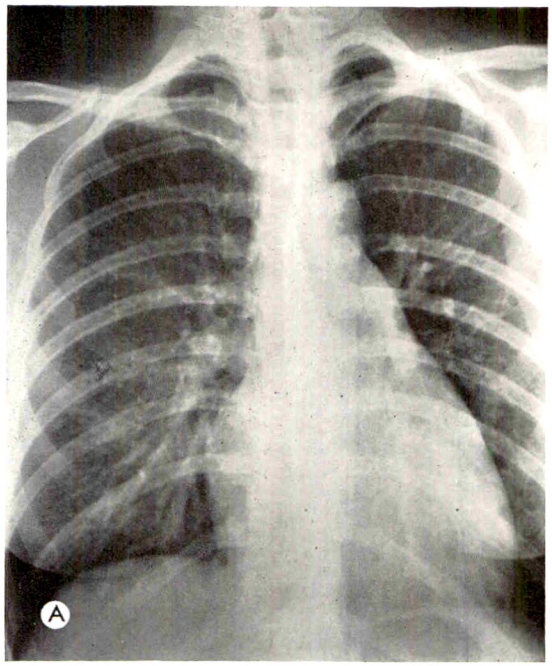


FIG. 2. Case II. (*A*) Frontal esophagogram of the chest showing enlargement of the heart and plethoric lungs.

She was seen in the Cardiac Clinic on November 29, 1965, and reported that she was feeling well and doing light housework. A harsh shrill murmur of mitral regurgitation was heard at the apex radiating medially and laterally. She died suddenly 2 weeks later at home (December 9, 1965). Autopsy was not done.

CASE III. *Right coronary artery—right atrium fistula.* A 29 year old Chinese woman first learned of a heart murmur at the age of 10 years, while she was living in China. Her growth and development had been normal, and she had been completely free of symptoms. Moreover, she had been a champion sprinter during high school.

On physical examination, the blood pressure was 100/50 mm. Hg. The pulse was 64 per minute and regular; respirations were 14 per minute. She was well developed and well nourished and did not appear ill. Positive physical findings were limited to the heart. The position of maximum impulse of the heart was in the fifth intercostal space in the mid-clavicular line. There was a continuous machinery murmur (Grade 3/4) best heard along the left sternal border, but loudest in the second left intercostal space.

The urinalysis and blood studies were normal. The fasting blood sugar was 70 mg. per cent and the blood urea nitrogen was 16 mg. per cent.

Roentgenogram of the chest was normal (Fig. 3*A*).

The electrocardiogram revealed sinus bradycardia at a rate of 58 per minute.

Phonocardiogram recorded a faint continuous systolic and diastolic murmur at all areas, with the systolic component maximum at the second right intercostal space and S2 split physiologically. An early systolic ejection sound was present at the apex.

Retrograde aortography demonstrated no communication between the aorta and pulmonary artery, ruling out a patent ductus arteriosus. There was aneurysmal deformity of the right coronary sinus and the proximal right coronary artery (Fig. 3, *B* and *C*). The dilated portion of the right coronary artery appeared to communicate with the right atrium, while the second normal appearing branch passed inferiorly in the usual direction. On the basis of the continuous murmur and the roentgenographic findings, an arteriovenous fistula involving the right coronary artery and the right atrium was diagnosed.

On July 24, 1968, an operation was performed through a median sternotomy incision. The pericardium was opened using an inverted T-shaped incision. The right coronary artery was found to be greatly enlarged and tortuous, measuring 6 mm. in diameter. As it arose from the right coronary sinus it passed downward, angulating to the right and gave off a branch which extended into the atrioventricular groove and measured only 3 mm. in diameter. The artery then turned abruptly posteriorly and appeared to enter the right atrium adjacent to the appendage of the right atrium. The dilated portion of the vessel was dissected free at its origin from the right coronary artery and at the point where it entered the right atrium. During a 7 minute trial occlusion of the fistula with a non-crushing clamp, there was no significant change in the electrocardiogram, but there was a rise in systolic blood pressure from 90 to 104 mm. Hg. When the clamp was released, the systolic blood pressure fell to the preocclusion level. The fistula was then doubly ligated with No. 4 heavy braided silk, at its origin from the right coronary artery near the atrioventricular groove, and at the point where it entered the

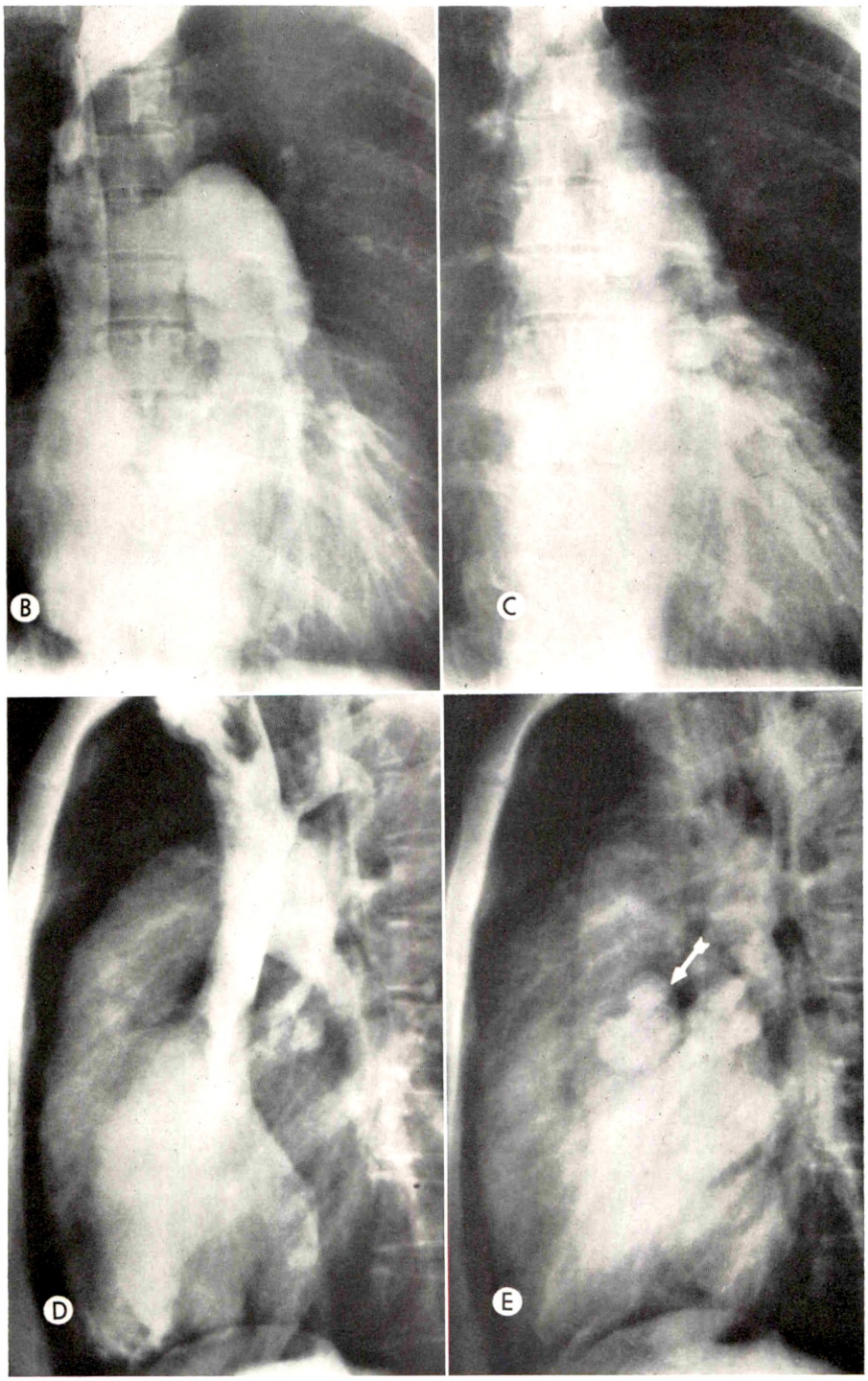


FIG. 2. (*B*) Frontal intravenous angiocardiogram shows enlargement of the right atrium, ventricle, pulmonary artery, and branches. (*C*) When the left heart is opacified, there is enlargement of the left ventricle and opacification of the pulmonary artery and branches. (*D*) Lateral angiocardiogram, biplane view of *B*, also shows the enlarged right atrium, ventricle, pulmonary artery and branches. (*E*) Lateral view, biplane view of *C*, shows the large left ventricle and a cluster of opacified vessels adjacent to the left side of the aorta (arrow).

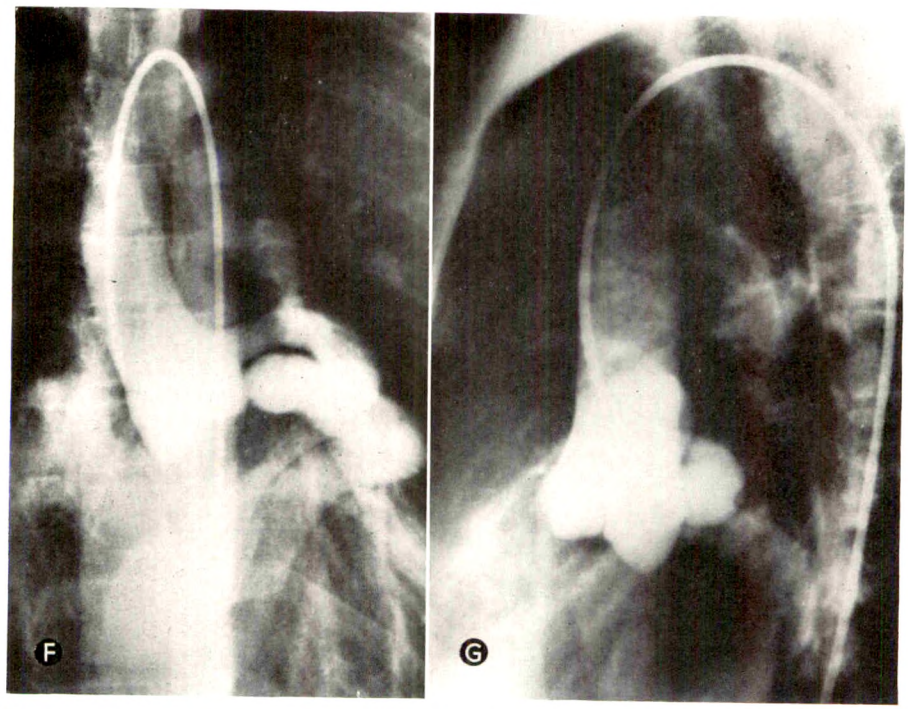


FIG. 2. (*F*) Frontal aortogram reveals the huge left coronary artery draining into the right heart. (*G*) Biplane lateral view of *F* also shows the large left coronary artery proceeding into the right heart.

right atrium. The mid-portion of the vessel was transfixed with a No. 1 braided silk. The thrill which had been palpable at the point where the vessel entered the right atrium was completely abolished. Systolic pressure was 10 mm. Hg higher than the preligation pressure. The median sternotomy incision was closed in the usual fashion.

The patient's postoperative course was smooth and uncomplicated. She was discharged from the hospital 10 days following operation at which time the heart sounds were normal and the murmur had disappeared. She has remained free of symptoms since her operation over 4 years ago; and had a normal pregnancy and delivery 2 years ago.

CASE IV. *Right coronary artery—right ventricle fistula.* A 23 year old man was asymptomatic but had a cardiac murmur since birth. He was first seen in The New York Hospital in 1951, and had a left precordial bulge, a loud (Grade 4) holosystolic murmur at the apex, and a soft blowing (Grade 1) diastolic murmur over the left border of the sternum.

The electrocardiogram was normal and the blood pressure was 135/80 mm. Hg.

The roentgenogram of the chest showed a normal sized heart. Intravenous angiocardiography revealed a left-to-right shunt. Cardiac catheterization (at another hospital) also had shown a left-to-right shunt.

He was readmitted in April 1968. The heart was not enlarged but the pulmonary vasculature was plethoric (Fig. 4*A*). Aortography showed a huge right coronary artery—right ventricle fistula (Fig. 4, *B* and *C*).

On August 21, 1968, operation by C. W. Lillehei disclosed a 1.5 cm. in diameter right coronary artery extending downward along the right atrioventricular groove to the bottom of the surface of the right ventricular cavity. Multiple slightly enlarged myocardial branches were also seen along the course of the coronary artery. The pulmonary artery was slightly enlarged and had a systolic thrill at the pulmonic valve. After cardiopulmonary bypass, opening of the right ventricle was made. A small probe was passed between the trabeculae into a branch of the anomalous right coronary artery. Two other branches appeared to drain normally into the myocardium. The fistulous opening was closed and the murmurs disappeared.

The patient has been entirely well for the past 3 years.

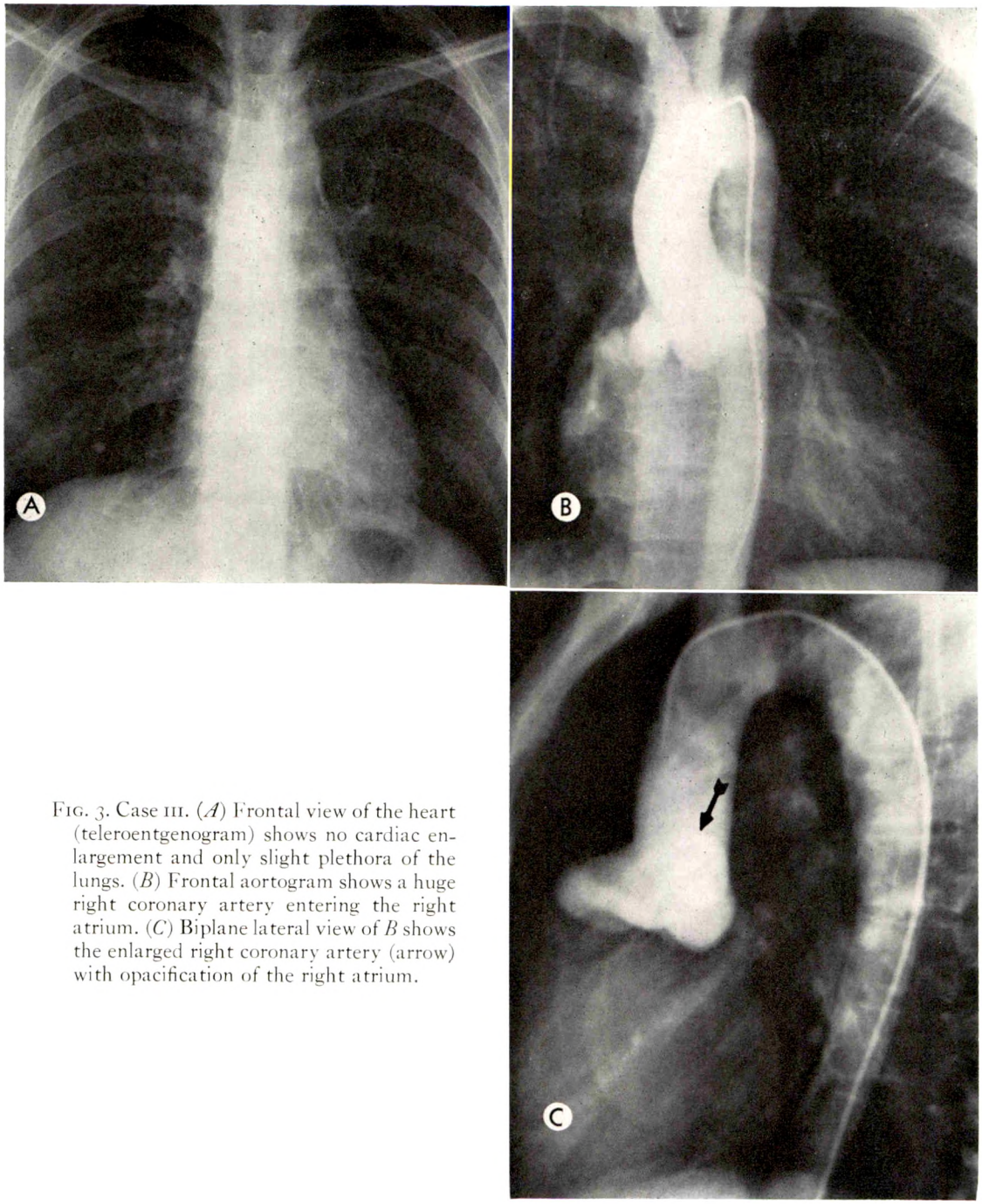


Fig. 3. Case III. (*A*) Frontal view of the heart (teleroentgenogram) shows no cardiac enlargement and only slight plethora of the lungs. (*B*) Frontal aortogram shows a huge right coronary artery entering the right atrium. (*C*) Biplane lateral view of *B* shows the enlarged right coronary artery (arrow) with opacification of the right atrium.

DISCUSSION

There has been discussion in the literature[3] whether the name of arteriovenous fistula is the correct term for the coronary anomalies herein reported. In our presentation of arteriovenous fistula in 1958,[14] it was stated that using the term when the coronary arteries communicated with cardiac chambers of the pulmonary artery was open to criticism. Effler and colleagues[3] apparently agree that the fact that the literature usually categorized the lesions as

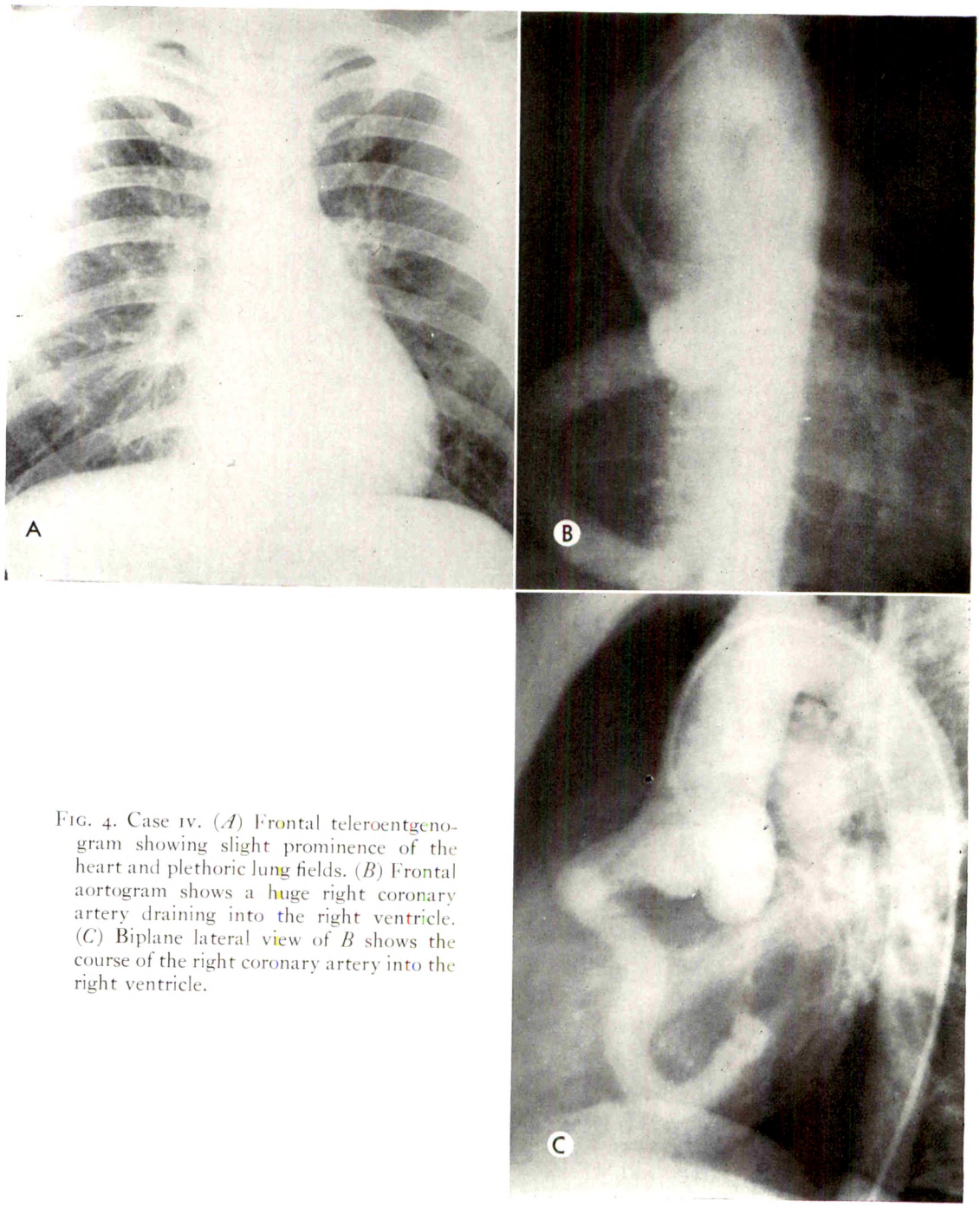


FIG. 4. Case IV. (*A*) Frontal teleroentgenogram showing slight prominence of the heart and plethoric lung fields. (*B*) Frontal aortogram shows a huge right coronary artery draining into the right ventricle. (*C*) Biplane lateral view of *B* shows the course of the right coronary artery into the right ventricle.

arteriovenous fistula makes this the preferable term. In this publication, we have retained the term coronary arteriovenous fistula in the title, but in each case have mentioned the coronary artery involved and the terminal site of the communication.

As indicated in a recent publication,[13] the anomalous origin of a coronary artery from a pulmonary artery is *not* an arteriovenous fistula, but an anomalous origin of the coronary artery.

Whereas in 1958, only 22 cases of coro-

nary arteriovenous fistula were found in the literature,[14] there are now well over 175 cases, and more than half have had operations for correction of the anomalies.[10] Every kind of anatomic combination between the coronary arteries and the 4 cardiac chambers, pulmonary artery, vena cava, and pulmonary veins have been recorded.[1–12,14,17] Slightly over half the fistulas originate from the right coronary artery and 92 per cent of all fistulas from either coronary artery enter the right side of the heart creating a left-to-right shunt[10] (Fig. 1, *B* and *C*; 2, *F* and *G*; 3, *B* and *C*; and 4, *B* and *C*). Only 1 of the cases herein reported had heart failure (Fig. 2, *A–E*). In the literature, other cases of cardiac failure due to coronary arteriovenous fistula are recorded.[14] Other uncommon complications are aortic sinus rupture,[9] and subacute bacterial endocarditis.[16]

Suspicion of coronary arteriovenous fistula depends upon hearing a continuous murmur resembling that of ductus arteriosus but atypical in location (Cases I–IV). Catheterization of the heart may reveal a left-to-right shunt, but may confuse the diagnosis.[14] Coronary arteriography and aortography are the definitive means of diagnosis[3,10,13] (Fig. 1, *B* and *C*; 2, *F* and *G*; 3, *B* and *C*; and 4, *B* and *C*). Intravenous angiocardiography, although it showed the left-to-right shunt and abnormal vessels along the left border of the aortic sinuses, failed to reveal the definitive data well (Fig. 2, *B–E*). In Case IV, intravenous angiocardiography performed at a time when aortography had not come into general use caused delay in diagnosis. However, the 17 year delay did not harm the patient, who had remained asymptomatic, and it probably benefited him because bypass techniques were now available for correction of the shunt.

For treatment, an individualized approach, depending upon the anatomic distribution of the fistula, is advocated.[3,10,14] As in treatment of pulmonary arteriovenous fistula, which first began with lobectomy, then proceeded to segmental removal, and finally excision of the fistula,[15] localized excision of the fistulous tract is recommended. Although many patients with coronary arteriovenous fistula are asymptomatic, some may have angina and this has been alleviated by surgical correction of the abnormal shunt.[3]

CONCLUSIONS AND SUMMARY

Coronary arteriovenous fistulas are no longer rare. They should be suspected whenever there are atypical continuous murmurs over the sternum. Aortography and coronary arteriography reveal the origin of the anomalous coronary arteries and show their termination, most often in the right heart structures.

Whether to operate is an individual decision. In some instances, because cardiac failure may result from coronary arteriovenous fistula, early operation is advised.

Aortography and coronary arteriography usually delineate the problems to be encountered, and the availability of bypass techniques makes surgery even more successful.

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REFERENCES

1. Amplatz, C., Aquirre, J., and Lillehei, C. W. Coronary arteriovenous fistula into main pulmonary artery: preoperative diagnosis by selective aortography. *J.A.M.A.*, 1960, *172*, 1384.
2. Currarino, G., Silverman, F. N., and Landing, B. H. Abnormal congenital fistulous communications of coronary arteries. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1959, *82*, 392–402.
3. Effler, D. B., Sheldon, W. C., Turner, J. J., and Groves, L. K. Coronary arteriovenous fistulas: diagnosis and surgical management; report of 15 cases. *Surgery*, 1967, *61*, 41–50.
4. Engle, M. A., Goldsmith, E. I., Holswade, G. R., Goldberg, H. P., and Glenn, F. Congenital coronary arteriovenous fistula: diagnostic evaluation and surgical correction. *New England J. Med.*, 1961, *264*, 856–858.
5. Floyd, W. L., Young, W. G., and Johnsrude, I. S. Coronary arterial—left atrial fistula: case with obstruction of inferior vena cava by giant left atrium. *Am. J. Cardiol.*, 1970, *25*, 716–722.

6. GENSINI, G. G., PALACIO, A., and BUONANNA, C. Fistula from circumflex coronary artery to superior vena cava. *Circulation*, 1966, *33*, 297–301.
7. HALLMAN, G. L., COOLEY, D. A., MCNAMARA, D. G., and LATSON, J. R. Single left coronary artery with fistula to right ventricle: reconstruction of two coronary systems with dacron graft. *Circulation*, 1965, *32*, 293–297.
8. HIPONA, F. A. Congenital coronary arterial fistula to persistent left superior vena cava. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *97*, 355–358.
9. HOLMES, J. C., FOWLER, N. O., and HELMSWORTH, J. A. Coronary arteriovenous fistula and aortic sinus rupture. *A.M.A. Arch. Int. Med.*, 1966, *118*, 43–54.
10. MCNAMARA, J. J., and GROSS, R. E. Congenital coronary artery fistula. *Surgery*, 1969, *65*, 59–69.
11. MYER, M. H., STEPHENSON, H. E., JR., KEATS, T. E., and MART, J. M. Coronary artery resection for giant aneurysmal enlargement and arteriovenous fistula: five-year follow up. *Am. Heart J.*, 1967, *74*, 603–613.
12. SERRATTO, M., and KEZDI, P. Congenital coronary artery—right ventricular fistula corrected surgically. *Brit. Heart J.*, 1965, *27*, 781–785.
13. STEIN, H. L., HAGSTROM, J. W., EHLERS, K. H., and STEINBERG, I. Anomalous origin of left coronary artery from pulmonary artery: angiographic and pathologic study of case. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1965, *93*, 320–330.
14. STEINBERG, I., BALDWIN, J. S., and DOTTER, C. T. Coronary arteriovenous fistula. *Circulation*, 1958, *17*, 372–390.
15. STEINBERG, I. Diagnosis and surgical treatment of pulmonary arteriovenous fistula: report of three new cases and review of nineteen consecutive cases. *S. Clin. North America*, 1961, *41*, 523–535.
16. TSAGARIS, T. J., and HECHT, H. H. Coronary artery aneurysm and subacute bacterial endocarditis. *Ann. Int. Med.*, 1962, *57*, 116–121.
17. UPSHAW, C. B., JR. Congenital coronary arteriovenous fistula; report of case with analysis of seventy-three reported cases. *Am. Heart J.*, 1962, *63*, 399–404.

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THE PITFALLS IN INTERPRETING ARTIFICIAL PNEUMOPERICARDIUM*

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THE introduction of air into the pericardial space after pericardiocentesis has long been a standard procedure for roentgenographically examining the pericardial and cardiac surfaces.[2,3,6,8] Short of surgery or autopsy, this is the only technique for direct visualization of the pericardium. When incorporated with the clinical and other laboratory data, this method facilitates an accurate diagnosis of a wide variety of effusive pericardial diseases. Most of the time the roentgenograms are self-explanatory and easy to understand. Occasionally, however, erroneous diagnoses may be made and unnecessary surgery undertaken because of misinterpretation of some normal but unfamiliar phenomena under artificial circumstances. Indeed, the heart and vessels, when enveloped in a thick blanket of air within a free pericardial cavity, pulsate much more vigorously because of much greater freedom to expand and to move. One has to become accustomed to such altered anatomy and dynamics of the heart to avoid erroneous diagnoses and consequent errors in management of the patient.

It is the purpose of this communication to describe the normal findings of the heart in artificial pneumopericardium utilizing illustrative case reports.

REPORT OF CASES

CASE 1. A 63 year old female was admitted to the hospital because of recurrent episodes of palpitation, weakness and shortness of breath.

On physical examination, she appeared chronically ill but in no distress. Blood pressure was 110/70 mm. Hg, and the pulse was 70 per minute and regular. The neck veins were flat in the supine position, and no paradoxic pulse was noted. The lungs were clear, while the cardiac dullness was greatly enlarged to both sides. The heart sounds were quite distant. A mitral diastolic rumble and an opening snap were faintly audible, consistent with mitral stenosis. The extremities showed no edema.

Laboratory findings were unremarkable except for the low voltage and T wave changes on the electrocardiograms.

Chest roentgenograms showed marked enlargement of the cardiac silhouette, consistent with a pericardial effusion. Pericardiocentesis was performed and 1,000 ml. of clear straw colored fluid was removed, which did not clot, had a specific gravity of 1.014, and contained only a few polymorphonuclear and mononuclear cells. Bacterial and tuberculous smears and cultures were negative. A cell block of the sediment showed some atypical cells, but no definite diagnosis was made.

Introduction of air into the pericardium following pericardiocentesis was performed and roentgenograms in different projections taken. In the posteroanterior view, there were peculiar shadows extending dome-like above the layer of fluid on either side of the pericardium. These were thought possibly to be tumor masses (Fig. 1, *A* and *B*).

Exploratory thoracotomy was performed which revealed nothing except a large amount of pericardial fluid and a thin, essentially normal pericardium. It was decided then to explore the mitral valve, which was thought to be stenotic and calcified. A closed mitral commissurotomy was performed.

Her postoperative course was uneventful. The pathologic report showed both the parietal and visceral pericardium to be slightly thickened and contained chronic inflammatory cells. Since the operation, she had been on steroid therapy with considerable improvement. Her steroid therapy was considered no longer needed and was discontinued 3 years later.

Comment. This lady apparently had a benign idiopathic pericarditis with effusion which responded to steroid therapy, although we cannot

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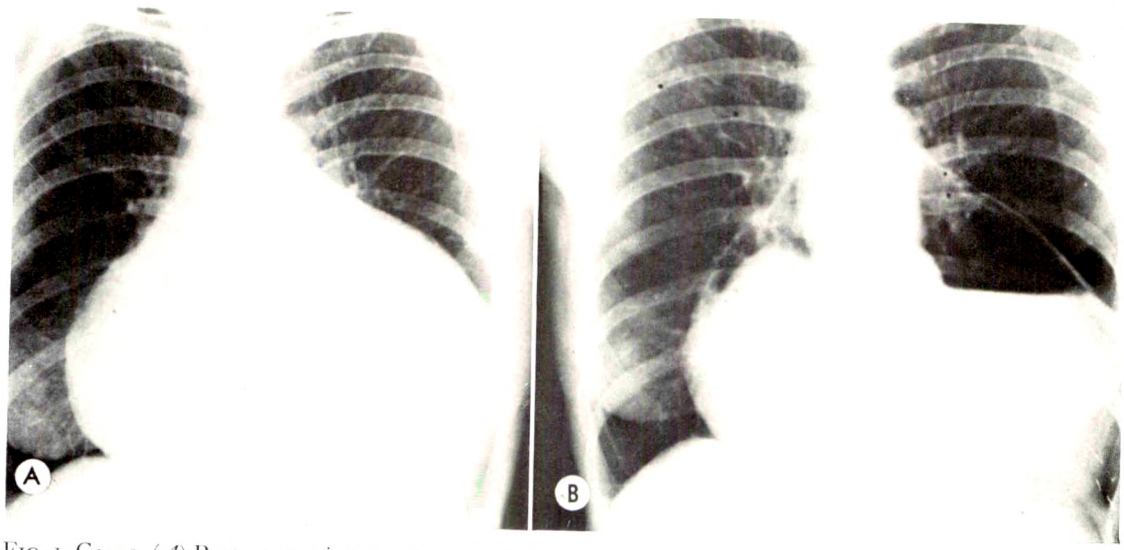


FIG. 1. Case I. (*A*) Posteroanterior roentgenogram showing generalized enlargement of the cardiac silhouette. (*B*) Posteroanterior view of induced pneumopericardium. Note the dome-like density above the fluid level beside the heart on the right, probably representing a fluid wave. A smaller rounded density above the fluid level along the left heart border either represents a fluid wave or an enlarged left atrial appendage.

rule out the mitral stenosis as playing a role in the pericardial effusion. The dome-like densities above the fluid level on either side of the heart within the pneumohydropericardium most likely represented large fluid waves produced by vigorous cardiac action, although the smaller density on the left side could represent a large left atrial appendage.

CASE II. A 69 year old white male came into the hospital with progressive malaise, exertional dyspnea, tachycardia, and mild pedal edema for 3 months.

Positive physical findings included neck vein distention, bilateral basilar rales, enlarged cardiac dullness, S_4 gallop, distended abdomen with hepatomegaly and no cardiac murmur.

Roentgenographic examination showed evidence of pleural and pericardial effusions. An electrocardiogram showed low voltage and nonspecific ST-T wave changes. Hemodynamics at cardiac catheterization were consistent with cardiac tamponade.

Pericardiocentesis was performed and 750 ml. of purple fluid was removed. The spun down sediment showed abnormal cells consistent with lymphosarcoma. The nodular appearance of the upper anterior border of the cardiovascular silhouette in the pneumopericardium was considered to be tumor implantation on the epicardium (Fig. 2). The visualized parietal pericardium in both posteroanterior and lateral projections, nevertheless, appeared thin and smooth (Fig. 3). The patient was treated with irradiation and cytoxan (cyclophosphamide)

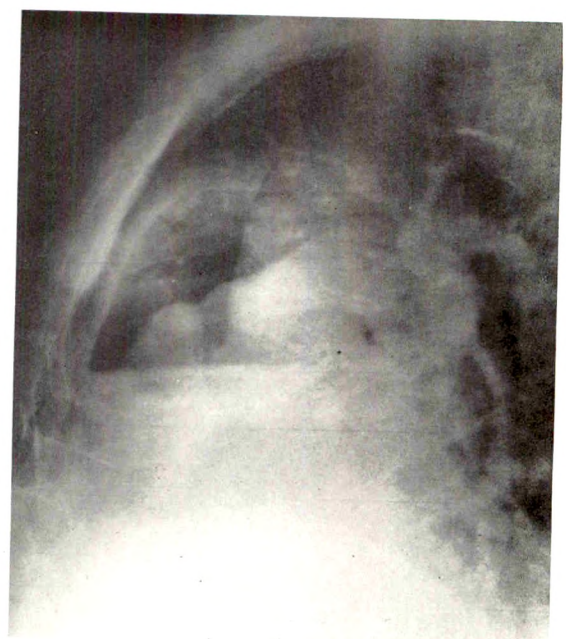


FIG. 2. Case II. Lateral view of the induced pneumopericardium showing a nodular appearance of the anterior superior border of the heart (compare with Fig. 5, *A* and *B*).

with a good initial response. He expired suddenly 3 months later. No autopsy was obtained.

Comment. The nodular appearance of the anterior border of the cardiovascular silhouette within the pneumopericardium was considered to be due to epicardial tumor implantation from the patient's lymphosarcoma. However, from the retrospective experience realized from the next patient, Case III, we now believe that the anterior nodularity most likely represented normal structures. Subsequently, 3 more patients (not included in this report) with pericardial effusion, but without tumor or other abnormality, all showed the same anterior nodularity in air.

Case III. A 49 year old white female was referred by her family physician because of persistent cardiomegaly for approximately 1 year. The patient was completely asymptomatic.

On physical examination, there was no neck vein distention, ascites, or peripheral edema. Both lungs were clear. The cardiac dullness was enlarged to both sides (Fig. 4). Pulses were full and normal. There was no significant cardiac murmur.

The patient was referred to the x-ray department with a presumptive diagnosis of cardiomyopathy. On fluoroscopic examination, however, the diagnosis of pericardial effusion was immediately apparent. The subepicardial fat lines moved vigorously with the myocardium, well within the large cardiac silhouette. The active movement of the fat lines actually formed a striking contrast against the exterior and immobile fluid band.

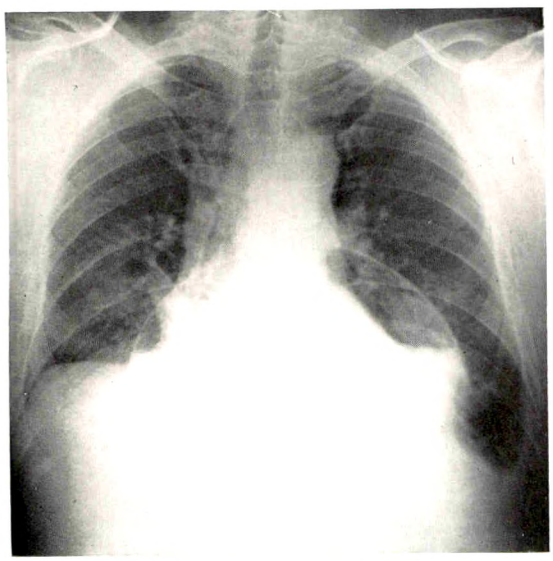


Fig. 3. Case II. Posteroanterior view of the artificial pneumopericardium showing the parietal pericardium to be thin and smooth without adhesions.

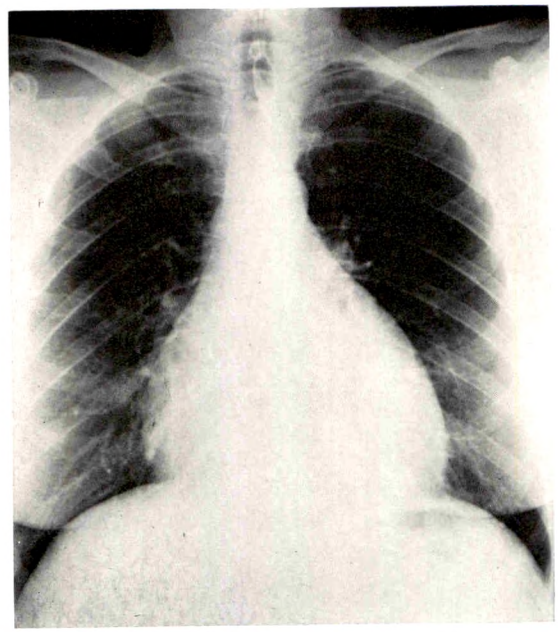


Fig. 4. Case III. Posteroanterior view showing generalized enlargement of the cardiac silhouette.

The patient underwent cardiac catheterization and the hemodynamic data were essentially normal, with no evidence of tamponade. Left ventriculography revealed a normally contracting ventricle. A right atrial injection showed evidence of pericardial fluid but no other abnormality. Right and left coronary arteriograms were normal. However, there was marked interior displacement of the vessels from the border of the cardiac silhouette by the large amount of pericardial fluid.

Approximately 350 ml. of straw colored fluid was removed without difficulty by pericardiocentesis. Smears and culture for both fungus, bacteria, and acid fast organisms were negative. Cytologic examination for tumor was also unrevealing. Following pericardiocentesis, 120 ml. of air was injected into the pericardial sac. Roentgenograms taken in multiple projections showed both the parietal and the visceral layers of the pericardium to be smooth and free of adhesions. The lateral view of the pneumopericardium again showed nodular appearance of the anterior border of the cardiovascular silhou-

ette precisely like that observed in Case II (Fig. 5, *A–C*).

The patient was re-examined under image intensification fluoroscopy, and the movements of the cardiovascular structures were found to be very active. The heart and vessels apparently had considerably more freedom to expand and move in air without the normal confinement of the pericardium. Furthermore, the air fluid level within the pericardial sac was not motionless, but was constantly thrown into waves and splashes primarily by the exaggerated cardiac action and secondarily by respiratory and body movements. The appendage of the left atrium spread out in the air to touch the parietal pericardium in ventricular systole in the posteroanterior and left anterior oblique projections. In the lateral view, the aorta, pulmonary trunk, right ventricle and right atrial appendage were all well delineated by air. The "nodularity" of the anterior border below the ascending aorta actually represented the anterior sinus of the pulmonary artery above and the right atrial appendage below (Fig. 5, *A–C*). These nodular structures were most conspicuous during ventricular systole when they were maximally distended and spread out in the air toward the parietal pericardium.

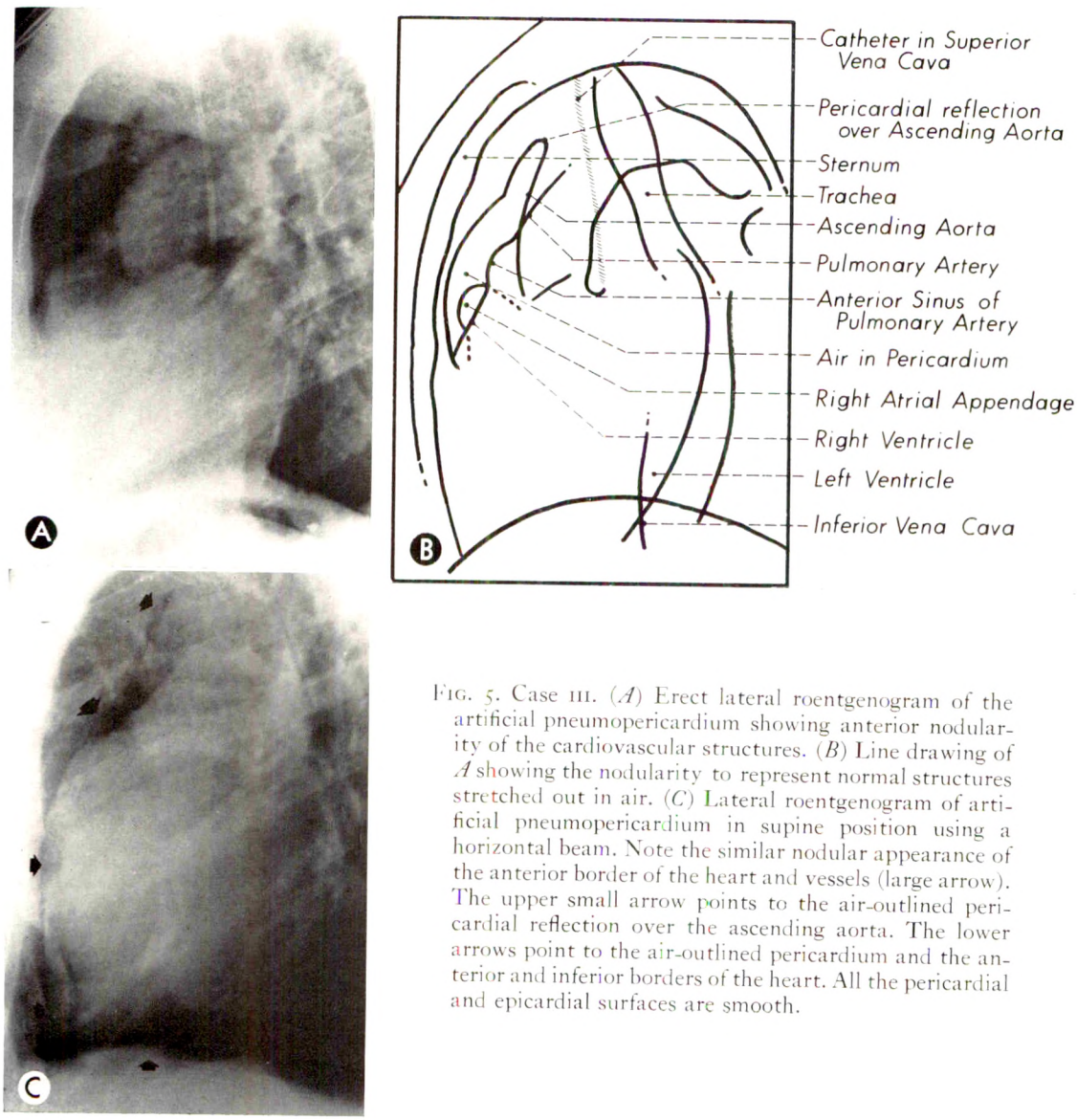


FIG. 5. Case III. (*A*) Erect lateral roentgenogram of the artificial pneumopericardium showing anterior nodularity of the cardiovascular structures. (*B*) Line drawing of *A* showing the nodularity to represent normal structures stretched out in air. (*C*) Lateral roentgenogram of artificial pneumopericardium in supine position using a horizontal beam. Note the similar nodular appearance of the anterior border of the heart and vessels (large arrow). The upper small arrow points to the air-outlined pericardial reflection over the ascending aorta. The lower arrows point to the air-outlined pericardium and the anterior and inferior borders of the heart. All the pericardial and epicardial surfaces are smooth.

This patient was treated with corticosteroids with dramatic response. The cardiac silhouette returned to normal within a 2 week period of time.

Comment. This patient presented several interesting points. First, she was completely asymptomatic with the pericardial effusion manifesting as an enlarged cardiac silhouette and suggesting a primary cardiomyopathy. Secondly, this case has demonstrated the value of fluoroscopy in the diagnosis of pericardial effusion.[5,10] Thirdly, it taught us the importance of recognizing the altered anatomy and dynamics of the cardiovascular structures in pneumohydropericardium.[1,4,6,7,9,11] When one is familiar with this phenomenon, erroneous diagnosis of tumor of the pericardium can be avoided.

Case IV. A 13 year old male was admitted with shortness of breath, peripheral edema and a massive cardiac silhouette.

The diagnosis of pericardial effusion with cardiac tamponade was established by roentgenographic, angiocardiographic, and hemodynamic data. A pericardiocentesis recovered 1,500 ml. of straw colored fluid which was high in cholesterol.

Roentgenograms following purposely induced pneumopericardium were taken in different projections. In the posteroanterior projection, a large, smooth bordered nodular density was seen in the left upper area of the pericardium below the pulmonary artery (Fig. 6). A tumor was suspected. However, during the operation for removal of the pericardium, this nodular density was found to be the appendage of the left atrium! The parietal pericardium was found to be perfectly normal. The epicardium was covered with a thick layer of fibrin. The fibrous peel from the epicardium was removed using the rather abundant subepicardial fat as a guideline in order not to injure the coronary vessels and nerves.

Comment. This case demonstrates the possibility of an erroneous diagnosis of tumor within the pneumopericardium. The rounded density in the superior portion of the pericardium on the left in Figure 6 actually represents a spread-out left atrium appendage.[1,6,7,9]

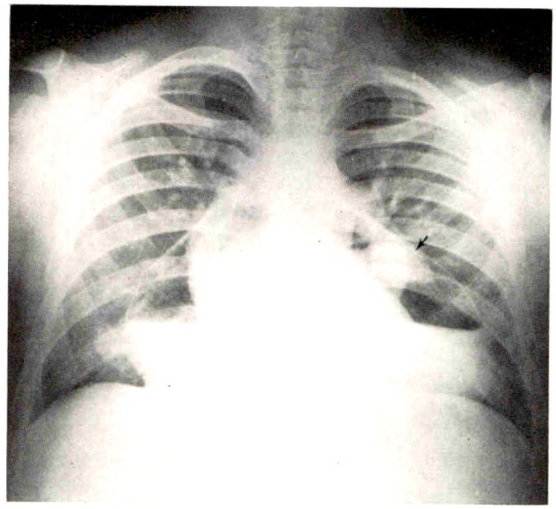


Fig. 6. Case IV. Posteroanterior roentgenogram of the artificial pneumopericardium showing the nodular density along the left cardiac border (arrow) to be the spread-out left atrial appendage.

DISCUSSION

The interrelationships among different cardiac chambers and the great vessels were elaborately described by Dotter and Steinberg using angiocardiography in 1951.[1] The anterior sinus of the pulmonary artery produces a nodular bulge distal and above the outflow tract of the right ventricle.[1,4] The roentgen appearance of the atrial appendages normally varies considerably and the variation is even greater in diseased hearts.[9] It is generally recognized that the right atrial appendage can be superimposed upon the outflow tract of the right ventricle in lateral or left anterior oblique angiocardiograms.[1] This is particularly frequent when the right atrium is enlarged. The anterior expansion of the right atrial appendage is most marked during ventricular systole.

Following induced pneumopericardium, the normal confinement of the heart and vessels by the pericardium has been literally removed. Therefore, one can logically expect the cardiovascular structures to move more freely in the air.[6,7,11] This is easily seen fluoroscopically under an image intensification system. Actually, all normal movements of the different structures are markedly exaggerated. The right atrial appendage extends well into the retrosternal region anterior to the outflow tract of the

right ventricle and inferior to the nodular appearing anterior sinus of the pulmonary artery. Thus, these normal structures form a nodular appearance between the aorta above and the right ventricle below in the lateral view (Fig. 2; and 5, *A–C*).

It is a common experience to see a bulge along the left cardiac border below the pulmonary artery in mitral valve disease. This bulge represents an enlarged left atrial appendage. With a pneumopericardium, a normal sized left atrial appendage can expand far more laterally than when normally confined by the pericardium. Thus, a nodular density is formed in the air by the spread out left atrial appendage (Fig. 6).

With exaggerated movements of the heart and the great vessels within the pneumohydropericardium, the fluid is constantly thrown into waves and splashes seen during fluoroscopy. With a fast exposure, it is possible to record the fluid waves on the film. This was presumed to have happened in Case 1 (Fig. 1*B*).

It is, therefore, important to appreciate the altered anatomy and dynamics of the cardiovascular structures under artificial circumstances.

CONCLUSIONS AND SUMMARY

1. When the heart is surrounded by a blanket of air, it has considerably more freedom to expand and to move. Therefore, both the radiologist and the cardiologist must understand the altered dynamics and anatomy of the heart in order to avoid erroneous diagnosis and unnecessary surgery.

2. Fluoroscopy of the heart under image intensification is useful both for diagnosis of pericardial effusion and for evaluation of the pericardium and the heart following artificial pneumopericardium.

3. Emphasis is placed on the 3 "do nots:" (1) do not call a fluid wave within the pneumohydropericardium a tumor; (2) do not call the anterior nodularity of the heart in air implantation on the epicardium; and (3) do not call the nodular appearance of the left atrial appendage in pneumopericardium a tumor.

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REFERENCES

1. DOTTER, C. T., and STEINBERG, I. Angiocardiography. Paul B. Hoeber, Inc., New York, 1951, pp. 55–73.
2. ELLMAN, P. Tuberculous pericarditis with effusion. *Brit. Heart J.*, 1945, *7*, 147–160.
3. GOODKIND, M. J., BLOOMER, W. E., and GOODYER, A. V. N. Recurrent pericardial effusion after non-penetrating chest trauma. *New England J. Med.*, 1960, *263*, 874–881.
4. GRAY, H., and GOSS, C. M. Anatomy of the Human Body. Twenty-seventh edition. Lea & Febiger, Philadelphia, 1959, pp. 583.
5. JORGENS, J., KUNDEL, R., and LIEBER, A. Cinefluorographic approach to diagnosis of pericardial effusion. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1962, *87*, 911–916.
6. SCHINZ, H. R., BAENSCH, W. E., FROMMHOLD, W., GLAUNER, R., UEHLINGER, E., and WELLAUER, J. The Heart and Great Vessels. In: Roentgen Diagnostics. Volume IV, Part I. Second American edition. Edited by L. G. Rigler. Grune & Stratton, Inc., New York, 1970, pp. 478.
7. SCHWEDEL, J. B. Clinical Roentgenography of the Heart. Paul B. Hoeber, Inc., New York, 1946, pp. 311.
8. SPODICK, D. H. Chronic and Constrictive Pericarditis. Grune & Stratton, Inc., New York, 1964, pp. 294–298.
9. SWANSON, G. E., and STEINBERG, I. Roentgen features of atrial appendages. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1964, *91*, 311–320.
10. TORRANCE, D. J. Demonstration of subepicardial fat as aid in diagnosis of pericardial effusion or thickening. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1955, *74*, 850–855.
11. YU, P. N. G., LOVEJOY, F. W., JOOS, H. A., NYE, R. E., and SIMPSON, J. H. Unusual case of massive pericardial effusion with hemodynamic studies. *Ann. Int. Med.*, 1953, *39*, 928–936.

A METHOD FOR THE ESTIMATION OF PULMONARY VENOUS AND ARTERIAL PRESSURES FROM THE ROUTINE CHEST ROENTGENOGRAM*

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THE need has long been recognized for a safe, simple, noninvasive, and reasonably accurate method for evaluating the presence and degree of heart failure. If the *sine qua non* of left heart failure is elevation of the pulmonary venous pressure, then a reasonably accurate method of assessing pulmonary venous pressure would provide a means of determining the degree of left heart failure.

We have developed a two-component concept which we feel allows a reasonably accurate assessment of the pulmonary venous pressure, as well as providing an estimate of the total pulmonary artery pressure from the interpretation of the routine chest roentgenogram.

The changes in the pulmonary vasculature as seen on the routine chest roentgenograms in failure have been well described.[3,4,6,9,10,17,18,20,23,25,26,28,29,31] Many investigators have made attempts to estimate the pulmonary vascular pressures.[2,14,19,24,27]

Our concept evolved out of a trial-and-error evaluation of roentgenograms taken immediately before and after catheterization, correlated with pulmonary angiograms and the catheterization data on more than 1,000 patients.

METHOD

The right ventricle is the power generator for the pulmonary circuit. One can conceive of the pulmonary circulation as a two-component system. The first component is energy utilized in maintaining forward flow across the capillary bed to ensure oxygenation of the blood. The second component is energy required to fill the left ventricle. The first component is the pre-capillary component, while the second component is the pulmonary venous pressure and is called the post-capillary component. Since the pulmonary vascular bed has virtually no resistance, and energy requirements are minimal for left ventricular filling, the pulmonary circuit is a low-pressure system. An increase in resistance across the pulmonary vascular bed and/or an increase in filling pressures of the left ventricle would increase the energy demands of the right ventricle.

For the purpose of analysis, the two components are considered separately. The *post-capillary component* is derived from the evaluation of the regional distribution of pulmonary blood flow, while the *pre-capillary component* is assessed by changes in the main pulmonary artery and its major branches.

POST-CAPILLARY COMPONENT

In the normal, full-inspiration, upright chest roentgenogram, flow is directed to the lower lung zones by gravity.[5,22,30,33] The remarkable sensitivity of the human lung to gravity is illustrated by the changes in perfusion related to body position. There is equal perfusion of the upper and lower zones in the supine position, and increased perfusion of the upper zones in the inverted position.[5,30] Thus, the size and number of vessels per unit area in the lower lung zone is greater than the size and number of ves-

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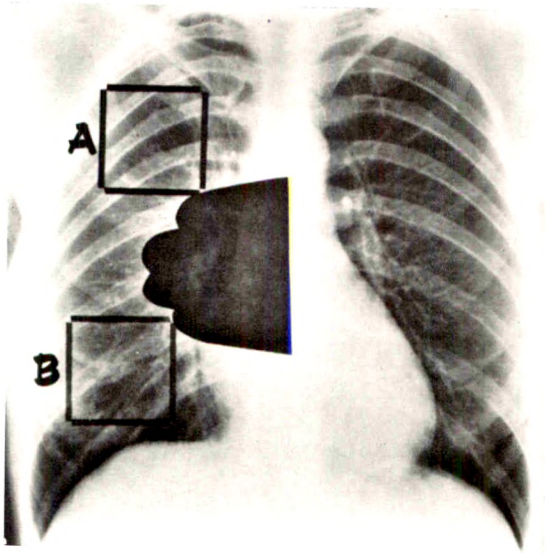


FIG. 1. Chest roentgenogram with hilar area covered and areas A and B marked to represent comparative regions in upper and lower zones. Areas A and B are compared for the size and number of blood vessels to assess regional blood flow. Area B has more vessels than area A; therefore, blood flow to area B is assessed as greater than area A.

sels visualized per unit area of lung in the upper zones in the upright position.[32] In Figure 1, the hilar vessels are covered, since they may show enlargement in response to elevated pulmonary *arterial* pressure. Two areas, A and B, are compared visually. We see that the size and number of vessels are greater in area B than area A, reflecting greater blood flow. The distribution criteria for the post-capillary component have been divided into 5 categories as follows: normal, 1+, 2+, 3+, and 4+ redistribution changes.

Normal pulmonary vessels show an orderly tapering and branching toward the periphery, where they become invisible in the subpleural regions of the lungs (Fig. 2, *A–C*).

1+ Redistribution is present when there is equal perfusion of the upper and lower lung zones. This increased perfusion in the upper zones is due to a shifting of blood from the lower zones. There is a true oligemia in the lower zones as a result of this shift (Fig. 3, *A–C*). This uniform appearance should not be confused with the increased total pulmonary blood flow, where there is also an increased perfusion of the upper zones. This upper zone increase is in addition to increase in perfusion in the lower zones and not a diversion of blood.

2+ Redistribution is present when there is greater perfusion in the upper than the lower lung zones. This represents an inversion of the normal pulmonary blood flow pattern (Fig. 4, *A–C*).

3+ Redistribution is present when 2+ redistribution is accompanied by interstitial edema. This occurs when the hydrostatic pressure in the venous system exceeds the oncotic pressure of the proteins, and, therefore, fluid leaks through the capillaries. On the roentgenogram, interstitial edema may be present as perivascular haziness, hilar indistinction, septal lines, well-defined fine nodules, or a mosaic pattern at the bases[8] (Fig. 5, *A–C*).

4+ Redistribution is represented by frank alveolar edema (Fig. 6).

PRE-CAPILLARY COMPONENT

The pre-capillary component is derived from the sum of two factors. The first factor is the normal kinetic energy required to set a static column of blood into motion. The second factor is excessive kinetic energy required to overcome any resistance in the pulmonary circuit. The excessive kinetic energy is assessed by changes in the main pulmonary artery and its major branches. The main pulmonary artery is graded as normal, 1+, 2+, or 3+ enlarged, and its major branches are assessed as normal, proximal dilatation, or distal attenuation. It has been noted that when there is marked distal attenuation, the pulmonary arterial pressure is usually above 75 mm. Hg. This uneven response is explained by thin or negligible musculature in the main pulmonary artery and its major branches.[26,28,31] An example of our grading system is found in Figure 7.

ASSIGNMENT OF NUMERICAL VALUES

Tables I (Fig. 8) and II were constructed

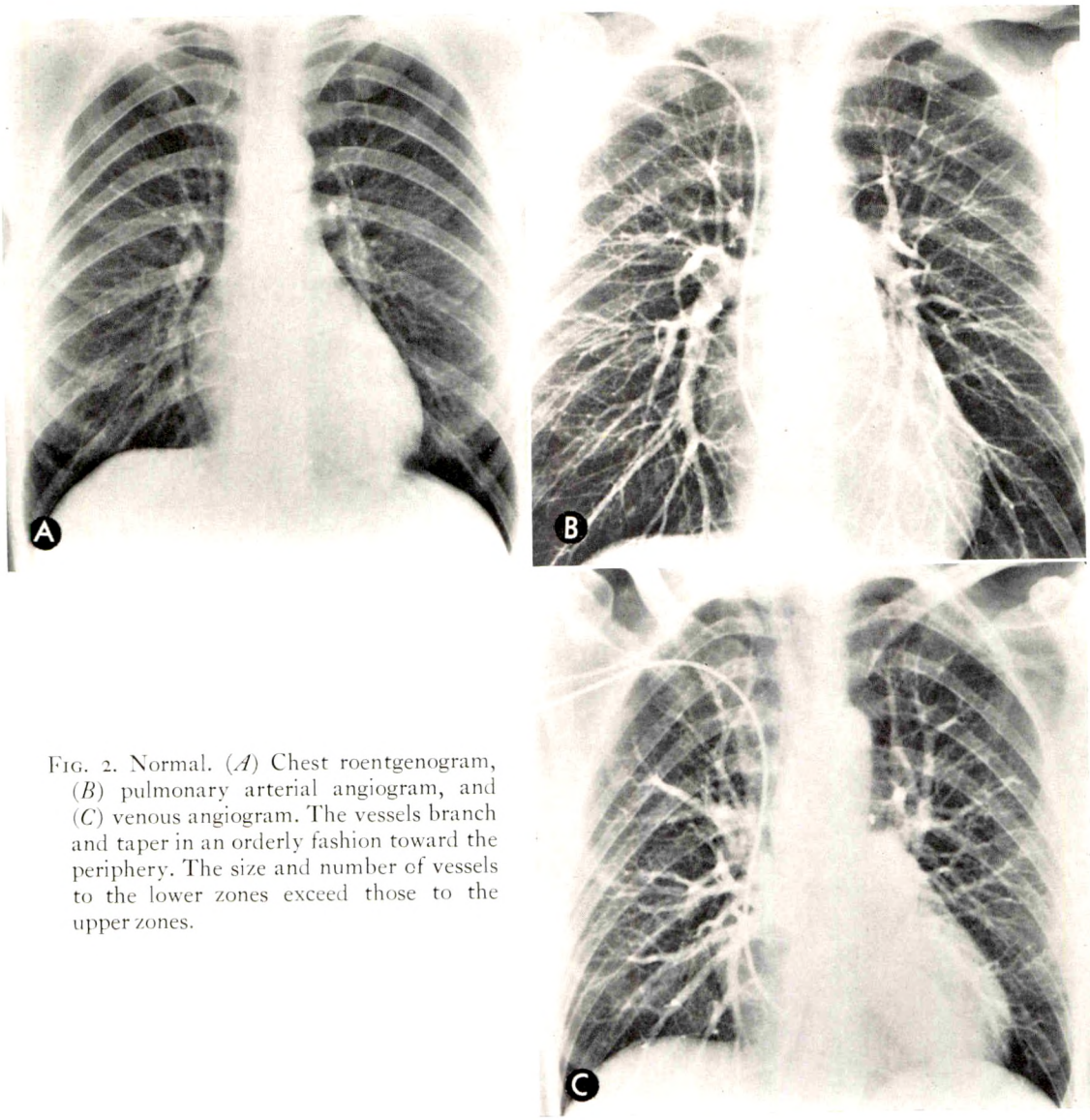


FIG. 2. Normal. (*A*) Chest roentgenogram, (*B*) pulmonary arterial angiogram, and (*C*) venous angiogram. The vessels branch and taper in an orderly fashion toward the periphery. The size and number of vessels to the lower zones exceed those to the upper zones.

to give values to each factor used in assessing the post-capillary and pre-capillary components. These represent the units assigned each factor after many trial-and-error assessments.

By addition of the pre-capillary and post-capillary components, one can estimate the total pulmonary artery pressure. For example, Figure 9 is a routine chest roentgenogram of a patient with known heart disease. With a step-by-step analysis, one can arrive at an estimation of the pulmonary venous and arterial pressures.

Step 1 is the comparative assessment of the size and number of vessels in the upper and lower lungs. There are more vessels in the upper than the lower lungs, and there is no interstitial edema. This is 2+ redistribution. Pressure estimation can be obtained from Table 1. The mean left atrial pressure would be estimated between 15 and 25 mm. Hg.

Step 2 represents analysis of the main pulmonary artery and its major branches. The main pulmonary artery is 3+ enlarged, and its major branches are dilated

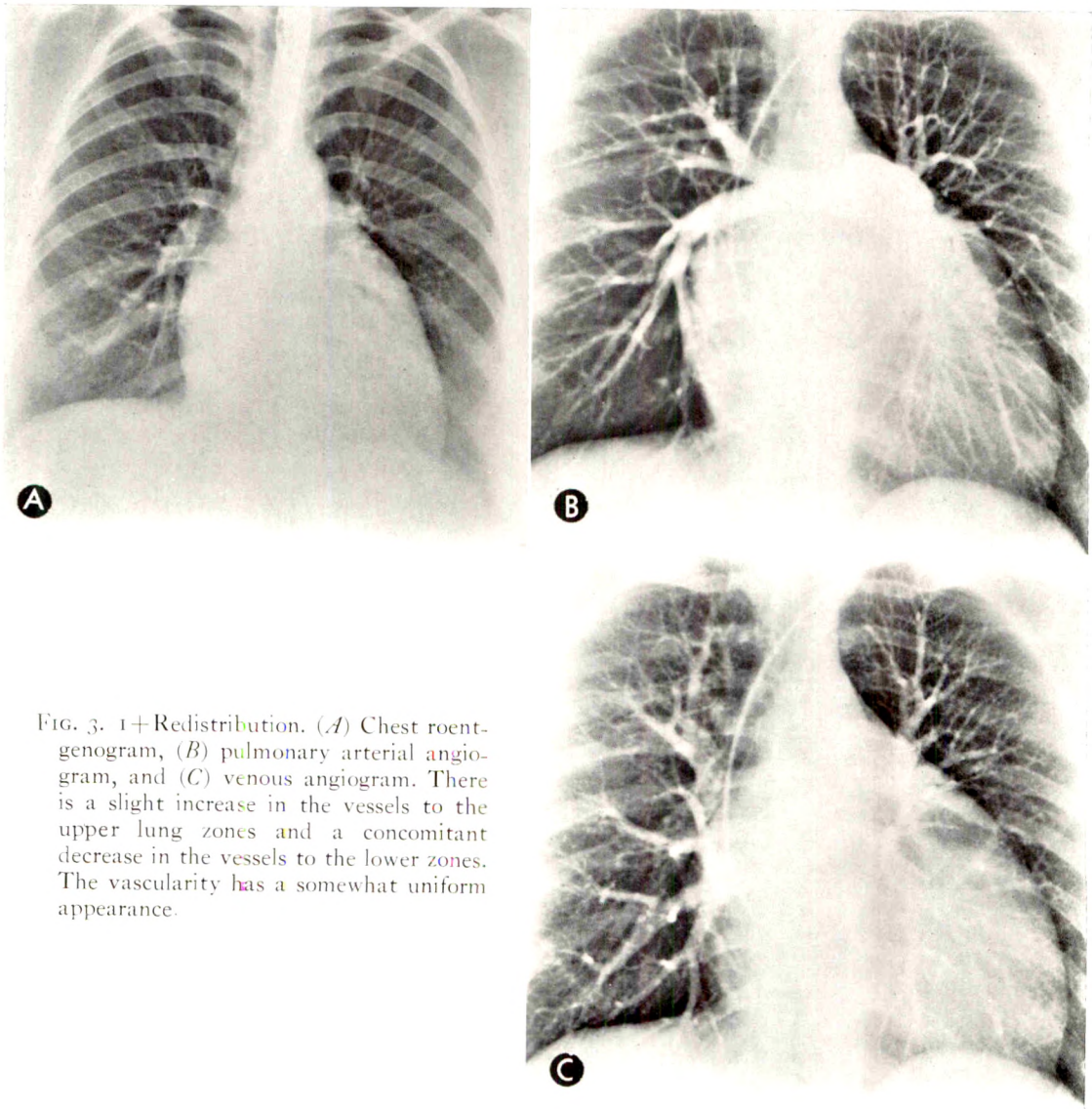


FIG. 3. 1+Redistribution. (*A*) Chest roentgenogram, (*B*) pulmonary arterial angiogram, and (*C*) venous angiogram. There is a slight increase in the vessels to the upper lung zones and a concomitant decrease in the vessels to the lower zones. The vascularity has a somewhat uniform appearance.

TABLE I

POST-CAPILLARY ASSIGNED VALUES*

Redistribution Pattern	Mean Left Atrial Pressure
Normal	5–10 mm. Hg
1+	10–15 mm. Hg
2+	15–25 mm. Hg
3+	25–35 mm. Hg
4+	35+ mm. Hg

* A graphic representation of this information may be found in Figure 8.

TABLE II

PRE-CAPILLARY ASSIGNED VALUES

Normal Kinetic Energy	15 mm. Hg
Excessive Kinetic Energy Added for Enlargement of the Main Pulmonary Artery:	
1+ enlargement	10 mm. Hg
2+ enlargement	20 mm. Hg
3+ enlargement	30 mm. Hg
Excessive Kinetic Energy Added for Changes in the Major Pulmonary Artery Branches:	
Proximal dilatation	5 mm. Hg
Distal attenuation	15 mm. Hg

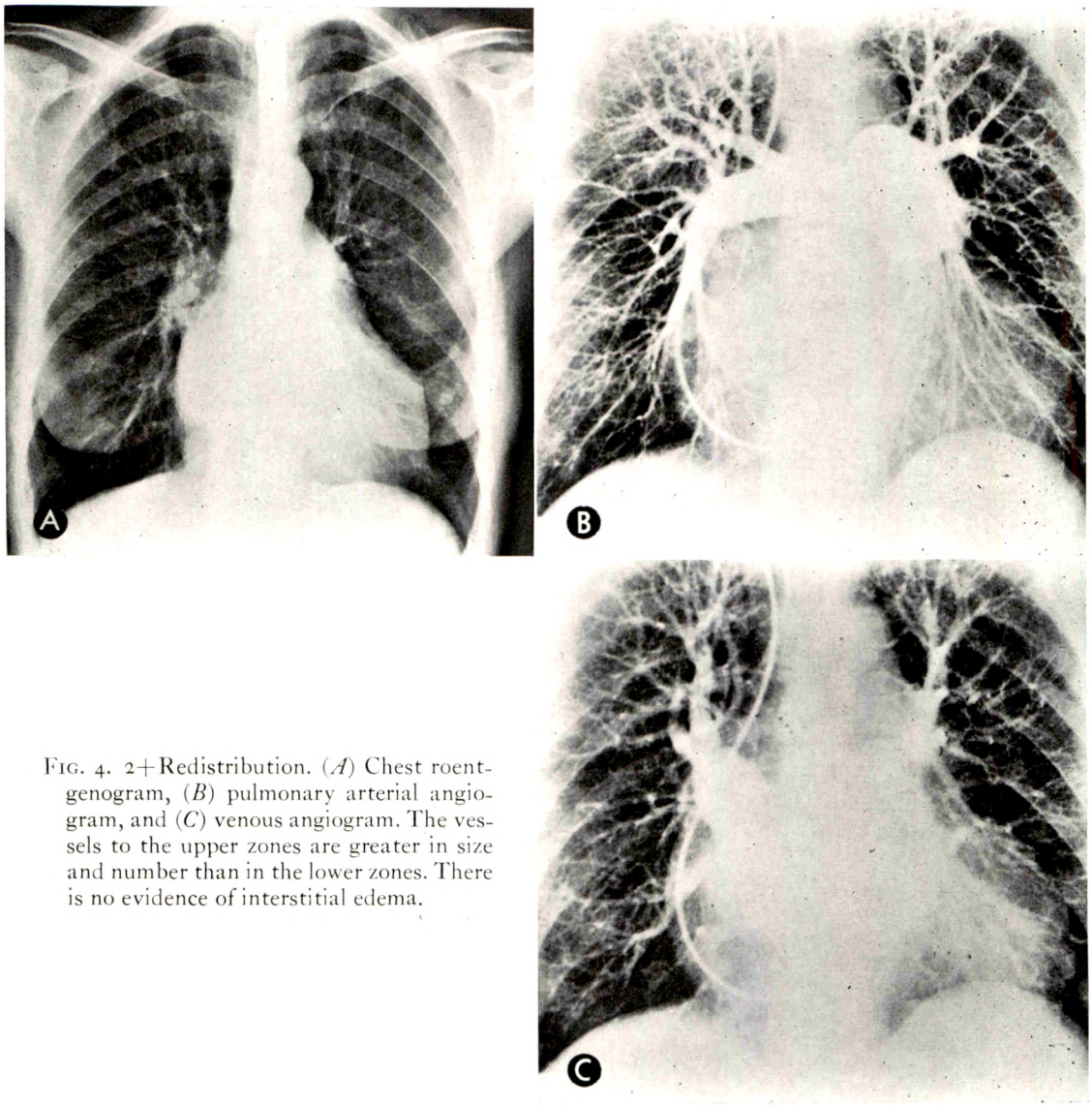


Fig. 4. 2+Redistribution. (*A*) Chest roentgenogram, (*B*) pulmonary arterial angiogram, and (*C*) venous angiogram. The vessels to the upper zones are greater in size and number than in the lower zones. There is no evidence of interstitial edema.

proximally and attenuated distally. From Table II, we would assign 15 mm. Hg for normal kinetic energy. The normal kinetic energy is always considered the same for purposes of estimation. We would further assign an additional 30 mm. Hg for the 3+ enlargement of the main pulmonary artery, 5 mm. Hg for the proximal dilatation of the major branches, and 15 mm. Hg for the distal attenuation of the major branches. The pre-capillary component would, therefore, be $15+30+5+15=65$ mm. Hg. The total pulmonary artery pressure would then be the sum of the pre- and post-capillary components, or $15-25$ mm. Hg $+$ 65 mm. Hg $=80-90$ mm. Hg. The pulmonary arterial pressure measured at catheterization was 100/45, with a mean pulmonary venous pressure of 20 mm. Hg. Although the distal attenuation of the major pulmonary branches would have told us that the pressure was over 75 mm. Hg, using the

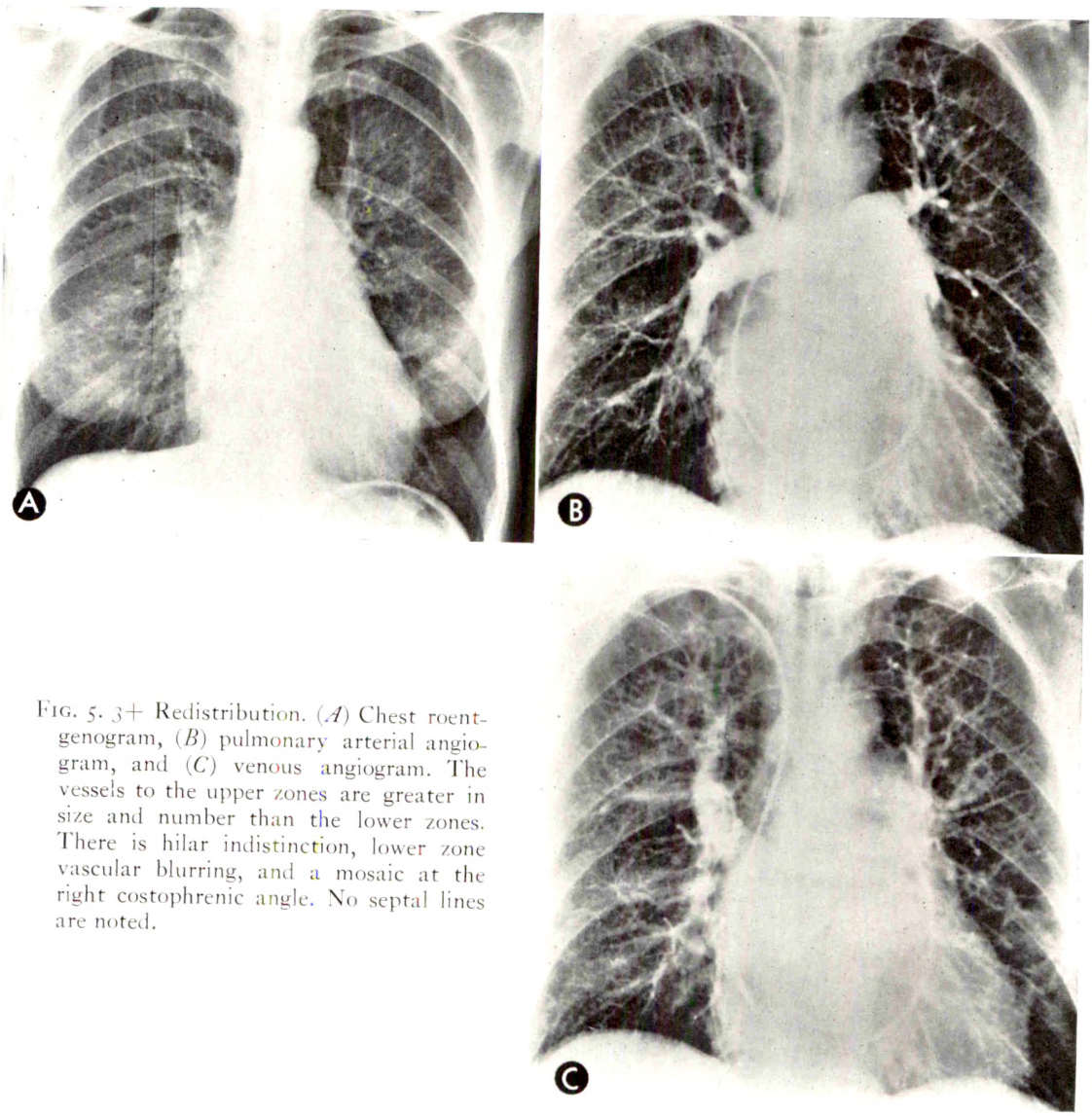


FIG. 5. 3+ Redistribution. (*A*) Chest roentgenogram, (*B*) pulmonary arterial angiogram, and (*C*) venous angiogram. The vessels to the upper zones are greater in size and number than the lower zones. There is hilar indistinction, lower zone vascular blurring, and a mosaic at the right costophrenic angle. No septal lines are noted.

step-by-step analysis, we have come closer to the actual pressure and have also obtained the pulmonary venous pressure.

DISCUSSION

A normal pulmonary artery pressure of 22/7 mm. Hg can be divided into a pre-capillary and a post-capillary component. The post-capillary component would be 7 mm. Hg, since the pulmonary arterial diastolic pressure, mean left atrial pressure, and left ventricular end-diastolic pressures are virtually the same.[16] The post-capillary component represents the pulmonary venous pressure. It has been shown experimentally that as the pulmonary venous pressure rises, there is redistribution of blood flow within the lungs due to perivascular edema.[34] Friedman and Braunwald[5] have shown by radioisotope studies that the magnitude of the shift of flow from the lower to the upper zones has a linear relationship to the mean left atrial pressure. We have classified the magnitude of shift from lower to upper zones utilizing the routine chest roentgenogram. A 1+ through

4+ redistribution has been assigned numerical values which indicate the degree of pulmonary venous hypertension.

The pre-capillary component is the total pulmonary artery pressure minus the post-capillary component. With a normal pressure of 22/7 mm. Hg, that would be 15 mm. Hg. This 15 mm. Hg is interpreted as repre-

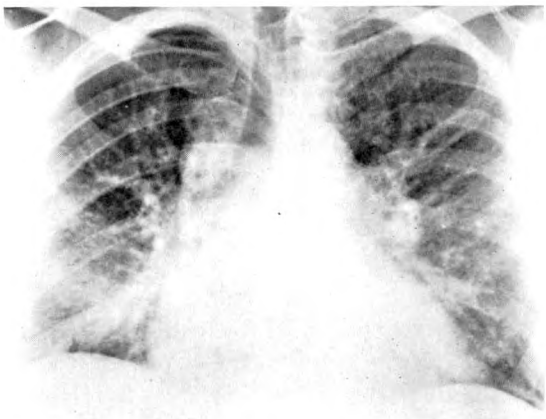


Fig. 6. 4+ Redistribution. Chest roentgenogram demonstrates interstitial and alveolar edema. Air bronchograms confirm the alveolar component. The vascularity shows a shift to the upper zones.

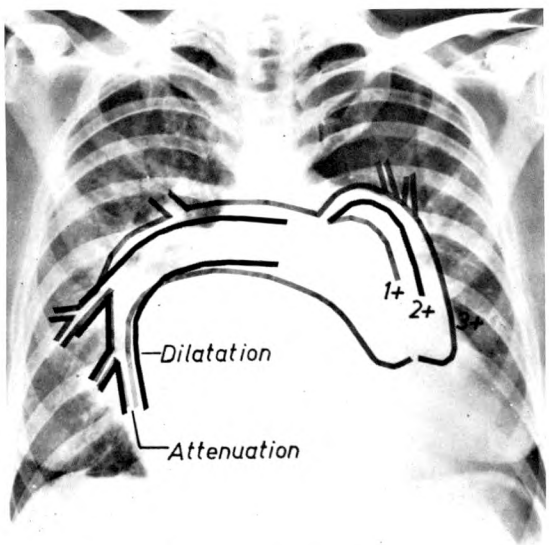


Fig. 7. Chest roentgenogram with overlay to illustrate the arbitrary grading of the main pulmonary artery: 1+ gray, 2+ black, and 3+ gray. Dilatation of the right branch is shown in black and attenuation in gray.

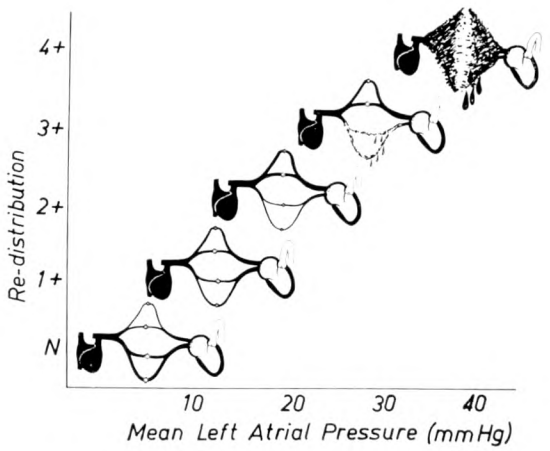


Fig. 8. Graphic representation of Table 1 showing that the magnitude of shift has a linear relationship to the mean left atrial pressure.

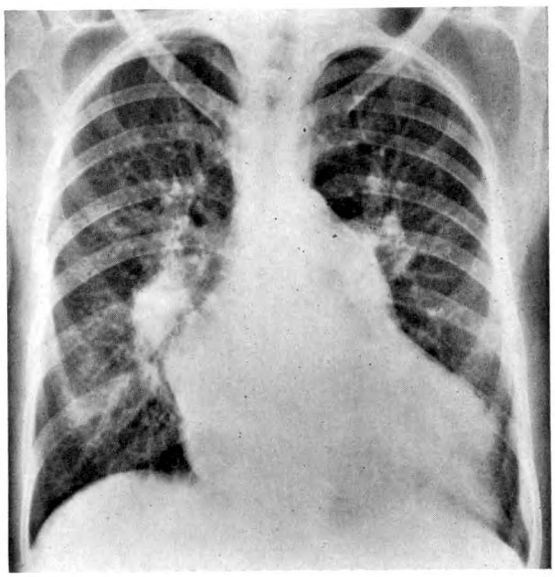


Fig. 9. Routine chest roentgenogram of a patient with known heart disease for estimation of pulmonary venous and arterial pressures.

senting kinetic energy required to set a static column of blood into motion through a pulmonary vascular bed that has no resistance. When a resistance develops across the pulmonary circuit, a gradient appears between the pulmonary arterial diastolic pressure and the pulmonary wedge pressure. An increase in the pre-capillary component is necessary to maintain the for-

ward flow of blood, thus the pre-capillary component must rise to whatever level is necessary to overcome any resistance in the pulmonary vascular bed.

Sources of error are related to factors that alter the pulmonary vascular resistance. Pulmonary disease may impose structural changes on the pulmonary vascular bed or expose it to stimuli which produce resistance to flow, and as a consequence, distribution of flow within the lungs may be altered. When moderate interstitial disease is present, vascular structures are not clearly identified, and may be invisible or extremely blurred, and an assessment of regional blood flow may not be possible.

Vascular tone and reactivity are present in both arteries and veins, and constriction of either can markedly affect the longitudinal distribution of vascular resistance of the lung.[1] Capillary blood flow can be altered by alveolar pressure, provided the alveolar pressure exceeds the pulmonary venous pressure. As alveolar pressure increases sufficiently, blood flow through the capillaries virtually ceases.[21] Increased vascular resistance can develop by compression of the capillaries as a result of interstitial fluid in the pericapillary spaces. Fluid in the pericapillary spaces is one of the mechanisms responsible for the redistribution of the pulmonary blood flow which accompanies elevation of the pulmonary venous pressure.[34]

Alveolar oxygen tension and hydrogen ion concentration appear to be interacting stimuli which are determinants of pulmonary arterial diastolic pressure, and also a major factor in the distribution of blood flow within the lungs. The level of either augments or attenuates the other, with all available knowledge indicating that the site of action is at the level of the most muscular pulmonary vessels, which have a diameter of 100–400 micra.[11] Hypoxia and hypercapnia are probably the major factors in the random redistribution of pulmonary blood flow in pulmonary emphysema.[15] The duration of venous hypertension changes the amount of pressure required to cause alveolar edema. Chronic venous hypertension encourages the formation of fibrin, which inhibits the passage of fluid into the intra-alveolar space.[12,13] In chronic failure, high pulmonary venous pressures may be present without the presence of alveolar edema.

Often overlooked are the length of diastolic filling period (heart rate) and the cardiac output (flow rate). To illustrate these dynamic effects, we have used the well-known Gorlin[7] formula for mitral valve area in Table III. In our calculations, a mitral valve area of 1 cm.^{2}, representing moderate mitral stenosis, was used for convenience.

$$LA_m = \left(\frac{MVF}{31 \times MVA}\right)^2 - LV_{dm},$$

where LA_m = mean left atrial pressure in mm. Hg; MVF = mitral valve flow in ml./sec.; MVA = mitral valve area in cm.^{2}; and LV_{dm} = left ventricular mean diastolic pressure in mm. Hg. In line 2 of Table III, one can see that doubling the heart rate with a fixed output requires a doubling of mean left atrial pressure, while line 3 of Table III shows that a 60 per cent increase

TABLE III

CALCULATION OF MITRAL VALVE AREA

Mitral Valve Area (cm.^{2})	Heart Rate (beats/min.)	Diastolic Time (sec./min.)	Cardiac Output (ml./min.)	Mean Left Atrial Pressure (mm. Hg)
1.0	60	40	5,000	11 mm. Hg
1.0	120	30	5,000	21 mm. Hg
1.0	60	40	8,000	34 mm. Hg

in cardiac output at a fixed heart rate requires a three-fold increase in mean left atrial pressure.

Changes in the heart rate and cardiac output associated with exercise or uncontrolled arrhythmias can cause significant alterations in the pulmonary venous and pulmonary arterial pressures. This has been demonstrated in our laboratory with the taking of pre- and post-catheterization chest roentgenograms. Changes from 1+ redistribution to 3+ redistribution have been noted on the chest roentgenograms, and these have correlated well with elevations of the end-diastolic pressure that have accompanied the use of contrast media.

The roentgenogram of the chest may be used to detect early left heart failure, and allows one to follow cardiac patients and assess their response to therapy. Relating pressure estimates that approximate catheterization values with specific chamber enlargement adds a greater impact to the interpretation of the chest roentgenogram and cardiac study.

SUMMARY

A simplified method for estimating pulmonary venous and arterial pressures utilizing a step-by-step approach to the assessment of post-capillary and pre-capillary components is presented.

The correlation with cardiac catheterization values is remarkably good.

Pulmonary disease and increased total pulmonary blood flow are the major sources of inaccurate estimations.

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REFERENCES

1. Brody, J. S., Stemmler, E. J., and DuBois, A. B. Longitudinal distribution of vascular resistance in pulmonary arteries, capillaries and veins. *J. Clin. Invest.*, 1968, *47*, 783–808.
2. Davies, L. G., Goodwin, J. F., Steiner, R. E., and Van Leuven, B. D. Clinical and radiological assessment of pulmonary arterial pressure in mitral stenosis. *Brit. Heart J.*, 1953, *15*, 393–400.
3. Doyle, A. E., Goodwin, J. F., Harrison, C. V., and Steiner, R. E. Pulmonary vascular patterns in pulmonary hypertension. *Brit. Heart J.*, 1957, *19*, 353–365.
4. Fleischner, F. G., and Reiner, L. Linear x-ray shadows in acquired pulmonary hemosiderosis and congestion. *New England J. Med.*, 1954, *250*, 900–905.
5. Friedman, W. F., and Braunwald, E. Alterations in regional blood flow in mitral valve disease studied by radioisotope scanning. *Circulation*, 1966, *34*, 363–376.
6. Goodwin, J. F., Steiner, R. E., and Lowe, K. G. Pulmonary arteries in mitral stenosis demonstrated by angiocardiography. *J. Fac. Radiologists*, 1952, *4*, 21–27.
7. Gorlin, R., and Gorlin S. G. Hydraulic formula for calculation of area of stenotic mitral valve, other cardiac valves and ventral circulatory shunts. *Am. Heart J.*, 1951, *31*, 1–29.
8. Gough, J. Correlation of Radiological and Pathological Changes in Some Diseases of Lung. Harvey Lecture Series 1957–1958. Academic Press, Inc., New York, pp. 171–185.
9. Grainger, R. G. Interstitial pulmonary oedema and its radiological diagnosis: sign of pulmonary venous and capillary hypertension. *Brit. J. Radiol.*, 1958, *31*, 201–217.
10. Harley, H. R. Radiological changes in pulmonary venous hypertension, with special reference to root shadows and lobular pattern. *Brit. Heart J.*, 1961, *23*, 75–87.
11. Harvey, R. M., and Enson, Y. Pulmonary vascular resistance. *Advan. Int. Med.*, 1969, *15*, 73–93.
12. Hayward, G. W. Pulmonary oedema. *Brit. M. J.*, 1955, *1*, 1361–1367.
13. Heard, B. E., Steiner, R. E., Herdan, A., and Gleason, D. Oedema and fibrosis of lungs in left ventricular failure. *Brit. J. Radiol.*, 1968, *41*, 161–171.
14. Jacobson, G., Schwartz, L. H., and Sussman, M. L. Radiographic estimation of pulmonary artery pressure in mitral valvular disease. *Radiology*, 1957, *68*, 15–24.
15. Jacobson, G., Turner, A. F., Balchum, O. J., and Jung, R. Vascular changes in pulmonary emphysema. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *100*, 374–396.
16. Kaltman, A. J., Herbert, W. H., Conroy, R. J., and Kossman, C. E. Gradient in pressure across pulmonary vascular bed during diastole. *Circulation*, 1966, *34*, 377–384.
17. Lavender, J. P., Doppman, J., Shawdon, H.,

and STEINER, R. E. Pulmonary veins in left ventricular failure and mitral stenosis. *Brit. J. Radiol.*, 1962, *35*, 293–302.

18. LOGUE, R. B., ROGERS, J. V., and GAY, B. B. Subtle roentgenographic signs of left heart failure. *Am. Heart J.*, 1963, *65*, 464–473.
19. MILNE, E. N. Physiological interpretation of plain radiograph in mitral stenosis, including review of criteria for radiological estimation of pulmonary arterial and venous pressures. *Brit. J. Radiol.*, 1963, *36*, 902–913.
20. ORMOND, R. S., and POZNANSKI, K. Pulmonary veins in rheumatic heart disease. *Radiology*, 1960, *74*, 542–549.
21. PERMUTT, S., HOWELL, J. B., PROCTOR, D. F., and RILEY, R. L. Effect of lung inflation on static pressure-volume characteristics of pulmonary vessels. *J. Appl. Physiol.*, 1961, *16*, 64–70.
22. RODBARD, S. Distribution of flow through pulmonary manifold. *Am. Heart J.*, 1956, *51*, 106–125.
23. ROSENOW, E. C., III, and HARRISON, C. E., JR. Congestive heart failure masquerading as primary pulmonary disease. *Chest*, 1970, *58*, 28–36.
24. SIMON, M. Pulmonary veins in mitral stenosis. *J. Fac. Radiologists*, 1958, *9*, 25–32.
25. SIMON, M. Pulmonary vessels in incipient left ventricular decompensation. *Circulation*, 1961, *24*, 185–190.
26. SIMON, M. Pulmonary vessels: their hemodynamic evaluation using routine radiographs. *Radiol. Clin. North America*, 1963, *1*, 363–376.
27. SIMON, M., SASAHARA, A. A., and CANNILLA, J. E. Radiology of pulmonary hypertension. *Seminars Roentgenol.*, 1967, *2*, 368–388.
28. STEINER, R. E. Radiological appearances of pulmonary vessels in pulmonary hypertension. *Brit. J. Radiol.*, 1958, *31*, 188–200.
29. STEINER, R. E. Radiology of pulmonary circulation: Chamberlain Lecture. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1964, *91*, 249–264.
30. TOW, D. E., WAGNER, H. N., JR., LOPEZ-MAJANO, V., SMITH, E. M., and MIGITA, T. Validity of measuring regional pulmonary arterial blood flow with macroaggregates of human serum albumin. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *96*, 664–676.
31. VAN EPPS, E. F. Roentgen manifestations of pulmonary hypertension. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1958, *79*, 241–250.
32. WEHRMACHER, W. H. Estimation of cardiovascular dynamics from routine radiograph of chest. *Current Med. Dig.*, 1969, 212–217.
33. WEST, J. B. Distribution of gas and blood in normal lungs. *Brit. M. Bull.*, 1963, *19*, 53–58.
34. WEST, J. B., DOLLERY, C. T., and HEARD, B. E. Increased pulmonary resistance in dependent zone of isolated dog lung caused by perivascular edema. *Circulation Res.*, 1965, *17*, 191–206.

Vol. 116, No. 1

THE AORTIC-PULMONARY MEDIASTINAL STRIPE*

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FOR A number of years, I have been intrigued by an infrequently seen shadow of increased density coursing across the main pulmonary artery and aortic knob in chest roentgenograms of normal individuals. This shadow has apparently not been described in the radiologic literature and I have only recently been able to identify its nature.

This report is concerned with a descrip-

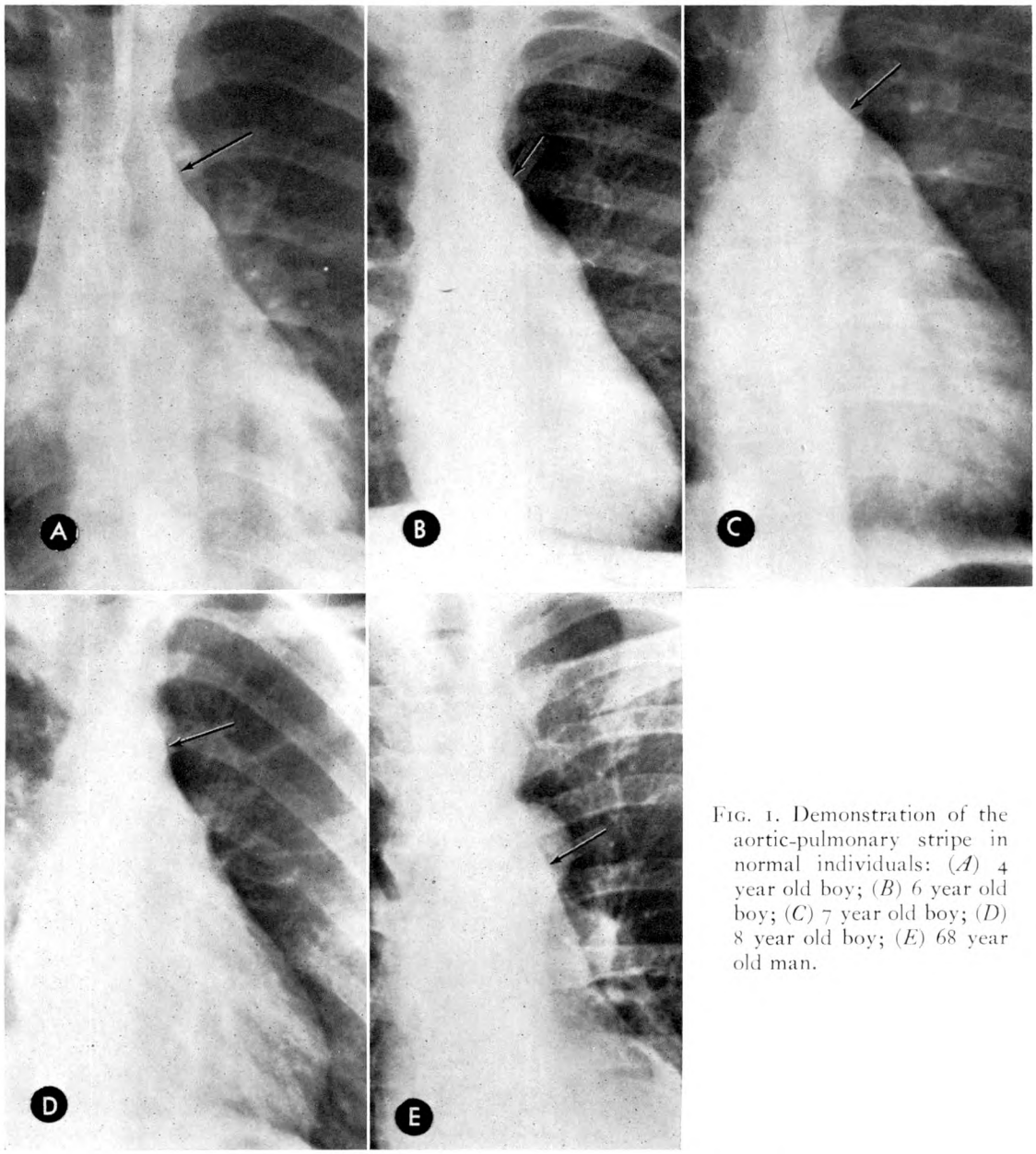


FIG. 1. Demonstration of the aortic-pulmonary stripe in normal individuals: (*A*) 4 year old boy; (*B*) 6 year old boy; (*C*) 7 year old boy; (*D*) 8 year old boy; (*E*) 68 year old man.

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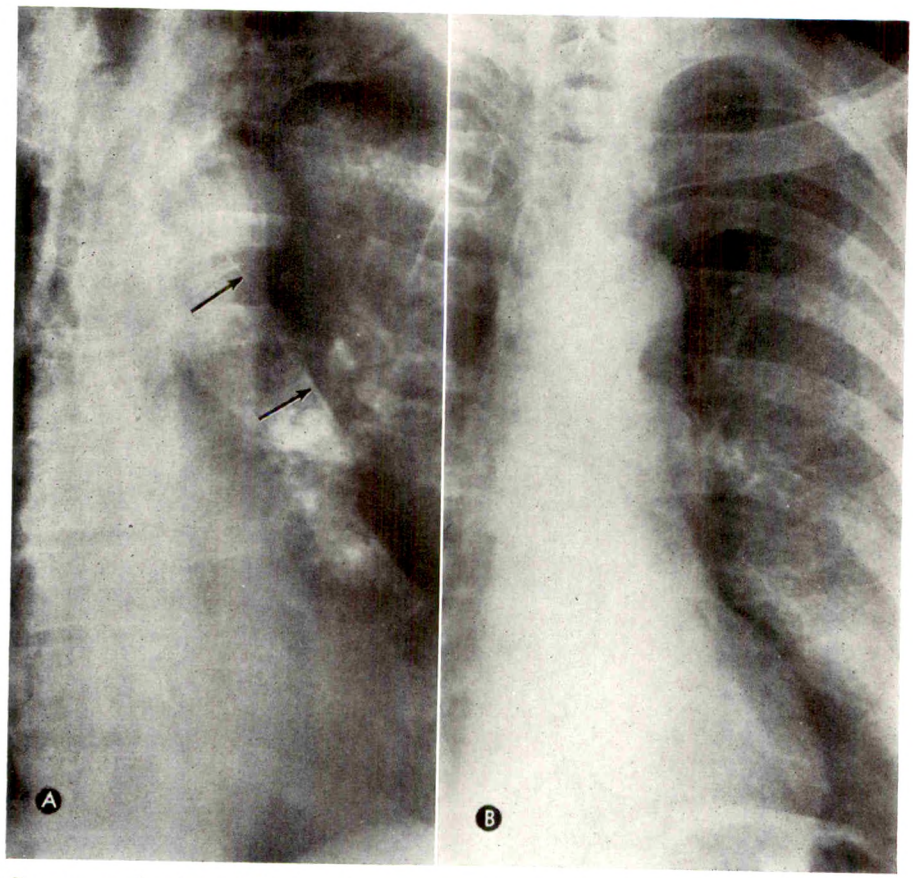


FIG. 2. (*A*) Pneumomediastinum following chest trauma. The aortic-pulmonary pleural reflection is indicated by the upper arrow. (*B*) Following resolution of the pneumomediastinum, the aortic-pulmonary stripe is no longer seen.

tion of this entity and its anatomic origin. The intent is not to add still another mediastinal stripe to the literature, but rather to call attention to an anatomic variation which may be of concern if its nature is not appreciated.

ROENTGEN FINDINGS

The shadow of interest, which I have termed the aortic-pulmonary stripe, is evident in the frontal view of the mediastinum as a triangular shadow with an oblique, lateral edge that extends across the outline of the aortic knob superiorly and the pulmonary artery inferiorly. Its size and prominence are somewhat variable and it may be seen in children, as well as adults (Fig. 1, *A–E*).

It appeared to be a logical assumption that the aortic-pulmonary stripe represented the reflection of the mediastinal pleura across the pulmonary artery and aortic arch, but I was unable to document this assumption until a recent experience with 2 patients with pneumomediastinum provided evidence to indicate that this assumption was correct.

The first of these, a 72 year old man, injured his chest in a fall. Chest roentgenograms showed no recent rib fracture but did indicate the presence of pneumomediastinum as evidenced by visualization of the mediastinal pleura along the left heart border and the demonstration of the reflection of this pleura from the aortic arch to the pulmonary artery in a position identical with the aortic-pulmonary stripe (Fig. 2*A*). Lateral projection confirmed the presence

of air in the mediastinum. No pneumothorax was evident. After resolution of the pneumomediastinum, the chest roentgenogram reverted to normal appearance and the aortic-pulmonary stripe was no longer visible (Fig. 2*B*).

The second patient, a 22 year old man with Hodgkin's disease, underwent mediastinal aspiration by suprasternal puncture which was followed by a large complicating pneumomediastinum. Chest roentgenograms taken at this time showed the extensive pneumomediastinum and demonstrated, in striking fashion, the reflection of the mediastinal pleura from the aortic and pulmonary artery in a configuration and position which would coincide with the aortic-pulmonary stripe (Fig. 3).

DISCUSSION

The roentgenograms of the 2 patients with pneumomediastinum indicate that the mediastinal pleural reflection at the level of the aorta and pulmonary artery coincides in position and inclination with the aortic-pulmonary stripe seen in the chest roentgenograms of normal individuals. The thickness of the pleural shadow in the patients with pneumomediastinum indicates that it represents both the parietal and visceral pleuras in this area.

The aortic-pulmonary stripe is not commonly seen, but its visualization in the chest roentgenograms of individuals without chest disease indicates that its prominence is due to anatomic variation. Its visualization in these circumstances is of no significance, other than it should not be misconstrued as evidence of mediastinal or cardiac abnormality.

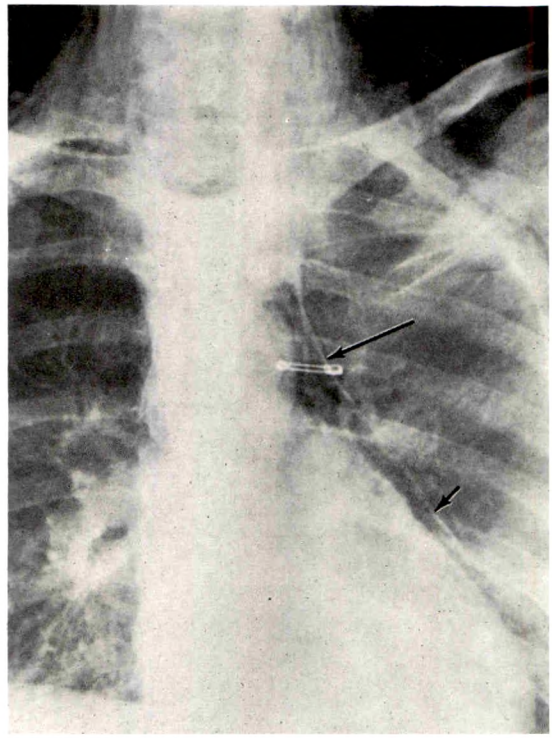


Fig. 3. Iatrogenic pneumomediastinum. The aortic-pulmonary pleural reflection is indicated by the arrows.

SUMMARY

The aortic-pulmonary stripe is the shadow of the mediastinal pleura reflected across the aortic arch and pulmonary artery.

It may be seen as a triangular density in normal individuals and should not be mistaken as a manifestation of a pathologic process.

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ABNORMALITIES OF LEFT VENTRICULAR WALL MOTION AND ANEURYSM FORMATION*

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MOST cardiac aneurysms are small and shallow and undiagnosable in life; the largest ones are easily diagnosed by roentgen ray, appearing as a bulge in the left border of the heart above the apex in the usual anterior posterior view and showing often an alarming, but apparently benign degree of expansile pulsation during systole.[9] This description appeared in a prominent text book only 20 years ago.

Although aneurysm of the left ventricle has been long recognized as a clinical entity,[4–6] until recently it has been diagnosed pre mortem only in its most blatant form.[2] With the advent of more precise diagnostic methods, specifically selective left ventricular angiography, more subtle forms of motion disturbance of the left ventricle have been described. The sensitivity of routine diagnostic tests, in particular the posteroanterior and lateral chest roentgenograms, to diagnose these abnormalities has not been previously studied.

This report is concerned with the degree of left ventricular wall motion abnormality, which can exist unsuspected, found by careful study of the routine chest roentgenograms, history, physical examination and electrocardiography.

MATERIAL AND METHOD

From a large experience with angiographic studies, primarily for coronary heart disease, at St. Luke's Hospital Center from 1969–1971, 11 illustrative cases have been selected. These cases seemed to represent the most extensive regional left ven-

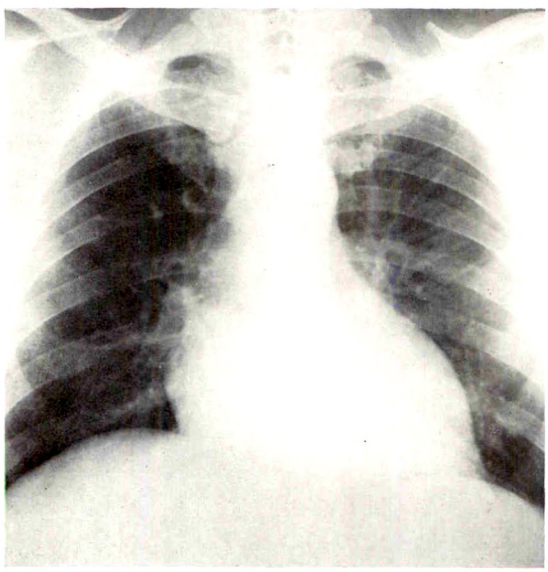


FIG. 1. Case 1. Frontal roentgenogram shows cardiac enlargement. No localized bulge or contour irregularities are present. A significant aneurysm would be difficult to suspect.

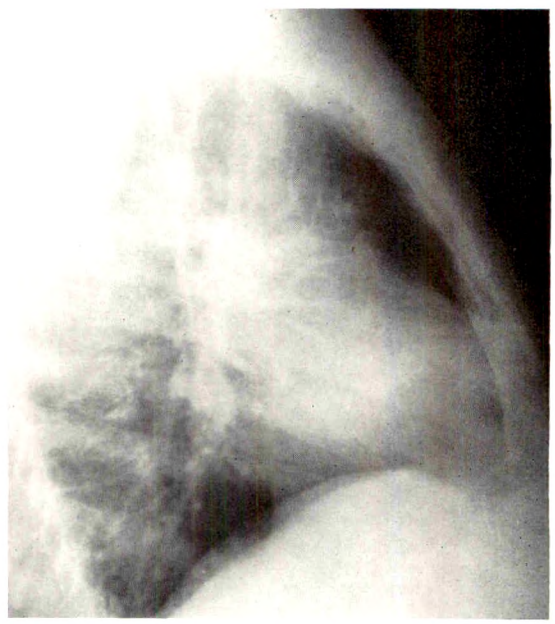


FIG. 2. Case 1. Lateral roentgenogram shows cardiac enlargement.

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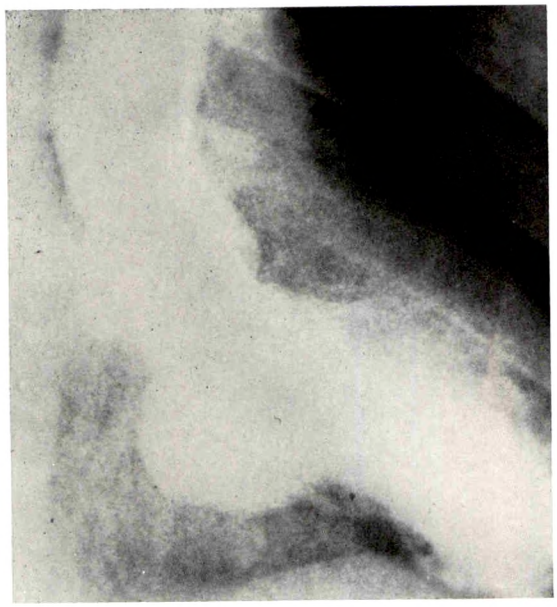


Fig. 3. Case 1. Selected frame from cine left ventriculography, right anterior oblique (RAO) projection in full systole. The distal half of the left ventricle does not contract; it is discrete and sac-like, projects laterally, and clearly pools contrast material.

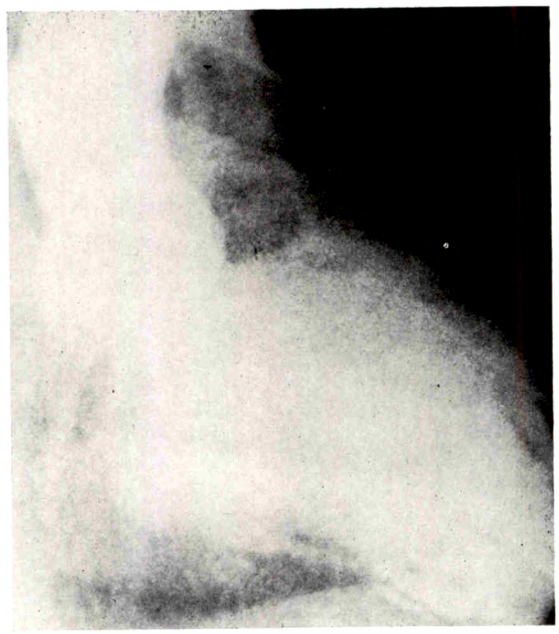


Fig. 4. Case 1. Selected frame from cine left ventriculography, RAO projection in full diastole. The aneurysm borders are lost.

tricular dysfunction, yet had normal or near-normal routine studies. Our patients were studied with a selective left ventricular injection using a retroaortic catheter. Cineangiography was recorded with a 35 mm. camera at 64 frames per second. All patients were studied in the 30° right anterior oblique (RAO) projection.

The medical modalities available for the diagnosis of ventricular aneurysm and the routine roentgenographic techniques available on each patient were reviewed, raising the question whether suspicious or definite evidence for ventricular aneurysm was present.

RESULTS

The subjects' clinical history, physical examination and electrocardiogram have been reviewed and are tabulated in Table 1.

The subjects of the study were predominantly males (10 males, 1 female), and ranged in age from 36 to 63 years. The age range is lower than the average noted in other series,[1,3] and may be attributed to

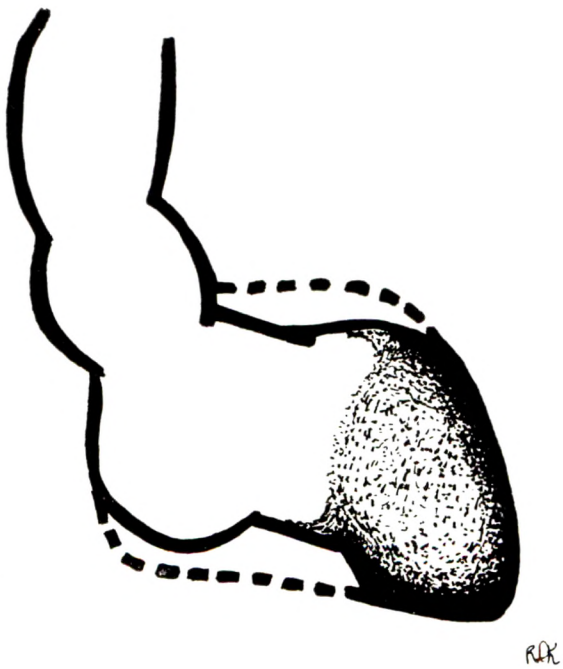


Fig. 5. Case 1. Composite drawing showing left ventricular aneurysm (stippled area) as seen during diastole and systole. Forty to 50 per cent of the left ventricle is involved.

TABLE I

CLINICAL DATA OF ELEVEN CASES OF LEFT VENTRICULAR ANEURYSM

Case	Age	Sex	Angina	M.I.	C.H.F.	Arrhythmia	Physical Examination	Electrocardiogram
I M.R.	44	M	No	Yes	Yes	No	Cardiac enlargement, S_3 and S_4	Anterolateral and diaphragmatic infarction patterns
II P.A.	36	F	Yes	Yes	Yes	No	Diffuse apical impulse, S_3	Anteroseptal infarction pattern
III C.B.	58	M	No	Yes	Yes	No	Cardiac enlargement	Anterolateral infarction pattern
IV J.T.	51	M	Yes	Yes	Yes	No	Cardiac enlargement, paradoxical split S_2, S_4	Persistent ST elevation
V M.S.	55	M	Yes	Yes	No	No	S_4	Anteroseptal infarction pattern
VI P.L.	63	M	Yes	Yes	No	No	Normal	Anteroseptal infarction pattern
VII A.H.	53	M	Yes	Yes	No	Yes	Cardiac enlargement with paradoxical apical motion S_3, S_4	Persistent ST elevation; anterior infarction pattern
VIII M.D.	59	M	Yes	Yes	No	No	Normal	Nonspecific T changes
IX E.S.	44	M	No	No	Yes	No	Cardiac enlargement, S_3	Inferior and anterolateral infarction patterns
X A.B.	48	M	Yes	Yes	No	No	S_3	Persistent ST elevation
XI C.T.	57	M	No	Yes	No	Yes	Wide apical impulse with ectopic impulse	Persistent ST elevation

M = male; F = female; M.I. = myocardial infarction; C.H.F. = congestive heart failure; S_3 = third heart sound; S_4 = fourth heart sound; S_2 = record heart sound.

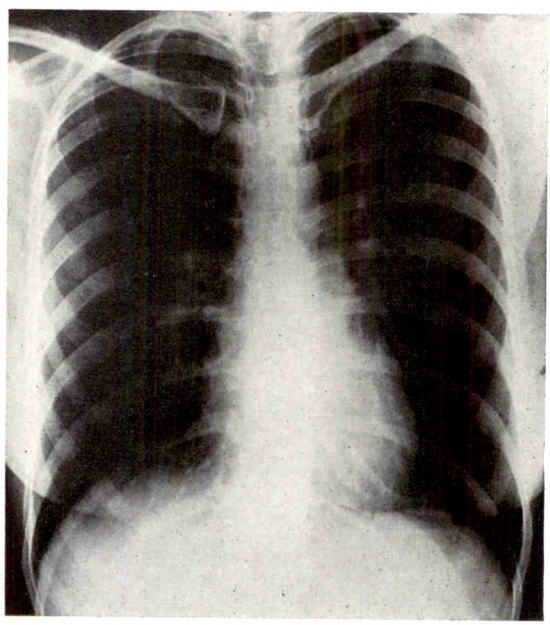


FIG. 6. Case II. Frontal roentgenogram shows normal cardiac contour and size.

patient selection, as the majority of these cases were studied as candidates for coronary bypass surgery. Seven of the 11 patients had angina pectoris for 6 months to 6 years prior to the detection of the aneurysm, while 10 of the 11 had had a myocardial infarction either by history (7 patients) or by an asymptomatic electrocardiographic finding (3 patients). None of the subjects had a history of systemic embolization. In only 2 were there arrhythmias (atrial fibrillation and ventricular tachycardia), while 5 of the 11 had congestive heart failure.

The physical examination was suggestive of an aneurysm in only 2 patients. One subject (Case VII) had paradoxical motion palpable at the apex, while another (Case XI) had an ectopic impulse medial to the wide apical impulse. In other patients, the findings were consistent with suspected

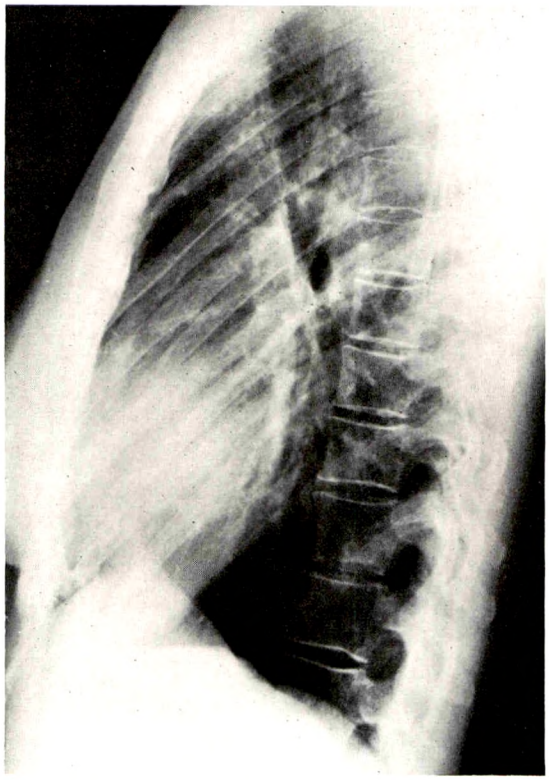


Fig. 7. Case II. Lateral roentgenogram is normal.

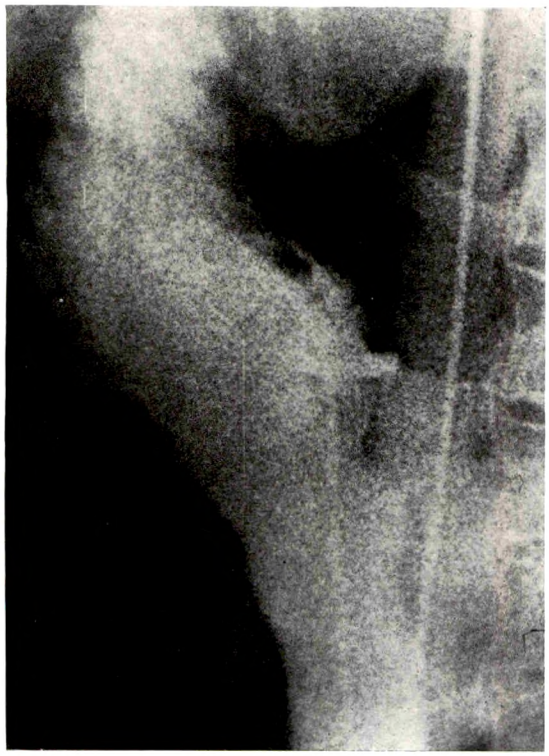


Fig. 8. Case II. Selected frame from cine left ventriculography, RAO projection in full systole. The diaphragmatic surface of the ventricle bulges downward in a clearly defined aneurysm as the more central portion of the left ventricle contracts; it is clearly sac-like and pools contrast material.

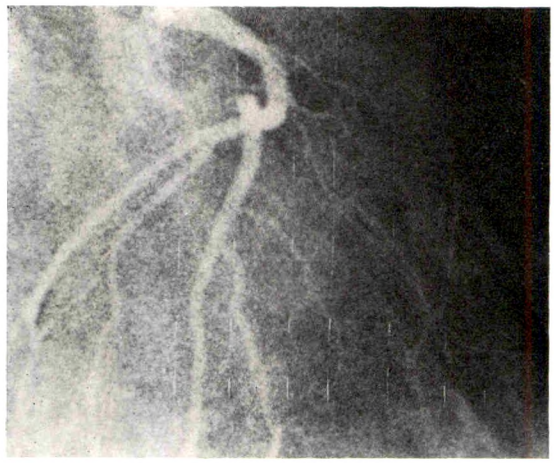


Fig. 9. Case II. Selected frame from cine left coronary arteriography in RAO projection, showing occlusion of the left anterior descending artery.

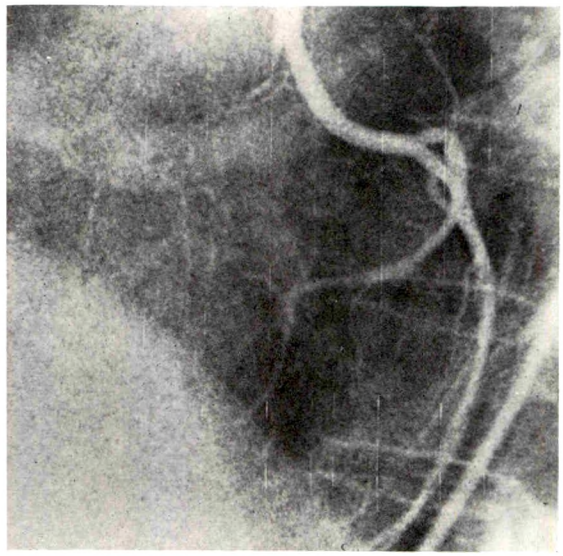


Fig. 10. Case II. Selected frame from cine left coronary arteriography in left anterior oblique (LAO) projection, showing occlusion of the left anterior descending artery.

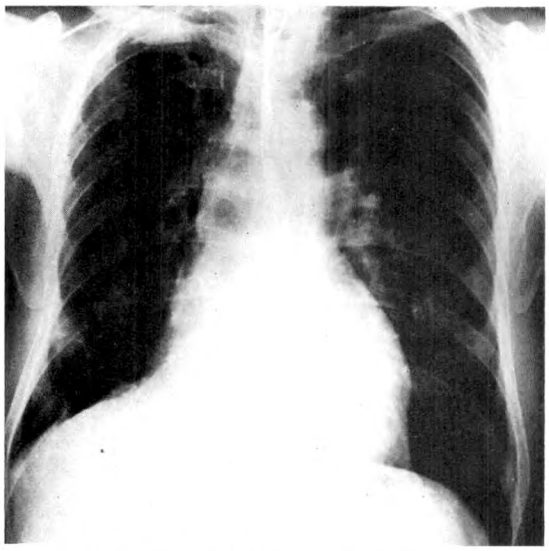


FIG. 11. Case III. Frontal roentgenogram shows some cardiac fullness. No left ventricular contour abnormality can be seen.

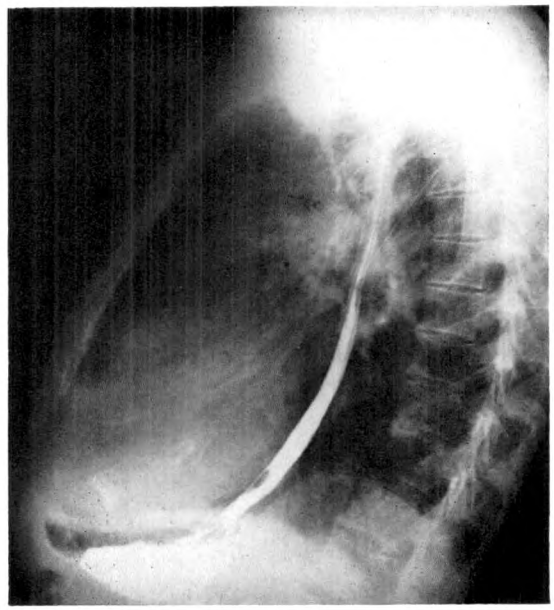


FIG. 13. Case III. Lateral roentgenogram is normal.

coronary artery disease with third or fourth heart sounds both commonly heard. In 2 patients cardiac examination was normal.

The electrocardiograms were reviewed and persistent ST elevation was noted in 4 of 11 (36 per cent) patients. Included in these 4 were the 2 subjects in whom the physical examination was suggestive of left ventricular aneurysm.

The patients' routine chest roentgenograms, cine examinations particularly in reference to the pulsatile and contractile activity of the left ventricle with and without contrast, and the selective coronary arteriograms were evaluated. The data were tabulated in Table II.

Routine posteroanterior and lateral chest

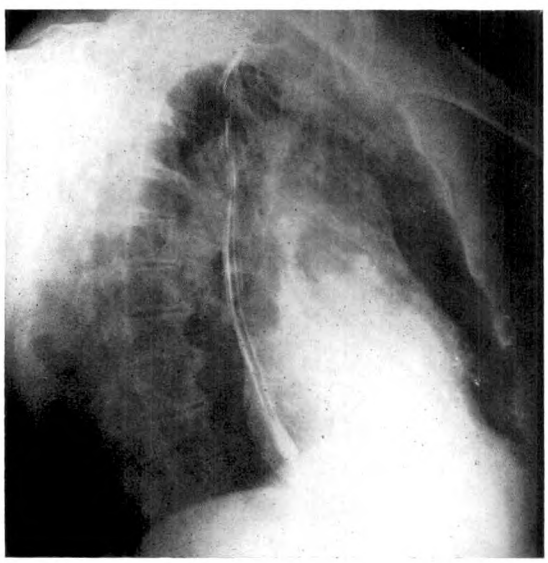


FIG. 12. Case III. RAO roentgenogram fails to reveal any irregularity of the left ventricular contour.

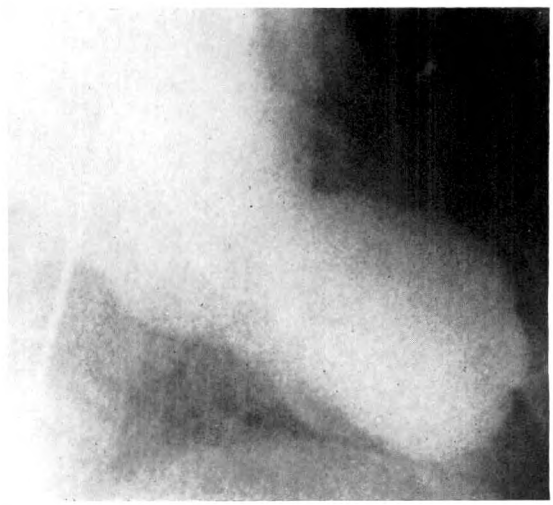


FIG. 14. Case III. Selected frame from cine left ventriculography, RAO projection in full systole. The distal half of the left ventricle does not contract; it is discrete, irregular in contour and clearly pools contrast material. It projects laterally during systole.

TABLE II

SUMMARY OF PERTINENT FINDINGS

Case	Chest Roentgenogram	Left Ventricular Activity	Coronary Arteriography	Left Ventriculography
I M.R.	*Fig. 1–5* Slight cardiac enlargement; slight left ventricular bulge	Slight apical lift with decreased contractility; systolic contraction at base of heart and diaphragmatic surface only	Total occlusion of left anterior descending; total occlusion of left circumflex marginal; 50 per cent occlusion of right coronary	*Fig. 3–5*
II P.A.	*Fig. 6–10* Normal heart size	Paradoxical expansion of apical diaphragmatic surface downward during systole; no lateral contractility; left ventricle quiet	Total occlusion of left anterior descending; normal left circumflex; small normal right coronary (*Fig. 9–10*)	*Fig. 8*
III C.B.	*Fig. 11–16* Heart at upper limits of normal	Decreased contractility; area of aneurysm showed no activity	Coronaries not studied	*Fig. 14–16*
IV J.T.	*Fig. 17–20* Large heart	Decreased contraction; apical aneurysm area showed no contractility or paradoxical motion	Normal left circumflex; subtotal occlusion proximally of left anterior descending; total occlusion of mid-portion of left anterior descending; subtotal occlusion of proximal right coronary; total at mid-portion	*Fig. 19–20*
V M.S.	*Fig. 21–23* Normal heart size	No paradoxical activity seen before contrast medium injection; slight apical paradoxical activity with contrast medium injection	Subtotal occlusion of left anterior descending; 50 per cent occlusion of right coronary	*Fig. 23*
VI P.L.	*Fig. 24–25* Slight cardiac enlargement	Decreased contraction; no paradoxical activity seen	Total occlusion of left anterior descending; 50 per cent occlusive change in left circumflex and right coronary	*Fig. 25*
VII A.H.	*Fig. 26–30* Large heart	Decreased contractility except at base	Coronaries not studied	*Fig. 28–30*
VIII M.D.	*Fig. 31–35* Heart at upper limits of normal	Decreased contractility at apex; base contracts slightly	Subtotal occlusion of left anterior descending; minimal irregularity of right coronary; subtotal occlusion of the left circumflex	*Fig. 33–35*
IX E.S.	*Fig. 36–38* Large heart	Decreased contractility; base contracts slightly	Total occlusion right coronary; subtotal occlusion of left anterior descending; 50 per cent occlusion of left circumflex	*Fig. 38*
X A.B.	*Fig. 39–41* Normal heart size	Decreased contractility; base contracts slightly better	Subtotal occlusion of left anterior descending; normal right coronary; normal left circumflex	*Fig. 41*
XI C.T.	*Fig. 42–48* Normal heart size; slight left ventricular lateral wall bulge in right anterior oblique projection	Paradoxical activity at site of aneurysm	Subtotal occlusion of right coronary; total occlusion of left anterior descending; 50 per cent occlusion of posterior atrioventricular branch of left circumflex	*Fig. 46–48*

roentgenograms revealed 3 patients with definite cardiac enlargement (Cases IV, VII, IX), 4 with minimal cardiac enlargement (Cases I, III, VI, VIII), and 4 with a perfectly normal contour (Cases II, V, X, XI). Pulsation during cineroentgenography without contrast material in the left ventricle showed 2 conclusive aneurysms with paradoxical motion and 9 inconclusive cinefluoroscopy examinations with decreased contractility. All patients during retrograde left ventricular catheterization with contrast material injection showed aneurysm formation.

Selective coronary arteriograms were abnormal in 9 patients studied with 5 cases of triple vessel, 2 cases of double vessel and 2 cases of single vessel disease. Two

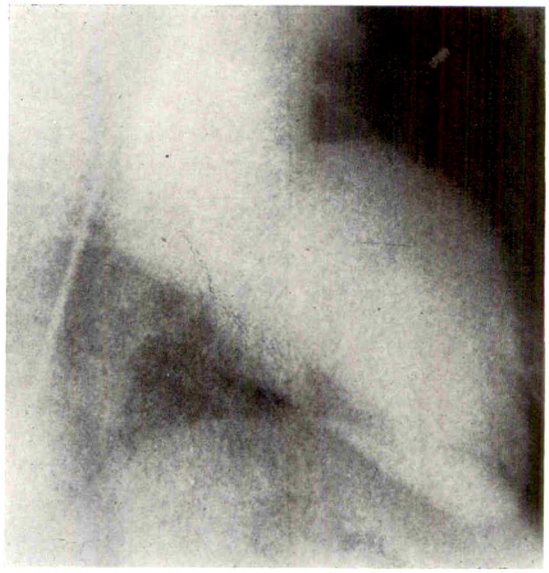


FIG. 15. Case III. Selected frame from cine left ventriculography, RAO projection in full diastole. The aneurysm borders are lost as the contour smooths out.

patients did not have selective coronary arteriography. The left ventricular angiograms and their descriptions are present in the Figures.

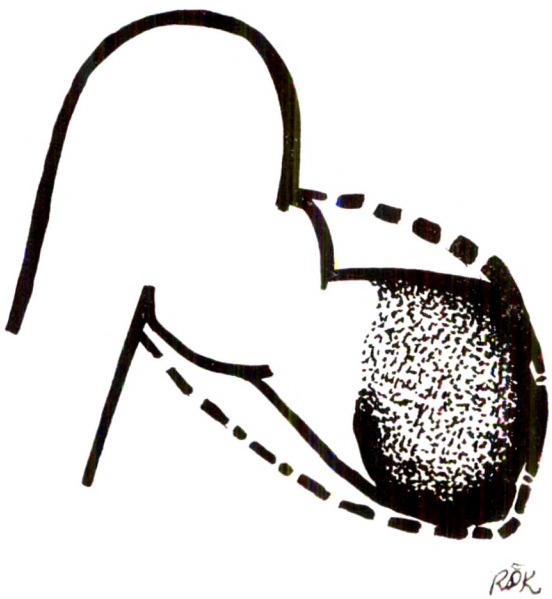


FIG. 16. Case III. Composite drawing showing left ventricular aneurysm (stippled area) as seen during diastole and systole. Over 50 per cent of the left ventricle is involved.

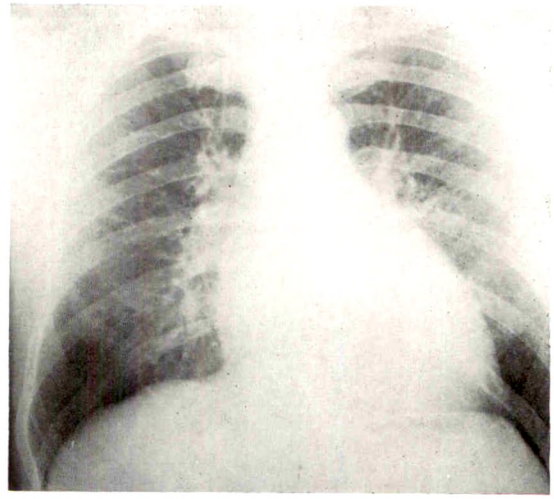


FIG. 17. Case IV. Frontal roentgenogram shows cardiac enlargement. No localized bulge or contour irregularities are present. A significant aneurysm would be difficult to suspect.

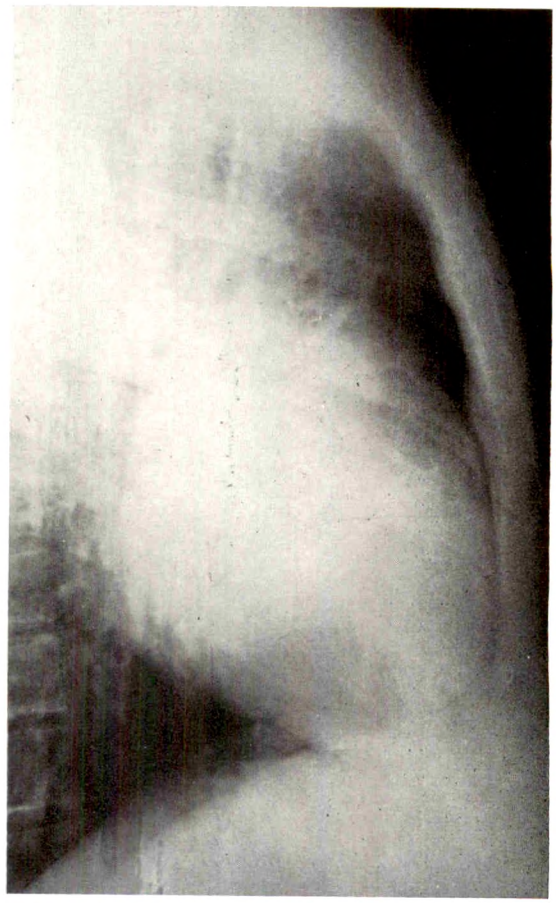


FIG. 18. Case IV. Lateral roentgenogram shows cardiac enlargement.



Fig. 19. Case IV. Selected frame from cine left ventriculography, RAO projection in full systole. There is marked loss of left ventricular contractility throughout; however, at the apex there is a small apical aneurysm, clearly defined by its sac-like contour and movement downward and laterally.

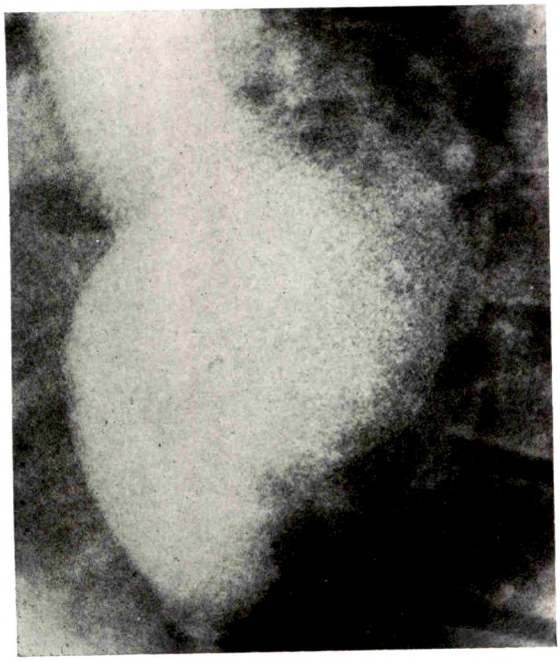


Fig. 20. Case IV. Selected frame from cine left ventriculography, LAO projection in full systole. The apical aneurysm and the general loss of contractility of the entire ventricle are seen again.



Fig. 21. Case V. Frontal roentgenogram shows normal cardiac contour and size

DISCUSSION

Obstructive disease of the coronary arteries may lead to a widely variable degree of disruption of contractility of the left ventricular myocardium.

The classic history of ventricular aneurysm with recurrent bouts of conges-

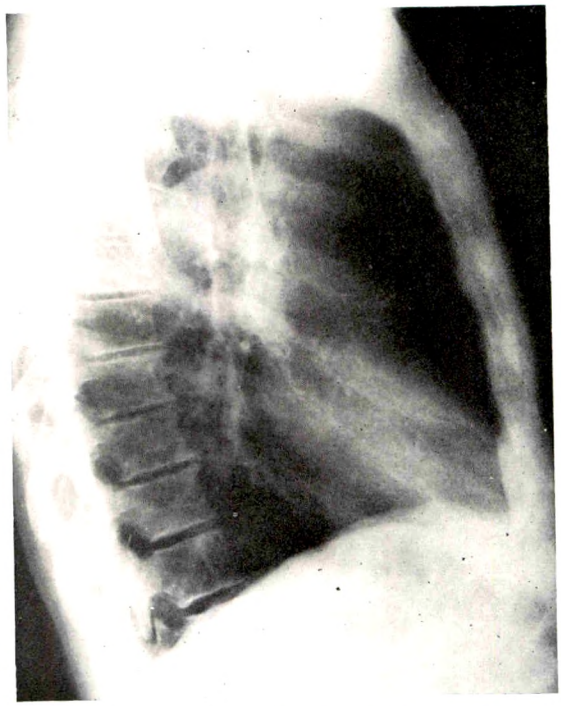


Fig. 22. Case V. Lateral roentgenogram is normal.

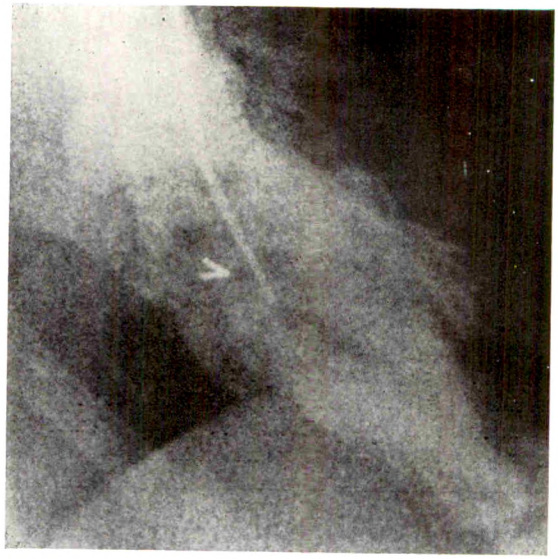


FIG. 23. Case V. Selected frame from cine left ventriculography, RAO projection in full systole. The diaphragmatic surface of the ventricle bulges downward in a clearly defined aneurysm as the more central portion of the left ventricle contracts; it is clearly sac-like and pools contrast material.

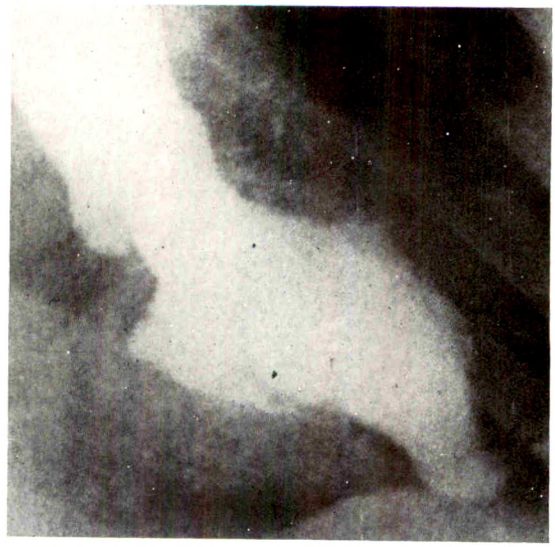


FIG. 25. Case VI. Selected frame from cine left ventriculography, RAO projection in full systole. There is a definite apical aneurysm bulging laterally and downward; it has clearly identifiable borders and pools contrast material.

tive heart failure, life threatening arrhythmias, systemic embolization, aberrantly placed or paradoxical apical impulses, and persistently elevated ST segments was not found in the majority of patients who subsequently proved to have large, discrete areas of left ventricular wall motor disturbance and aneurysm formation when examined with left ventricular angiography.

We have defined left ventricular aneu-

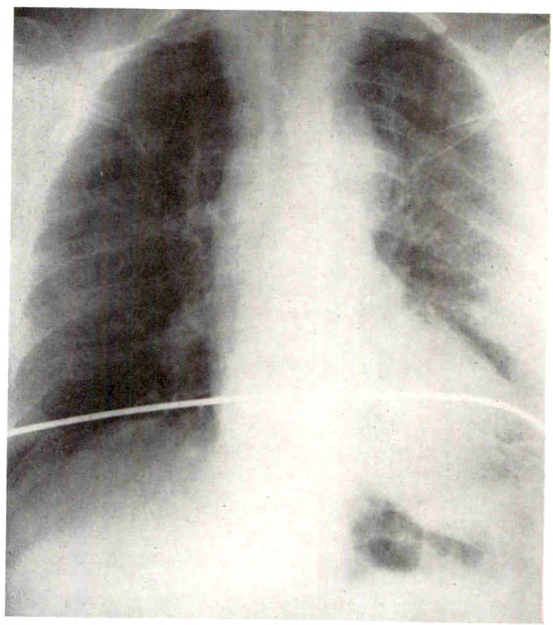


FIG. 24. Case VI. Frontal roentgenogram shows some cardiac fullness. No left ventricular contour abnormality can be seen.

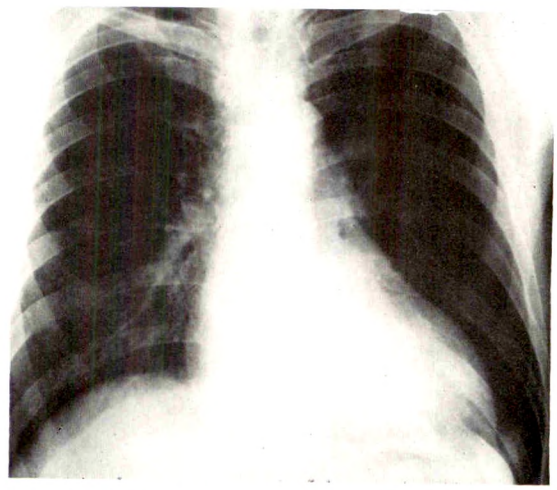


FIG. 26. Case VII. Frontal roentgenogram shows cardiac enlargement. No localized bulge or contour irregularities are present. A significant aneurysm would be difficult to suspect.

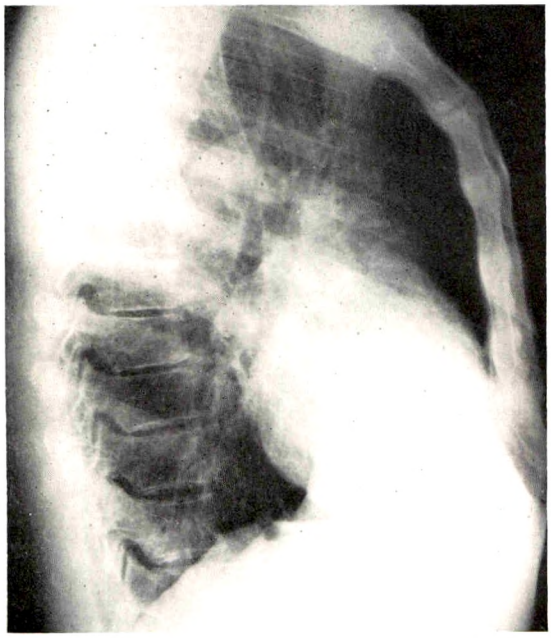


Fig. 27. Case VII. Lateral roentgenogram shows cardiac enlargement.

rysm as a permanent abnormal blood-filled dilatation of the left ventricle resulting from disease of the wall. This follows directly from the Greek "aneurysma" to

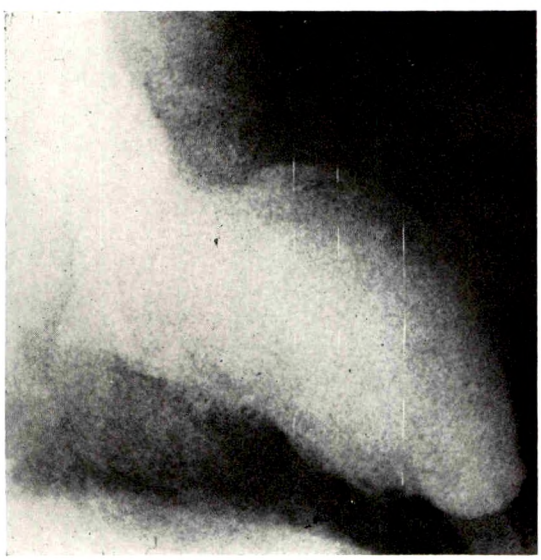


Fig. 28. Case VII. Selected frame from cine left ventriculography, RAO projection in full systole. There is marked loss of left ventricular contractility throughout; however, at the apex, there is a small apical aneurysm, clearly defined by its sac-like contour and movement downward and laterally.

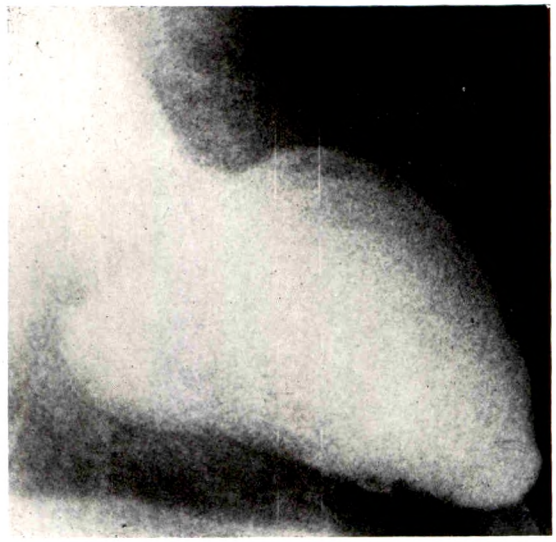


Fig. 29. Case VII. Selected frame from cine left ventriculography, RAO projection in full diastole. The aneurysm borders are lost. It should be noted that despite almost a completely akinetic ventricle as seen by comparing Figure 28 (systole) and this ventriculogram (diastole), the aneurysm is identifiable by its special characteristics.

dilate. As was demonstrated in all the subjects, the aneurysm does not contribute muscular contractions during systole; therefore, does not empty and was clearly seen pooling contrast material during cine contrast studies. It may or may not have demonstrated paradoxical activity, but was clearly discrete and sac-like in configura-

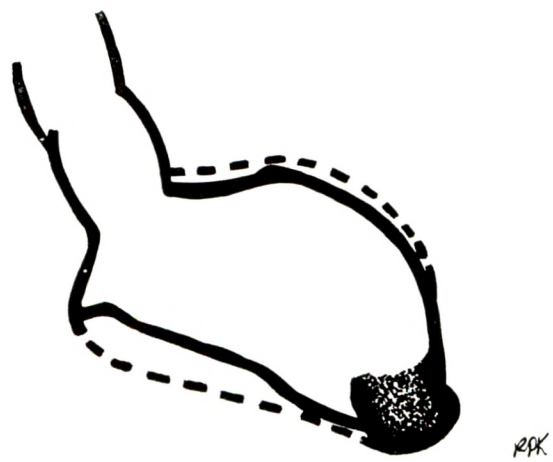


Fig. 30. Case VII. Composite drawing showing left ventricular apical aneurysm (stippled area), as seen in diastole and systole. Despite a complete akinetic ventricle, the aneurysm can be identified.

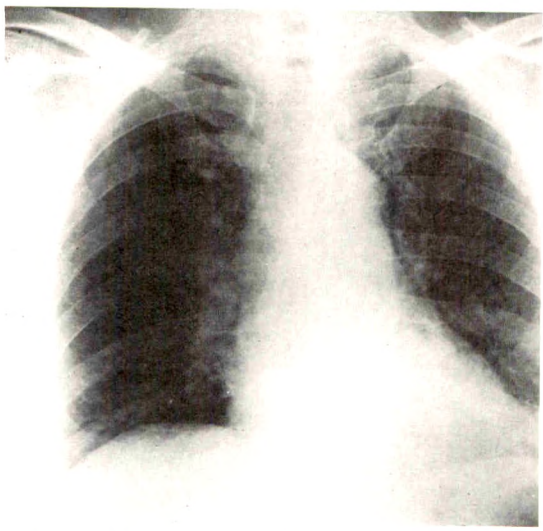


FIG. 31. Case VIII. Frontal roentgenogram shows cardiac enlargement.

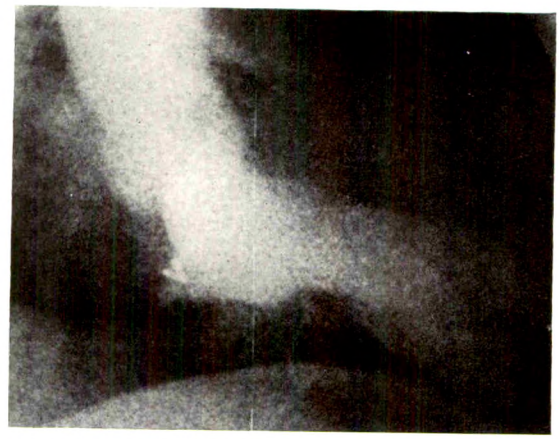


FIG. 33. Case VIII. Selected frame from cine left ventriculography, RAO projection in full systole. The distal half of the left ventricle does not contract; it is discrete and sac-like, projects laterally and clearly pools contrast material.

tion and was easily distinguished from an akinetic segment of heart wall by its characteristic shape, the pooling and stagnating of blood and the complete loss of normal heart muscle activity. All aneurysms selected had clearly identifiable borders. Location varied; *i.e.*, free wall, diaphragmatic surface and posterior wall were all represented in the series. In the right anterior oblique projection, the amount of wall involvement by aneurysm ranged from a small apical or diaphragmatic aneurysm of less than 25 per cent of the left ventricular silhouette to massive involvement of 70 per cent, converting the whole distal two-thirds of the left ventricle into a well defined discrete akinetic aneurysmal area with readily identifiable borders.

Routine studies, such as chest roentgenograms, have in our studies indicated a very

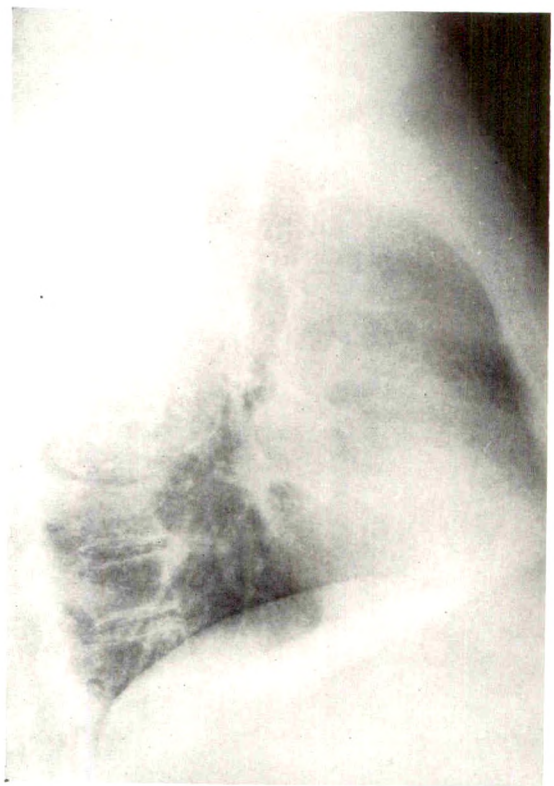


FIG. 32. Case VIII. Lateral roentgenogram shows cardiac enlargement.

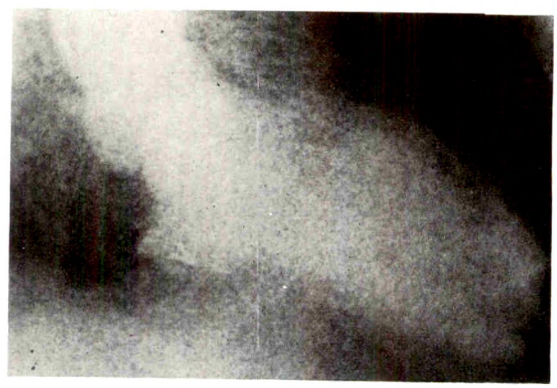


FIG. 34. Case VIII. Selected frame from cine left ventriculography, RAO projection in full diastole. The aneurysm borders are lost.



Fig. 35. Case VIII. Composite drawing showing left ventricular aneurysm (stippled area) as seen during diastole and systole. Forty to 50 per cent of the left ventricle is involved.

low sensitivity for even gross wall motion abnormalities. As much as 50–70 per cent of the left ventricular silhouette assessed in the right anterior oblique projection can be motionless or expand paradoxically with little or nothing on the chest roentgenogram to suggest the diagnosis of any wall motion defect of any kind.

We have noted that irregularity of the cardiac contour is often absent. Calcification of the myocardium appears to be very uncommon, if not rare. It was not present in any of the cases in this study.

An enlarged heart is often seen with left ventricular aneurysm but is so common without it that it has no diagnostic sig-

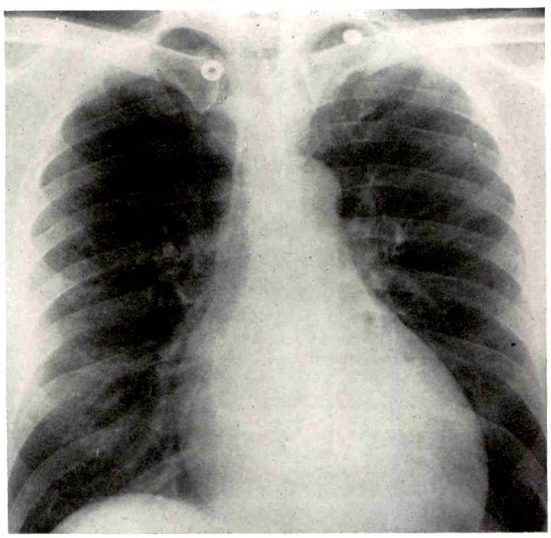


Fig. 36. Case IX. Frontal roentgenogram shows cardiac enlargement.

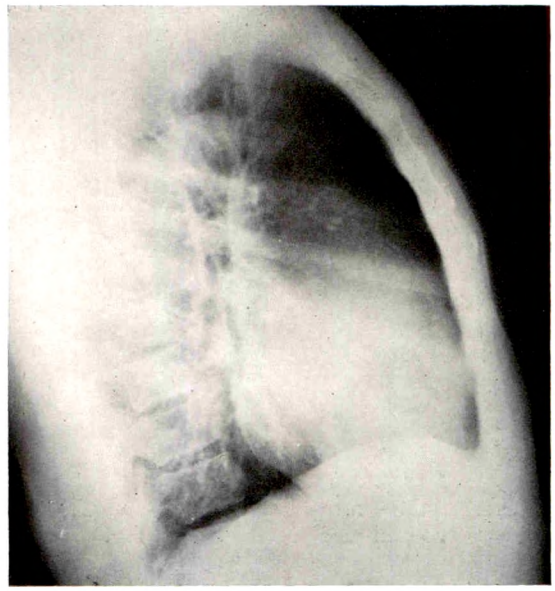


Fig. 37. Case IX. Lateral roentgenogram shows cardiac enlargement.

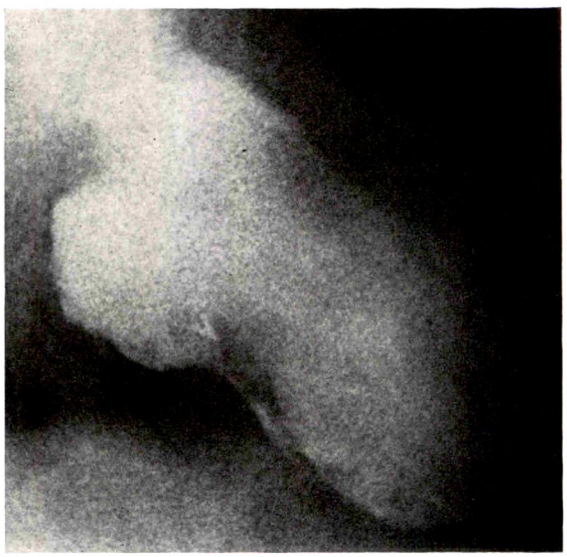


Fig. 38. Case IX. Selected frame from cine left ventriculography, RAO projection in full systole. The distal 70 per cent of the left ventricle does not contract; it is discrete and sac-like, projects laterally and clearly pools contrast material.

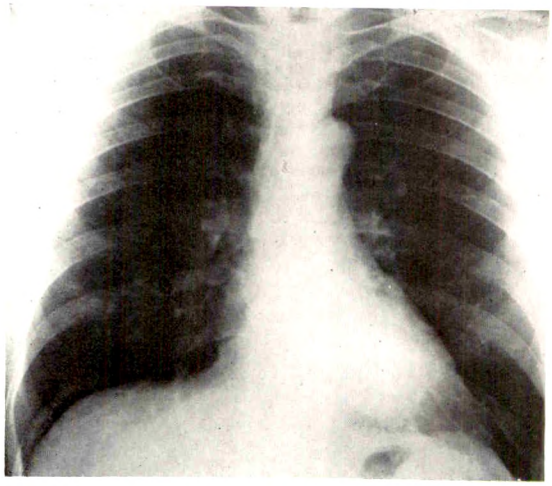


FIG. 39. Case X. Frontal roentgenogram shows normal cardiac contour and size.

nificance by itself. Massive aneurysm has been clearly seen in our cases with a normal cardiac contour and size (Cases II, V, X, XI).

Finally, fluoroscopy made more accurate by image intensification and cineroentgenography has been offered as a strong diagnostic method.[8] However, the intense frustration of trying to identify paradoxical motion of the cardiac contour by fluoroscopy

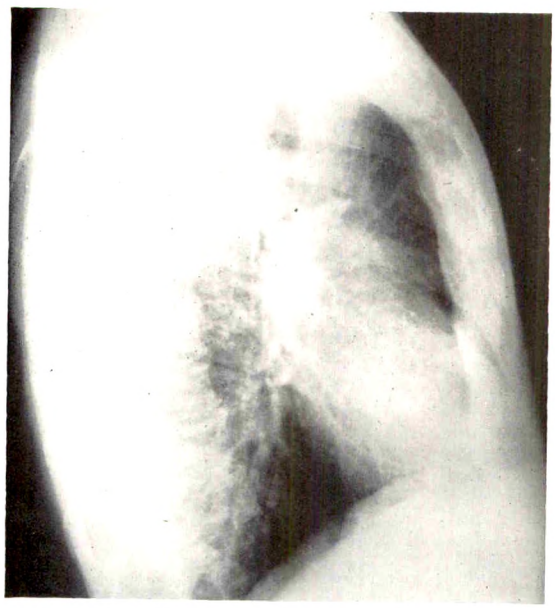


FIG. 40. Case X. Lateral roentgenogram is normal.

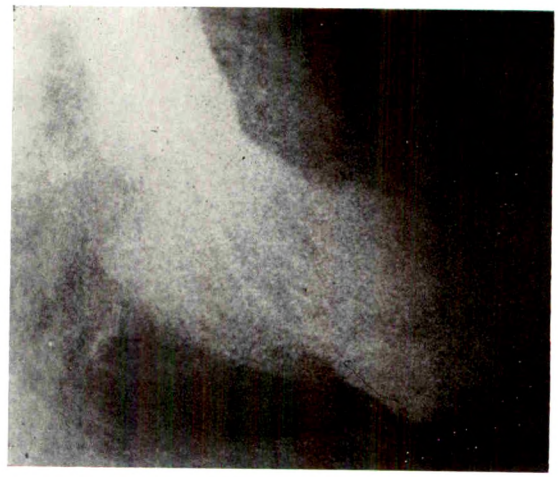


FIG. 41. Case X. Selected frame from cine left ventriculography, RAO projection in full systole. The distal half of the left ventricle does not contract; it is discrete and sac-like, projects laterally and clearly pools contrast material. A large distal aneurysm is easily identified.

in a heart with decreased contractions, some enlargement, and some degree of failure, must be common to us all.

We conclude that retrograde catheterization of the left ventricle followed by injection of contrast material documented by cineangiography in the right anterior oblique projection is the only definite way to establish abnormalities of left ventricle

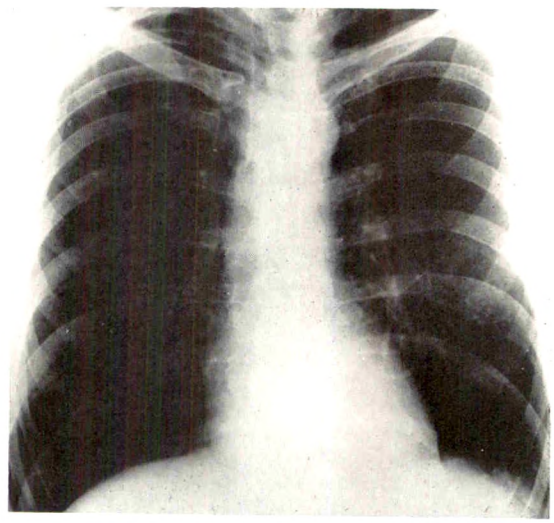


FIG. 42. Case XI. Frontal roentgenogram shows normal cardiac contour and size.

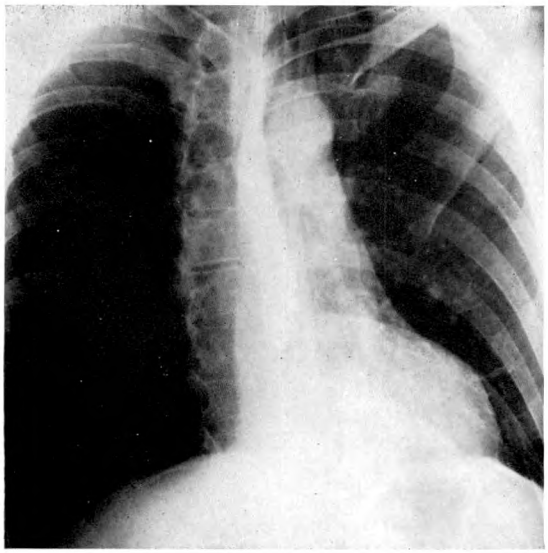


Fig. 43. Case xi. RAO roentgenogram shows left ventricular contour irregularity which suggests aneurysm.

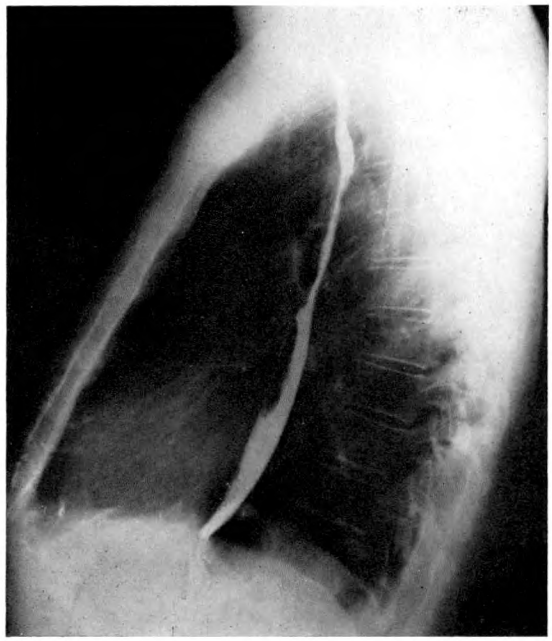


Fig. 45. Case xi. Lateral roentgenogram is normal.

wall activity and the presence of aneurysm formation.

SUMMARY

Despite recent advances in radiology and cardiology, the classic concept that abnormalities of left ventricular wall motion and aneurysm formation are identifiable by conventional roentgenographic and cardiologic techniques is still widely held.

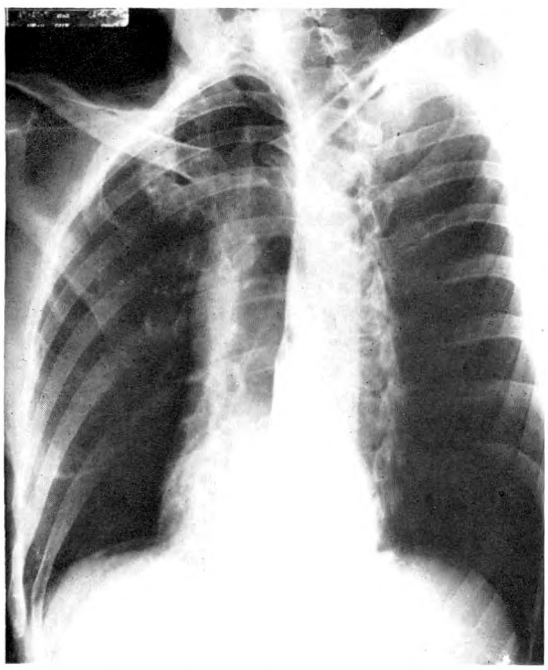


Fig. 44. Case xi. LAO roentgenogram is normal.

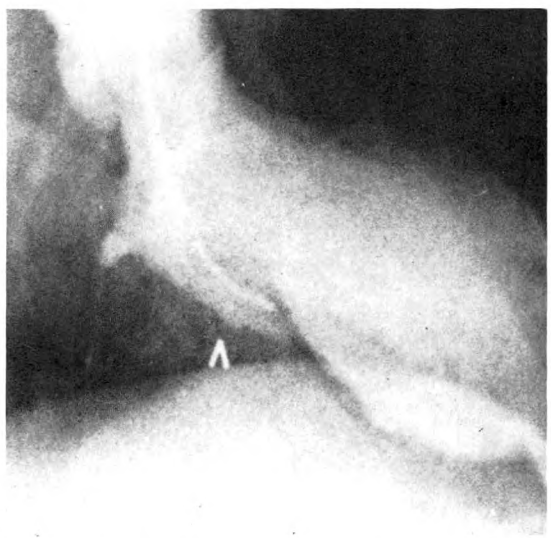


Fig. 46. Case xi. Selected frame from cine left ventriculography, RAO projection in full systole shows a large aneurysm involving two-thirds of the left ventricular chamber. Despite the contour abnormality in Figure 43, (RAO chest roentgenogram), the degree of involvement is striking.

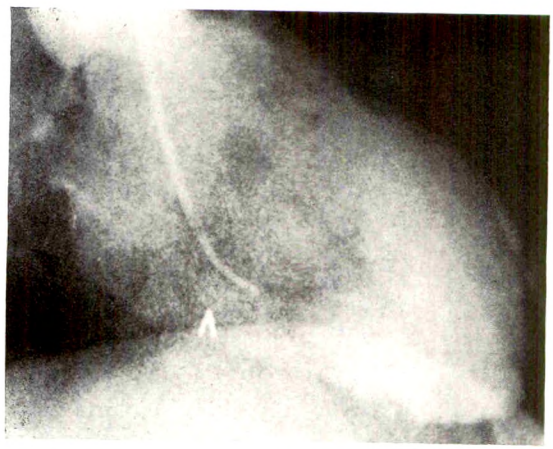


FIG. 47. Case XI. Selected frame from cine left ventriculography, RAO projection in full diastole.

Eleven cases are reported indicating that this is not true.*

Our experience clearly shows that, although the diagnosis of ventricular aneurysm is suspected, the only definite diagnostic procedure is left ventricular catheterization followed by contrast material injection and documentation of the ventricular chamber in the right anterior oblique position with cineroentgenography.

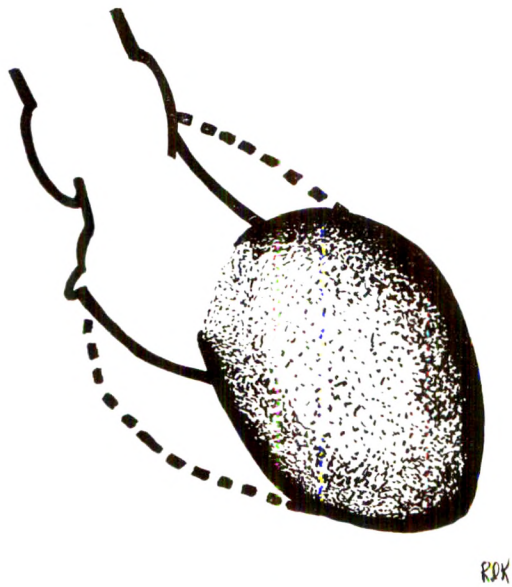


FIG. 48. Case XI. Composite drawing showing left ventricular aneurysm (stippled area) as seen during diastole and systole. Two-thirds of the left ventricle is involved.

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* Four additional cases with small normal heart size on chest roentgenography have shown large aneurysms during catheterization.

REFERENCES

1. ABRAMS, D. L., EDELIST, A., LURIA, M. H., and MILLER, A. J. Ventricular aneurysm: reappraisal based on study of sixty-five consecutive autopsied cases. *Circulation*, 1963, *27*, 164–169.
2. BENSON, R. L., HUNTER, W. C., and MANLOVE, C. H. Spontaneous rupture of heart: report of 40 cases in Portland, Oregon. *Am. J. Path.*, 1933, *9*, 295–299.
3. DOUGLAS, A. H., SFERRAZZA, J., and MARICI, F. Natural history of aneurysm of ventricle. *New York J. Med.*, 1962, *62*, 209–216.
4. DOUGLAS, A. H., and SAHIDI, F. Diagnosis of aneurysm of ventricle. *New York J. Med.*, 1963, *63*, 2949–2952.
5. DUBNOW, M. H., BURCHELL, H. B., and TITUS, J. L. Postinfarction ventricular aneurysm. *Am. Heart J.*, 1965, *70*, 753–760.
6. HOLMES, R. B., and MACFADYEN, L. R. Aneurysms of heart. *J. Canad. A. Radiologists*, 1964, *15*, 110–117.
7. KLEIN, M. D., LANE, F. J., and GORLIN, R. Hemodynamic implications of left ventricular aneurysm. *Circulation*, 1967, *35*, 614–630.
8. KURTSMAN, R. S., and LOFSTROM, J. E. Detection and evaluation of myocardial infarction by image amplification and cinefluorography. *Radiology*, 1963, *81*, 57–64.
9. WHITE, P. W. Heart Disease. Fourth edition. The Macmillian Company, New York, 1951, pp. 646–662.

THE ANGIOGRAPHIC FEATURES OF DOUBLE AORTIC ARCH*

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MOST vascular malformations in the superior mediastinum are of little or no clinical significance. A limited number of these malformations are, however, of considerable importance when they form a tight vascular ring compressing the trachea and esophagus.[4,5,8] Among the malformations necessitating surgical treatment of esophageal and tracheal compression, the double aortic arch is by far the most common.[4,8]

Although in older patients a double aortic arch may be suspected on chest roentgenograms, characteristic defects in the esophagogram are necessary for diagnosis. It has been stressed in the literature that the most precise diagnosis is afforded by angiography.[9,12] This is certainly true when both arches are patent and functioning. However, in an appreciable number of double aortic arch malformations, one arch is atretic,[1,3,10,13,14] and surprisingly little information is available regarding the angiographic features of this variation.[14] We have been impressed that under these circumstances considerable difficulty may be encountered in distinguishing double aortic arch with left arch atresia from some of the right aortic arch anomalies.

It is the purpose of this paper to analyze some of these diagnostic problems and to describe the angiographic features of double aortic arch when both arches are patent and when a portion of the left arch is atretic.

EMBRYOLOGIC CONSIDERATIONS

For understanding the development of double aortic arch anomalies, we have employed Edwards' hypothetical double aortic arch system as the basic pattern from which malformations of the aortic arch are derived.[2,11] This arch system consists of a functioning double aortic arch with a ductus arteriosus on each side (Fig. 1). The right common carotid and right subclavian arteries arise from the right arch, and the left common carotid and left subclavian arteries originate from the left arch. The descending aorta is in the midline.

This primitive double aortic arch system may remain intact or undergo one of several alterations.[2,12] Both arches may persist, and if all segments remain patent, then a functioning double aortic arch is the result;[2] or there may be regression of a segment in 1 arch. If the regression is complete, interruption of 1 arch occurs. Such is the case in the normal development when the right arch interrupts between the right subclavian artery and descending aorta.[11] Consequently, the left arch alone persists and provides continuity between the ascending and descending aorta.[11] If, however, regression is only partial, then a segment of 1 arch becomes atretic.[12]

In this communication, the term double aortic arch with partial atresia of 1 arch refers to a double aortic arch in which both arches are in continuity, but a segment of 1 arch is atretic and persists as a fibrous cord without a lumen. It is the description of the angiographic features of this anomaly and its differential diagnosis which will be presented.

CLASSIFICATION OF DOUBLE AORTIC ARCH

Double aortic arch may be classified into 2 types depending upon the patency of the 2 arches.[2,12] In Type 1, both arches are

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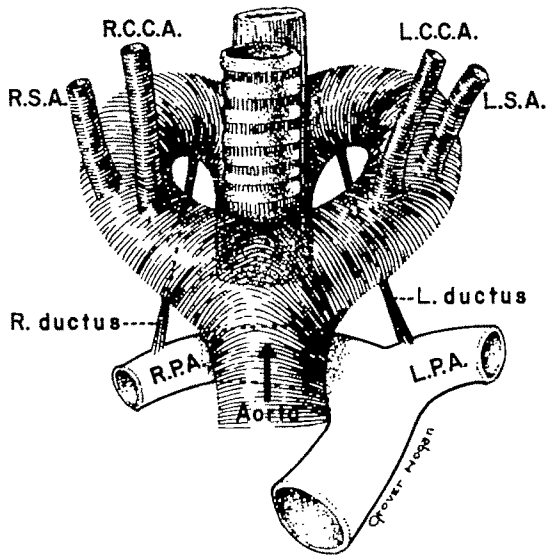


FIG. 1. Diagram of Edwards' hypothetical double aortic arch system showing bilateral aortic arches and bilateral ductus arteriosus. R.S.A. = right subclavian artery; R.C.C.A. = right common carotid artery; L.S.A. = left subclavian artery; L.C.C.A. = left common carotid artery; R.P.A. = right pulmonary artery; L.P.A. = left pulmonary artery.

patent and functioning.[12] In Type 2, 1 arch is patent but the other arch is atretic, the result of an embryologic regression.[12] Hypothetically, atresia may involve either the right or left arch; however, there are no reported cases involving the right arch.[12] When atresia of the left arch occurs, it is usually situated in the distal portion.[1,8,10,14] Theoretically, the atretic zone may be in 1 of 4 locations in the left arch, and we have classified double arch with left arch atresia into 4 subtypes, depending upon the location of the atretic segment. These sites of atresia are illustrated in Figure 2.

In Subtype 1, the atretic zone lies in the distal left arch between the left ductus arteriosus and the descending aorta.

In Subtype 2, the atretic zone is between the left subclavian artery and the left ductus arteriosus.

Subtype 3 includes those cases in which the atretic segment lies between the left common carotid and left subclavian arteries.

Those instances in which the atresia involves the anterior portion of the left arch proximal to the left common carotid artery make up Subtype 4.

REPORT OF CASES

DOUBLE AORTIC ARCH WITH BOTH ARCHES PATENT

CASE 1. *Functioning double aortic arch.* K.H., GMH C-338159. This 5 month old female was admitted with the history of difficulty swallowing and noisy respirations with stridor since birth. A routine chest roentgenogram was normal. A barium swallow examination revealed a large posterior and bilateral compression defect on the proximal esophagus (Fig. 3, *A* and *B*). Angiocardiographic studies (Fig. 4, *A* and *B*) showed a double aortic arch with both arches well formed and functioning. The right arch gave rise to the right common carotid and right subclavian arteries, and the left common carotid and left subclavian arteries originated from the left arch. The descending aorta was on the left side.

At surgery, a functioning double aortic arch

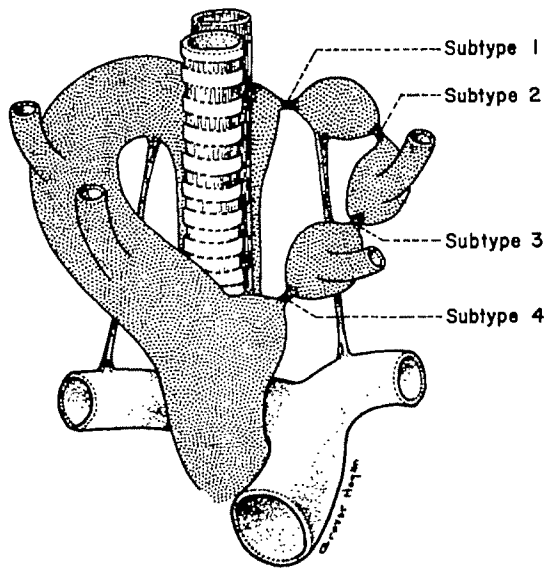


FIG. 2. Classification of double aortic arch and atresia of the left arch. The 4 subtypes are illustrated. In Subtype 1, the atretic segment is between the left ductus arteriosus and the descending aorta. In Subtype 2, the atresia is between the left subclavian artery and the left ductus arteriosus. In Subtype 3, the atretic segment lies between the left common carotid and left subclavian arteries. In Subtype 4, the atretic zone is proximal to the origin of the left common carotid artery.

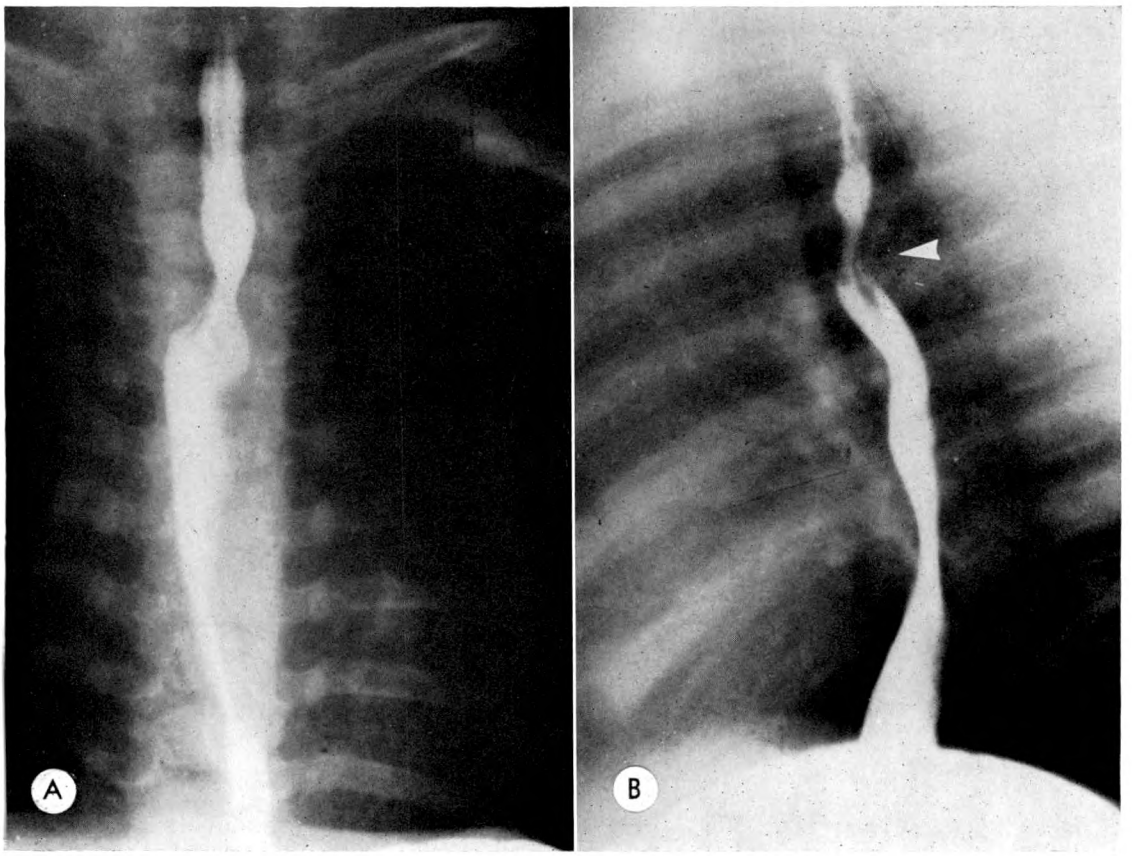


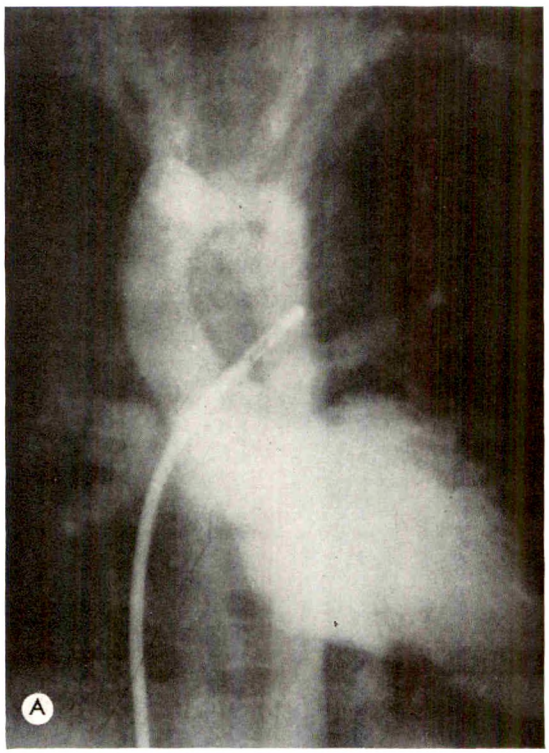
Fig. 3. Case 1. Functioning double aortic arch. (*A*) Bilateral indentations on the barium-filled esophagus at the level of the aortic arches. (*B*) Lateral esophagogram reveals posterior compression of the esophagus (arrow).

was identified. Transsection and ligation of the right arch just distal to the right subclavian artery was performed.

Case II. *Functioning double aortic arch.* D.W., a 7 month old male infant, was admitted with a history of stridor and respiratory difficulty since birth. Chest roentgenograms disclosed clear lung fields and the heart and great vessels appeared to be normal. The esophagogram showed notch-like defects on both sides of the esophagus at the level of the aortic arch. There was an oblique indentation on the posterior portion of the esophagus coursing upward from left to right.

Angiocardiography demonstrated a normal ascending aorta which bifurcated into 2 arches of approximately equal size, each giving rise to its respective brachiocephalic vessels. The 2 arches joined posteriorly to form a left-sided upper descending aorta.

Case III. *Functioning double aortic arch.* M.T., a 37 year old female with a history of difficulty of swallowing for years. She had undergone a thyroidectomy for goiter 2 years prior to admission. Following surgery she had laryngeal nerve paralysis and extensive scar formation. Her swallowing difficulties persisted, and on admission plain chest roentgenograms were normal. A barium esophagogram revealed a small pharyngeal pouch, and some motor disturbances in swallowing. Slight compression of the esophagus bilaterally and posteriorly at the level of the aortic arch was noted and a vascular ring was suspected. Catheter aortography demonstrated a functioning double aortic arch with the right brachiocephalic arteries arising from the right arch and the left brachiocephalic arteries originating from the left arch. The descending aorta was on the left side. Bronchoscopy was performed and showed no compression of the trachea. Because of the



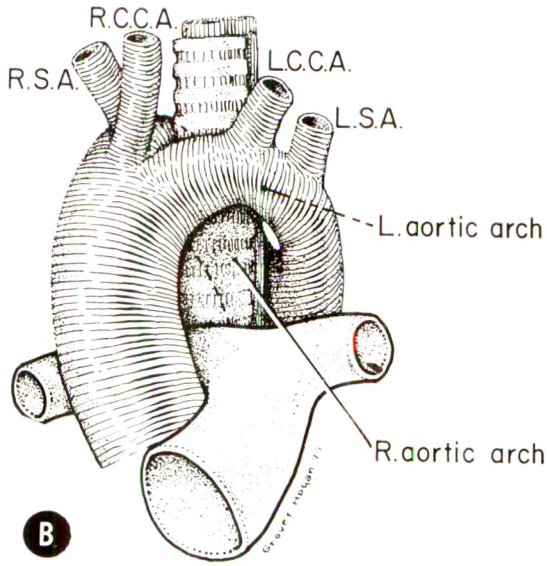


FIG. 4. Case I. Functioning double aortic arch. (*A*) Frontal levoangiocardiogram shows the ascending aorta dividing into 2 arches with the descending aorta on the left side. (*B*) Diagram of angiogram. Each arch gives rise to its common carotid and subclavian arteries, respectively.

minimal esophageal compression demonstrated on the esophagogram, surgery was not performed.

DOUBLE AORTIC ARCH WITH PARTIAL ATRESIA OF THE LEFT ARCH

CASE IV. *Double arch with left arch atresia and the atretic segment distal to the left ductus (Subtype 1).* P.T., GMH C-348891. A 9 week old male infant was found to have a heart murmur at birth. Cardiac catheterization showed the presence of a ventricular septal defect and mild pulmonary valvular stenosis. At 7 weeks of age he suddenly developed wheezing, cough, and stridor, which were relieved by extending the head and neck. A plain chest roentgenogram was unremarkable. Barium studies showed constriction of the esophagus at the level of the aortic arch (Fig. 5, *A* and *B*). A levoangiocardiogram (Fig. 6, *A–C*) revealed the presence of a right-sided aortic arch and an aortic diverticulum of the descending aorta. The first branch of the right arch was interpreted to be the left innominate artery followed by the right common carotid and right subclavian arteries. From the roentgenographic findings the impression was right aortic arch with mirror-image branching and a left ligamentum arteriosum connecting to the diverticulum of the descending aorta thus forming a vascular ring.

Thoracotomy revealed the presence of a double aortic arch, with the anterior arch the smaller of the 2 arches. The distal left arch was atretic beyond the left ligamentum arteriosum. The proximal portion of the left arch was patent and gave rise to the left common carotid and left subclavian arteries. The ligamentum arteriosum extended from the left pulmonary artery to the left subclavian artery. The distal left arch joined the descending aorta at the site of a diverticulum-like structure.

CASE V. *Double arch with left arch atresia and the atretic segment distal to the left ductus (Subtype 1).* D.J., GMH C-335854. An 8 day old infant was admitted because of persistent respiratory distress and stridor. A chest roentgenogram was within normal limits. Barium studies of the esophagus showed posterior indentation on the lateral view, and in the frontal projection there was bilateral notching at the level of the aortic arch. A levoangiocardiogram revealed a right-sided aortic arch and a right descending aorta. The left common carotid and

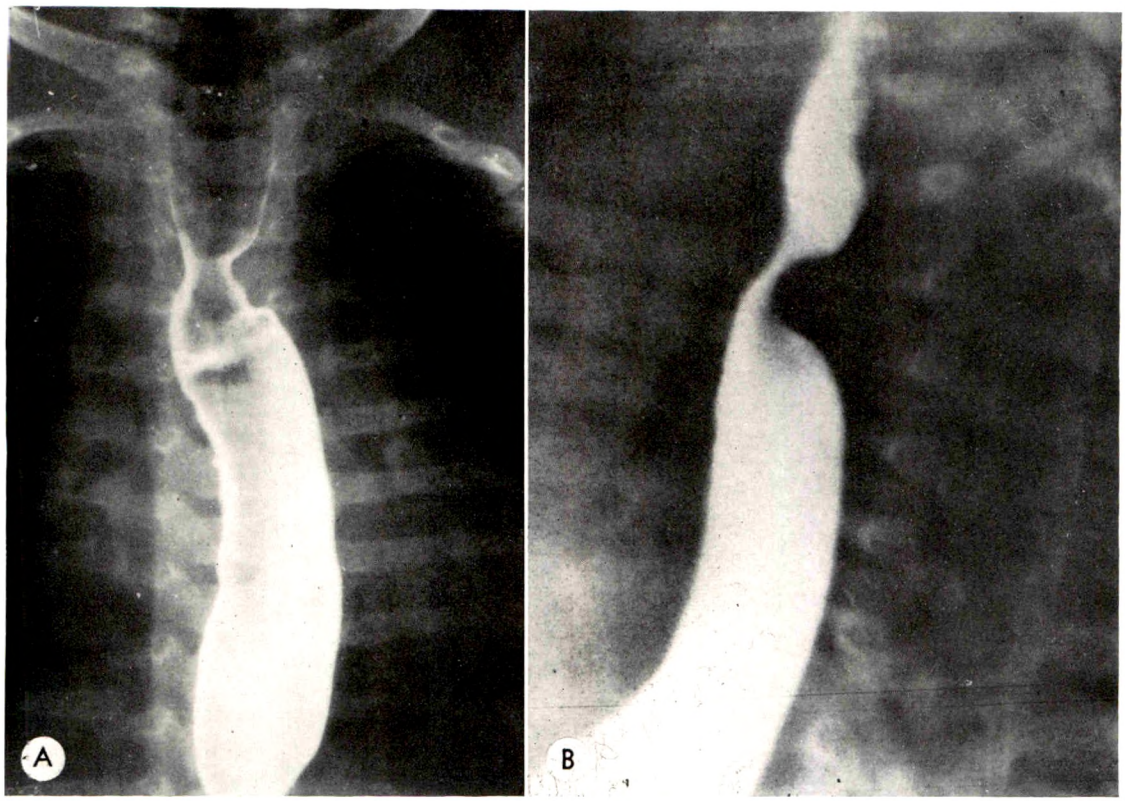


Fig. 5. Case IV. Double arch with left arch atresia (Subtype 1). (*A*) There is narrowing of the esophagus from both the right and left sides due to pressure from the 2 arches. (*B*) A large retroesophageal defect is visible on the lateral esophagogram.

left subclavian arteries appeared to have a common origin from the aorta. The right common carotid and right subclavian arteries originated as separate branches from the right arch. A diverticulum-like structure was present on the left lateral aspect of the distal right arch and was particularly prominent on the right anterior oblique retrograde aortogram. From the roentgenographic findings the impression was right aortic arch with mirror-image branching and a left ligamentum arteriosum connecting to the diverticulum of the descending aorta, thus forming a vascular ring.

Thoracotomy revealed a double aortic arch with the ascending aorta in normal position. The anterior arch was the smaller of the 2 arches and was the first branch of the ascending aorta. It crossed the midline giving rise to the left common carotid and left subclavian arteries. The left arch distal to the left ductus arteriosus was a cord-like structure and was attached to the diverticulum of the descending aorta. The right common carotid and right subclavian arteries originated from the posterior right arch.

Case VI. *Double arch with left arch atresia and the atretic segment proximal to the left ductus (Subtype 2).* T.W., GMH C-113885. This 5 month old female had developed pneumonia and respiratory distress at age 3 months. Follow-up clinical visits disclosed persistent wheeze and noisy respirations. Two days prior to admission the infant developed fever and respiratory distress. A frontal chest roentgenogram revealed clear lung fields. The heart was of normal size. A right aortic arch was present with a right-sided descending aorta. The frontal esophagogram showed the right arch to compress the right lateral wall of the esophagus with a notch-like defect in the left wall. In the lateral roentgenogram, a large retroesophageal impression was visible. A frontal levoangiocardiogram showed the ascending aorta coursing upward to the right of the trachea and descending on the right side. The right common carotid

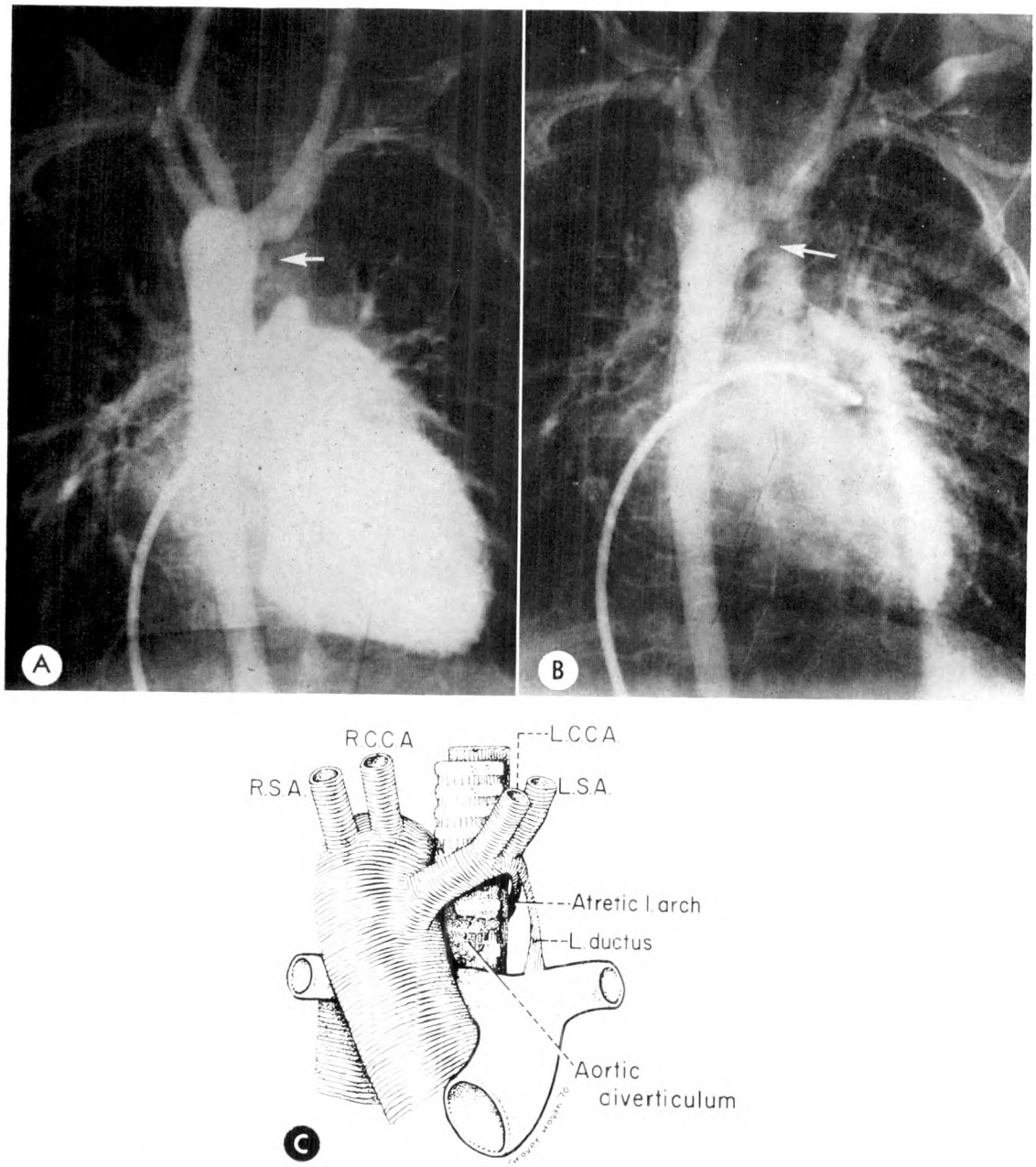


FIG. 6. Case IV. Double aortic arch with left arch atresia. (*A* and *B*) Levoangiocardiograms show right aortic arch and right-sided descending aorta. Left common carotid and left subclavian arteries appear to arise as a common trunk from the right arch. An aortic diverticulum projects from the left side of the distal right arch (arrow). (*C*) Drawing of vascular ring found at operation. The patent proximal portion of the left arch gives rise to the left common carotid and left subclavian arteries. The distal left arch is atretic and connects the left subclavian artery to an aortic diverticulum. The left ductus arteriosus extends between the left subclavian artery and the pulmonary artery.

and right subclavian arteries arose from the right arch. There was an overlap of the left common carotid and left subclavian arteries, and their site of origin could not be determined. A large outpouching was visible along the left lateral aspect of the distal right arch. From the roentgenographic findings the impression was right aortic arch with mirror-image branching

and a left ligamentum arteriosum connecting to the diverticulum of the descending aorta, thus forming a vascular ring.

Surgical exploration revealed the presence of a double aortic arch and a diverticulum on the distal portion of the right arch from which a left ligamentum arteriosum extended around the left side of the trachea and esophagus and inserted into the pulmonary artery. The anterior segment of the double arch was the smaller of the 2 arches and was the first branch of the ascending aorta giving rise to the left common carotid and left subclavian arteries. From the left subclavian artery, an obliterated vascular channel coursed to the left of the trachea and esophagus and connected the left subclavian artery to the aortic diverticulum at the point of attachment of the left ligamentum arteriosum. The right common carotid and right subclavian arteries originated as separate branches from the posterior large right arch.

Case vii. *Double arch with left arch atresia and the atretic segment proximal to the left ductus (Subtype 2)*. A.H., GMH C-115545. This 3 month old infant had a history of respiratory stridor, and difficulty with feeding since birth. A chest roentgenogram was negative except for the mediastinal configuration of a right aortic arch. A barium swallow examination revealed esophageal compression typical of a vascular ring. A levoangiocardiogram showed a large aorta which arched to the right of the trachea with a midline descending aorta. The right common carotid artery, right subclavian artery, left common carotid and left subclavian arteries all appeared to originate from the right arch. A prominent diverticulum-like structure protruded from the left upper descending aorta just above the level of the pulmonary artery. From the roentgenographic findings the impression was right aortic arch with mirror-image branching and a left ligamentum arteriosum connecting to the diverticulum of the descending aorta, thus forming a vascular ring.

Thoracotomy revealed a vascular ring formed by a large posterior right arch and a rudimentary anterior left arch. The left common carotid and left subclavian arteries arose from the patent proximal portion of the left arch. The distal portion of the left arch consisted of a stenosed vessel which connected the left subclavian artery to a diverticulum-like pouch on the descending aorta. The left ligamentum arteriosum ran from this aortic diverticulum to the pulmonary artery. The larger posterior right arch gave off the right common carotid and right subclavian arteries.

Case viii. *Double arch with left arch atresia and the atretic segment proximal to the left ductus (Subtype 2)*. P.B., GMH W-325596. A 5 month old female developed difficulty in breathing at 4 days of age. There were repeated respiratory infections with stridor. A chest roentgenogram showed the presence of a right aortic arch with the descending aorta on the right side. The esophagogram revealed constriction of the right and left sides of the esophagus, as well as a posterior indentation on the lateral projection. A right retrograde brachial aortogram disclosed a right aortic arch and a prominent diverticulum-like structure arising from the left lateral aspect of the distal arch. The left common carotid and left subclavian arteries appeared to arise as a common trunk as the first branch from the right arch, followed by the right common carotid and right subclavian arteries. From the roentgenographic findings the impression was right aortic arch with mirror-image branching and a left ligamentum arteriosum connecting to the diverticulum of the descending aorta.

Left thoracotomy showed the presence of a normal ascending aorta which divided into 2 arches. The posterior arch was the larger. The anterior arch supplied the left common carotid and left subclavian arteries and was atretic distal to the origin of the left subclavian artery. A left ligamentum arteriosum extended between the left pulmonary artery and the diverticulum of the descending aorta. The obliterated distal left arch connected the left subclavian artery to the descending aorta at the site of the attachment of the left ligamentum arteriosum.

ROENTGENOLOGIC FEATURES

DOUBLE AORTIC ARCH WITH BOTH ARCHES PATENT (TYPE I)

In 3 of our 8 patients both arches were patent and functioning. The diagnosis in these cases with functioning double aortic arch was readily established by angiography with excellent visualization of the 2 arches. Frontal angiograms revealed the following characteristics of the functioning double aortic arch (Fig. 4, *A* and *B*): the ascending aorta divided into 2 separate

arches; the left or anterior arch gave origin to the left common carotid and left subclavian arteries and pursued a course similar to that of the normal left aortic arch; the right or posterior arch coursed to the left behind the esophagus and joined the left arch and descending aorta to the left of the midline; the descending aorta was left-sided in all 3 cases.

In 1 patient, the posterior right arch appeared to be larger than the anterior left arch. In another patient, the anterior left arch appeared to be the larger arch, and in the third patient both arches were of approximately equal size.

The plain chest roentgenograms in all 3 patients revealed no abnormality of the heart or great vessels. This is not surprising as angiographic studies demonstrate both arches to cross from the right to the left and descend on the left side resembling closely the appearance of a normal left arch. Undoubtedly, the normal position of the ascending aorta, the close resemblance of the double arch to a normal left arch, and the left-sided position of the descending aorta all contributed to the normal appearance of the chest roentgenograms.

The esophagograms revealed bilateral and posterior compression defects at the level of the aortic arch (Fig. 3, *A* and *B*). Angiography showed the defect on the right side of the esophagus to be the result of pressure from the ascending aorta at its bifurcation. In each instance, an oblique defect on the posterior esophagus extending from below, upward and to the right, clearly was related to pressure from the posterior right arch. The narrowing of the left side of the opacified esophagus resulted from extrinsic pressure from the 2 arches as they joined to form the descending aorta (Fig. 4, *A* and *B*).

DOUBLE AORTIC ARCH WITH PARTIAL ATRESIA OF ONE ARCH (TYPE 2)

In 5 of our 8 cases the right arch was patent and the left arch was atretic. In 2 of the 5 patients, the atretic segment was distal to the aortic insertion of the left ductus arteriosus (Subtype 1) (Fig. 2). In the remaining 3 patients, the atretic segment was proximal to the aortic attachment of the left ductus arteriosus (Subtype 2) (Fig. 2).

The angiographic findings in all 5 patients were similar. In each instance, the principal finding was the presence of a right aortic arch. In 4 instances, the descending aorta was right-sided. In 1 patient, the descending aorta had a midline position. In all patients, the patent proximal portion of the left arch resembled a left innominate artery giving rise to a left common carotid and to a left subclavian artery. A large aortic diverticulum was visible at the junction of the right arch and upper descending aorta. In 2 patients studied in the oblique positions, this structure was particularly well demonstrated.

Plain chest roentgenograms in our patients with double aortic arch and left arch atresia were rewarding in showing the presence of a right aortic arch and a right descending aorta in 3 of the 5 cases. In none of our patients did the lateral chest roentgenogram disclose an indentation on the tracheal air shadow. Except for angiography, the most helpful roentgenographic study was the esophagogram. In each instance, there was a compression defect on both the right and left sides of the esophagus with an indentation visible on the posterior esophageal wall.

In our series there are no examples of Subtype 3 double aortic arch (atretic segment between the left common carotid and left subclavian arteries) (Fig. 2). A few cases of this anomaly have been reported.[6,7] In this malformation, the first branch of the arch is the left common carotid artery followed by the right common carotid and right subclavian arteries. The left subclavian artery arises as the fourth branch from an aortic diverticulum. This aortic diverticulum is present at the junction of the right arch and descending aorta and represents the most distal remnant of the embryonic left arch. The left ductus arteriosus extends between the aortic diverticu-

lum and the left pulmonary artery and passes to the left of the esophagus.

No examples of Subtype 4 double aortic arch (atretic segment between the ascending aorta and left common carotid artery) have been reported (Fig. 2).[12] Theoretically, the first branch of the right arch would be the right common carotid artery followed by the right subclavian artery and the left innominate artery in that order. A left ductus arteriosus would extend between the descending aorta and the left pulmonary artery. This anomalous configuration remains a hypothetical possibility. Therefore, we will not include Subtype 4 in our discussion of the differential diagnosis of double arch atresia.

PROBLEMS IN DIAGNOSIS

Most congenital malformations of the aortic arch producing complete encirclement of the trachea and esophagus resolve themselves into cases of either double aortic arch or some variant of right aortic arch.[4,5,8] Contrast studies readily identify the functioning double aortic arch. However, the differentiation of double aortic arch with partial atresia of the left arch from the right aortic arch anomalies presents considerable difficulty. In our 5 patients with double aortic arch with left arch atresia, the angiographic studies appeared to be examples of right aortic arch anomalies, and for that reason an erroneous preoperative diagnosis was made.

In double aortic arch with left arch atresia, the responsible embryologic defect is partial regression of a portion of the embryonic left arch.[2] If, instead of partial regression, there is complete interruption of the embryonic left arch at the same location, then a right aortic arch malformation is the result.[11]

In Figure 7, *A–C*, the development of double aortic arch Subtype 1 is illustrated. Partial regression of the left arch between the left ductus arteriosus and descending aorta results in a fibrous cord in this segment producing a vascular ring. In this anomaly, the patent proximal portion of the left arch gives rise to the left common carotid and left subclavian arteries and has the appearance of a left innominate artery. The left ductus arteriosus is connected to the left subclavian artery. The distal portion of the left arch may persist as an enlargement or diverticulum like structure. The barium swallow examination shows posterior and bilateral compression defects in the esophagus.

If, instead of partial regression, there is complete interruption of the embryonic left arch at this site, right aortic arch with mirror-image branching of the arch vessels is the result (Fig. 8, *A–C*).[11] A left ductus arteriosus connects the left subclavian

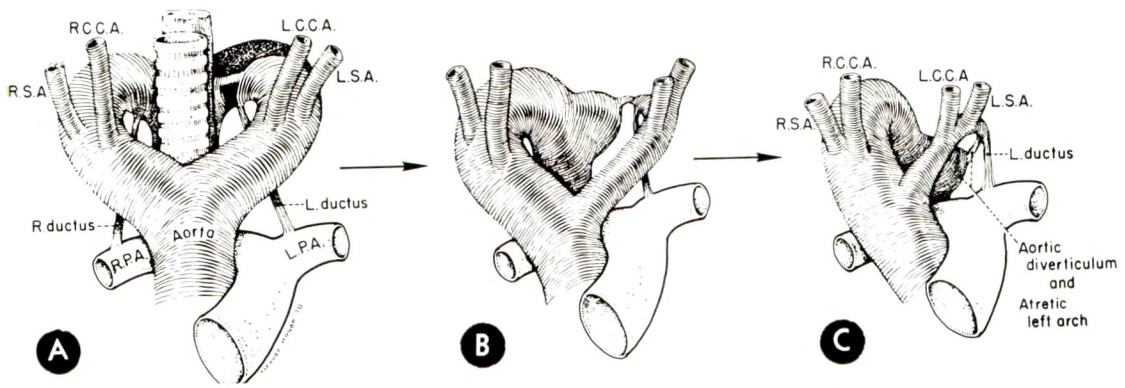


Fig. 7. (*A–C*) Development of double aortic arch with atresia of the left arch (Subtype 1). The atretic segment is distal to the left ductus arteriosus which connects to the left subclavian artery. A portion of the left arch between the left ductus arteriosus and descending aorta (shaded area) has undergone partial regression.

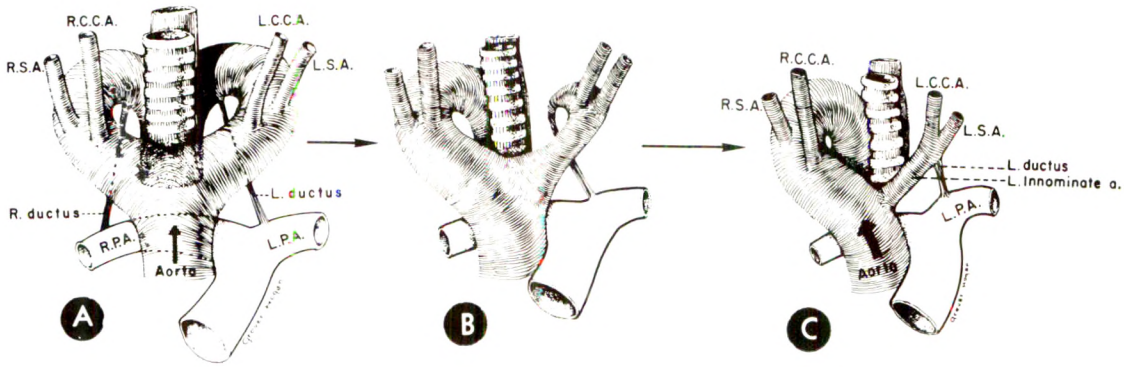


FIG. 8. (*A–C*) Development of right aortic arch with mirror-image branching of the arch vessels and a left ductus arteriosus connecting the pulmonary artery to the left subclavian artery. There is no vascular ring present.

artery to the left pulmonary artery. Thus, there is no retroesophageal component present to form a vascular ring and the esophagogram is normal. Therefore, these 2 conditions, although angiographically similar, can be differentiated by a barium swallow examination. This type of right aortic arch is usually associated with cyanotic congenital heart disease.[11]

In Subtype 2 double aortic arch, the atretic segment is between the left subclavian artery and the left ductus arteriosus (Fig. 9, *A–C*). This subtype is similar to Subtype 1 with the exception that in Subtype 2 the left ductus arteriosus extends from the bulbous remnant of the posterior left arch to the left pulmonary artery.

When there is complete interruption of the embryonic left arch in this same segment, a right aortic arch with mirror-image branching of the arch vessels is found (Fig. 10, *A–C*). Now, however, the left ductus extends from the aortic diverticulum of the descending aorta to the pulmonary artery. In both Subtype 2 double aortic arch and in this rare type of right aortic arch a vascular ring is present. These 2 conditions cannot be differentiated by roentgenologic studies from each other or from Subtype 1 double arch. Only a few examples of this type of right aortic arch have been reported.

The development of Subtype 3 double aortic arch is shown in Figure 11, *A–C*. The

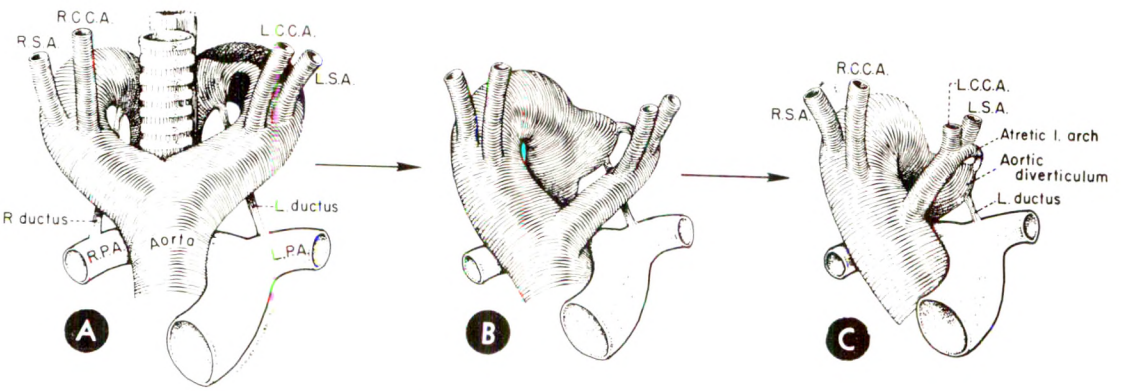


FIG. 9. (*A–C*) Development of double aortic arch with atresia of the left arch (Subtype 2). A portion of the left arch between the left subclavian artery and the left ductus arteriosus (shaded area) has undergone partial regression. The left ductus arteriosus extends from the aortic diverticulum to the left pulmonary artery.

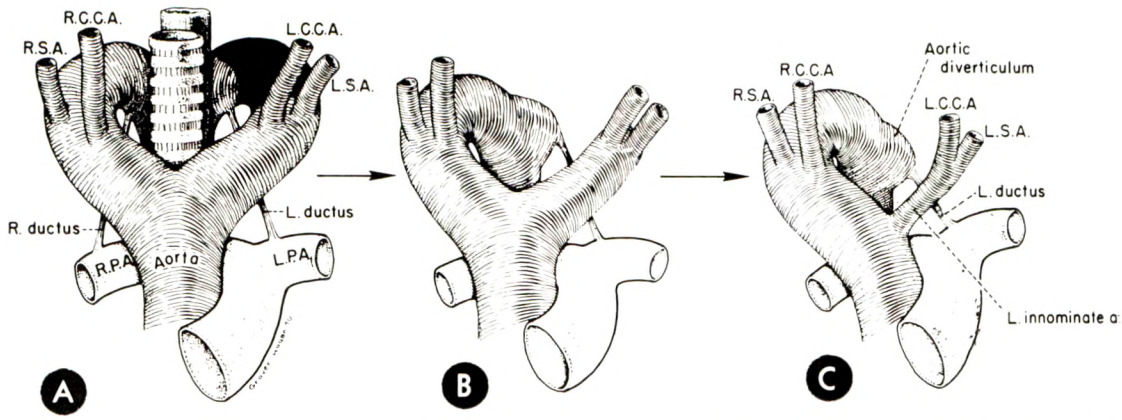


Fig. 10. (*A–C*) Development of right aortic arch with mirror-image branching of the arch vessels and a left ductus arteriosus connecting the pulmonary artery to the descending aorta. There has been complete regression of the left arch between the left subclavian artery and the left ductus in the area indicated in black. A vascular ring is present.

atretic zone lies between the left common carotid and left subclavian arteries. The first branch of the arch is the left common carotid artery followed by the right common carotid and right subclavian arteries. The left subclavian artery arises as the fourth branch from an aortic diverticulum of the descending aorta. This diverticulum represents the most distal remnant of the embryonic left arch. The atretic segment of the left arch persists as a fibrous band between the left common carotid and left subclavian arteries, producing a vascular ring. The left ductus extends between the aortic diverticulum and the left pulmonary artery, and passes to the left of the esophagus.

Complete interruption of the embryonic left arch between the left common carotid and left subclavian arteries results in a right aortic arch with an aberrant left subclavian artery (Fig. 12, *A–C*). The left ductus arteriosus extends from the descending aorta to the pulmonary artery, completing a vascular ring. This is the most common type of right aortic arch;[11] however, very few patients with this anomaly have clinical symptoms. The angiographic findings in right arch with aberrant left subclavian artery are identical to those of

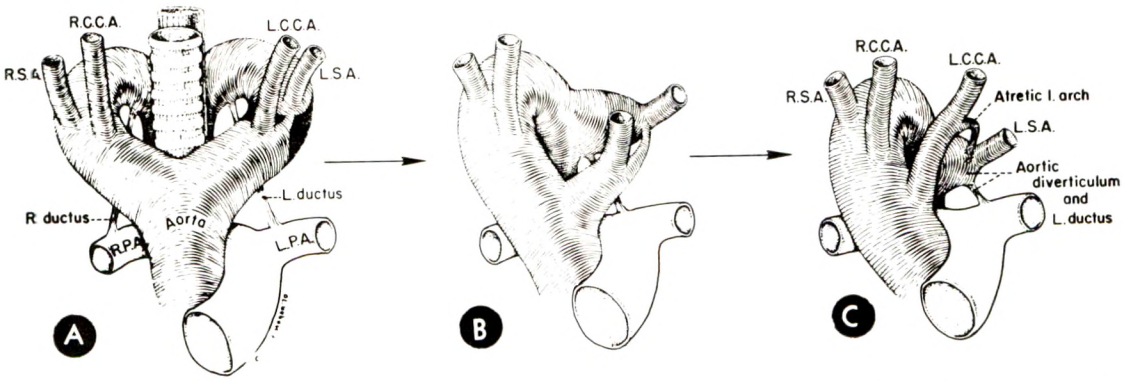


Fig. 11. (*A–C*) Development of double aortic arch with atresia of the left arch (Subtype 3). The atretic segment is between the origins of the left common carotid and left subclavian arteries. A portion of the left arch (shaded area) has undergone partial regression.

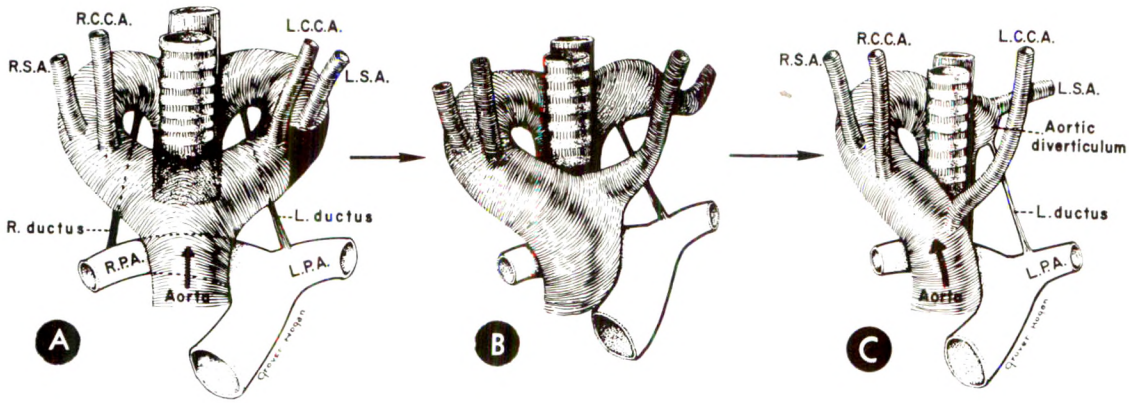


FIG. 12. (*A–C*) Development of right aortic arch with an aberrant left subclavian artery. There has been interruption of the left arch between the left common carotid and left subclavian arteries.

Subtype 3 double aortic arch. In both conditions the esophagogram shows compression defects, and these 2 anomalies cannot be differentiated from each other by roentgenologic studies.

Figure 13, *A–F*, shows some of the subtypes of double aortic arch with left arch atresia and their respective right arch counterparts. It is apparent from this diagram that the only anatomic difference between double aortic arch with left arch atresia and its right aortic arch counterpart is the presence of an atretic segment in the double arch. As the atretic segment is not patent and functioning, it cannot be demonstrated angiographically. Therefore, double aortic arch with left arch atresia and its right aortic arch counterpart will show identical angiographic findings.

The angiographic key to the separation of Subtypes 1 and 2 double aortic arch and right arch with mirror-image branching from Subtype 3 double aortic arch and right arch with an aberrant left subclavian vessel is the origin of the left subclavian artery.

When the aortic diverticulum is clearly visualized and the left subclavian artery does not arise from this structure, then either double aortic arch Subtypes 1 and 2 or right aortic arch with mirror-image branching is present (Fig. 14, *A–C*). When the left subclavian artery can be identified as the fourth branch of the arch arising from an aortic diverticulum, then it is either Subtype 3 double aortic arch or right aortic arch with an aberrant left subclavian artery (Fig. 14, *D* and *E*).

Good angiographic studies with oblique projections will usually portray the aortic diverticulum and reveal the origin of the left subclavian artery. However, at times there may be superimposition of the left brachiocephalic vessels on the frontal angiograms, obscuring the aortic diverticulum and the origin of the left subclavian artery. Under these circumstances, it is impossible to distinguish the double arch subtypes from each other or from their respective right arch counterparts.

From the above discussion, the following points are to be emphasized. When the angiographic findings are those of a right aortic arch with mirror-image branching in the presence of a symptomatic vascular ring, the most likely diagnosis is double aortic arch with left arch atresia. This is true as the type of right arch with mirror-image branching that produces a vascular ring is extremely rare. When the angiographic findings are those of a right aortic arch with an aberrant left subclavian artery, the diagnosis may be either double aortic arch with left arch atresia (Subtype 3) or the right aortic arch anomaly. In this instance, the most likely diagnosis is right

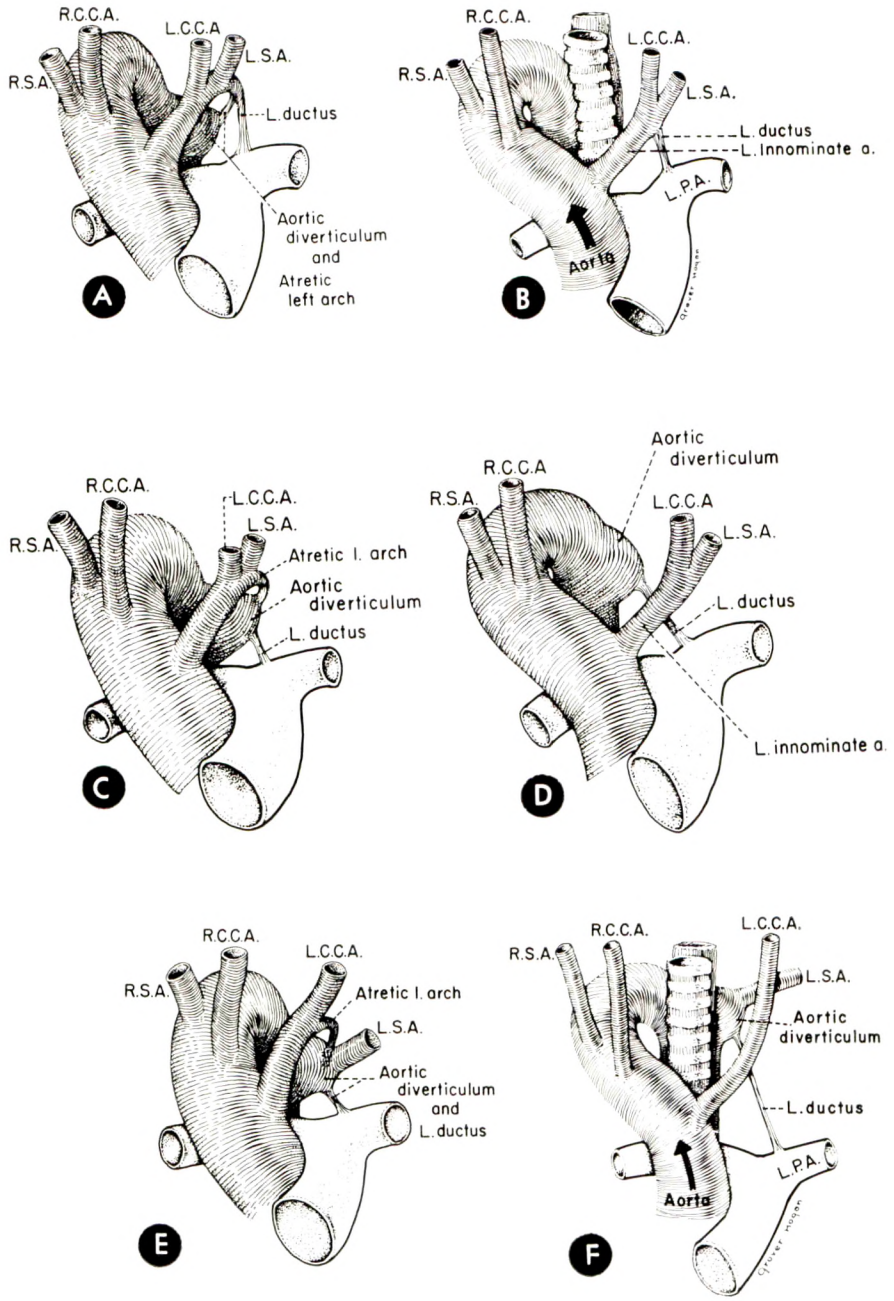


FIG. 13. Double aortic arch with left arch atresia, Subtypes 1, 2 and 3 (*left column*); and their respective right aortic arch counterparts (*right column*). (*A*) Subtype 1. (*B*) Mirror-image branching, no vascular ring. (*C*) Subtype 2. (*D*) Mirror-image branching, rare type. (*E*) Subtype 3. (*F*) Aberrant left subclavian artery. The anatomic difference between the double arch malformation and its right arch counterpart is the persistence of an atretic segment in the double arch anomaly.

aortic arch with an aberrant left subclavian artery, because double aortic arch Subtype 3 is extremely rare.

Table 1 summarizes some of the important roentgenologic features of double aortic arch and the right arch anomalies

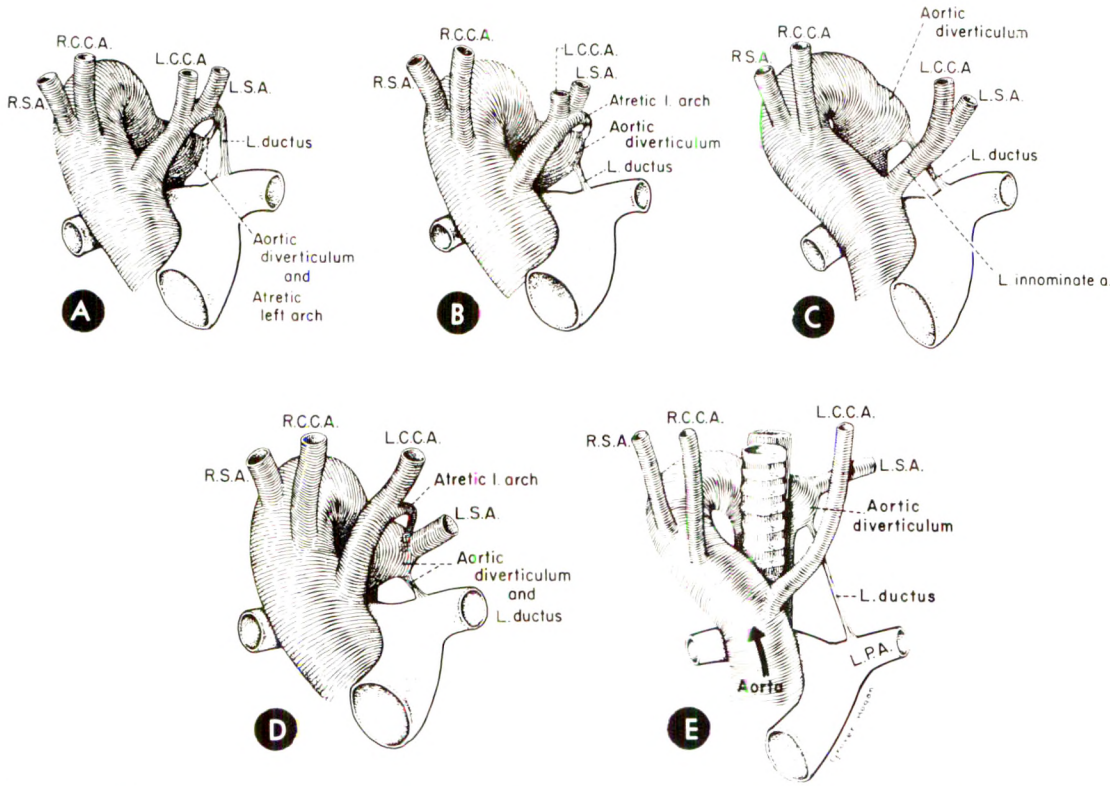


FIG. 14. Angiographic features important in the differential diagnosis of double aortic arch with left arch atresia. (*A–C*) The left subclavian artery does not arise from the aortic diverticulum in: *A*, Subtype 1; *B*, Subtype 2; and *C*, Right arch with mirror-image branching. (*D* and *E*) The left subclavian artery originates from the aortic diverticulum in: *D*, Subtype 3; and *E*, Right arch with aberrant left subclavian artery. Right aortic arch with mirror-image branching of the arch vessels and Subtypes 1 and 2 double aortic arch with left arch atresia show identical angiographic findings. These conditions can be differentiated from right arch with aberrant left subclavian artery and Subtype 3 double aortic arch by the origin of the left subclavian vessel.

that must be considered in the differential diagnosis.

TECHNICAL CONSIDERATIONS

Seven of our 8 patients with double aortic arch were infants, and angiocardiography with injection of contrast material either into the right heart or pulmonary artery was performed in 6 of these 7 cases. We believe that angiocardiography is the easiest and best method for visualizing the aortic arch and its branches in infants. In 2 infants, right countercurrent brachial aortography was performed, and in the adult patient catheter aortography was utilized to study the aortic arch.

While retrograde countercurrent brachial aortography may give a high degree of opacification of a portion of the aorta in infants, frequently all of the brachiocephalic arteries are not visualized. It is to be emphasized that if a retrograde injection is to be made, the left arm rather than the right should be used. In our patients with double aortic arch with left arch atresia, in whom retrograde injection of opaque material from the right brachial artery was performed, there was optimum visualization of the right common carotid and right subclavian arteries with only faint opacification of the proximal left arch. Because of the more proximal origin of the patent left

Table I

ROENTGENOLOGIC FEATURES OF DOUBLE AORTIC ARCH

	Chest Roentgenogram	Esophagogram	Angiogram
Functioning double aortic arch	Often negative in infants	Bilateral and posterior compression	Both arches opacify
Double aortic arch with left arch atresia *Subtype 1*	Usually right arch and right descending aorta	Bilateral and posterior compression	Right arch, right descending aorta, and aortic diverticulum
Right arch with mirror-image branching and left ductus connected to left subclavian artery	Right arch	No vascular ring	Right arch
Double aortic arch with left arch atresia *Subtype 2*	Usually right arch and right descending aorta	Bilateral and posterior compression	Right arch, right descending aorta, and aortic diverticulum
Right arch with mirror-image branching and left ductus connected to descending aorta	Right arch	Bilateral and posterior compression	Right arch and aortic diverticulum
Double aortic arch with left arch atresia *Subtype 3*	Right arch	Bilateral and posterior compression	Right arch, left subclavian artery arising from aortic diverticulum
Right arch and aberrant left subclavian artery and left ductus	Right arch	Bilateral and posterior compression	Right arch, left subclavian artery arising from aortic diverticulum

arch from which the left subclavian artery arises, retrograde injection of the left brachial artery would permit a more detailed visualization of the 2 aortic arches. Also, left retrograde brachial aortography would be helpful in identifying the origin of the left subclavian artery in either right aortic arch with an aberrant left subclavian vessel or double aortic arch Subtype 3.

Biplane anteroposterior and lateral roentgenograms were obtained in most cases. In a few instances, both anterior oblique projections were obtained. The anteroposterior study was the most reliable and provided the most information. The lateral view was only of value in showing the aortic diverticulum of the upper descending aorta. However, this structure was best visualized on the right anterior oblique study.

SUMMARY

1. Double aortic arch can be divided into 2 types, depending upon the patency of the 2 arches. In 1 type, both arches are patent and functioning. In the other type, 1 arch, usually the left, is atretic.

2. The embryologic, anatomic and angiographic features of double aortic arch

with left arch atresia are discussed.

3. The angiographic similarities of double aortic arch with left arch atresia to the various right arch malformations are illustrated.

4. In the presence of a symptomatic vascular ring, when the angiographic findings appear to be those of a right aortic arch with mirror-image branching, the most likely diagnosis is double aortic arch with left arch atresia.

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REFERENCES

1. ARKIN, A. Double aortic arch with total persistence of right and isthmus stenosis of left arch: new clinical and x-ray picture; report of six cases in adults. *Am. Heart J.*, 1936, *11*, 444–474.
2. EDWARDS, J. E. Anomalies of derivatives of aortic arch system. *M. Clin. North America*, 1948, *32*, 925–949.
3. GRISWOLD, H. E., JR., and YOUNG, M. D. Double aortic arch: report of two cases and review of literature. *Pediatrics*, 1949, *4*, 751–768.
4. GROSS, R. E. Arterial malformations which cause compression of trachea or esophagus. *Circulation*, 1955, *11*, 124–134.
5. HOLLMAN, G. L., COOLEY, D. A., and BLOODWELL, R. D. Congenital vascular ring. *S. Clin. North America*, 1966, *46*, 885–892.
6. HURLEY, L. E., and COATES, A. E. Case of right-sided aortic arch and persistent left superior vena cava. *J. Anat.*, 1927, *61*, 333–339.
7. ISSAJEW, P. O. Der doppelte Aortenbogen. *Anat. Anz.*, 1931, *73*, 153–158.
8. KIRKLIN, J. W., and CLAGETT, O. T. Vascular rings producing respiratory obstruction in infants. *Proc. Staff Meet. Mayo Clin.*, 1950, *25*, 360–367.
9. NEUHAUSER, E. Roentgen diagnosis of double aortic arch and other anomalies of great vessels. AM. J. ROENTGENOL. & RAD. THERAPY, 1946, *56*, 1–12.
10. RIKER, W. L. Anomalies of aortic arch and their treatment. *Pediat. Clin. North America*, 1954, 181–195.
11. SHUFORD, W. H., SYBERS, R. G., and EDWARDS, F. K. Three types of right aortic arch. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *109*, 67–74.
12. STEWART, J. R., KINCAID, O. W., and EDWARDS, J. E. An Atlas of Vascular Rings and Related Malformations of the Aortic Arch System. Charles C Thomas, Publisher, Springfield, Ill., 1964, pp. 14–37.
13. SYMBAS, P. N., SHUFORD, W. H., EDWARDS, F. K., and SEHDEVA, J. S. Vascular ring: persistent right aortic arch, patent proximal left arch, obliterated distal left arch and left ligamentum arteriosum. To be published.
14. WHEELER, P. C., and KEATS, T. E. Left aortic diverticulum as component of constricting vascular ring: report of two neonatal cases. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1963, *89*, 989–994.

COEXISTENT PULMONARY ARTERY AND AORTIC ARCH CALCIFICATION*

ITS SIGNIFICANCE AND ASSOCIATION WITH PATENT DUCTUS ARTERIOSUS

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ISOLATED calcification of the pulmonary artery[5,9,16,25] or the aortic arch[2,8,11,16,21,26] may be associated with several entities and pathologic processes (*vide infra*).

The purpose of this paper is to emphasize the fact that when they occur simultaneously, particularly in young people, the diagnosis of patent ductus arteriosus should be strongly considered. The following case reports illustrate this association.

REPORT OF CASES

Case I. A.B., a 41 year old female with a 1 year history of exertional dyspnea, had known of a heart murmur since childhood.

Physical examination revealed the apical impulse to be in the fifth intercostal space, 2 fingerbreadths outside the mid-clavicular line. P_2 was loud. At the second and third left intercostal spaces, a typical machinery murmur was audible. A hollow systolic blowing murmur was also audible at the apex.

Electrocardiogram revealed left ventricular hypertrophy.

Chest roentgenograms disclosed the presence of calcification in the aortic arch, as well as calcification in the pulmonary artery extending to the region of the patent ductus (Fig. 1). Cardiac fluoroscopy demonstrated evidence of left ventricular enlargement, left atrial enlargement and a prominent pulmonary artery segment. A right heart catheterization was performed which revealed the presence of a large left-to-right shunt at the pulmonary artery level. In addition, a prominent "V" wave was found in the wedge pressure.

Selective pulmonary angiography showed the pulmonary artery to be markedly enlarged and the pulmonary vasculature to be increased. The left atrium was enlarged and the left ventricle was dilated and hypertrophied. The aorta was dilated. Following aortic opacification, there was re-opacification of the pulmonary artery. These findings were interpreted as being compatible with a large left-to-right shunt at the pulmonary artery level. Retrograde aortography confirmed the presence of a left-to-right shunt through a patent ductus.

At surgery, in addition to a patent ductus arteriosus which was ligated, the mitral annulus was found to be dilated. A calcific plaque was palpated at the ductus.

Case II. L.T., an asymptomatic 26 year old female, had a history of a heart murmur since childhood.

Physical examination revealed the apical impulse to be diffuse in the sixth intercostal space at the anterior axillary line. A typical machinery murmur was audible at the third left intercostal space.

Electrocardiogram showed left ventricular hypertrophy.

Roentgenograms of the chest demonstrated the presence of a curvilinear calcification in the region of the pulmonary artery extending to the aortic arch (Fig. 2).

Cardiac catheterization and selective angiocardiography confirmed the presence of patent ductus arteriosus with left-to-right shunting.

Ligation of the ductus was performed.

Case III. J.C., a 43 year old male, had a 5 year history of progressive dyspnea, cyanosis and clubbing.

Physical examination revealed a cyanotic male. A left paraspinal heave was present. P_2 was loud and high in pitch. A soft ejection murmur was audible at the pulmonic area.

Electrocardiogram showed right ventricular hypertrophy.

Roentgenograms of the chest disclosed ex-

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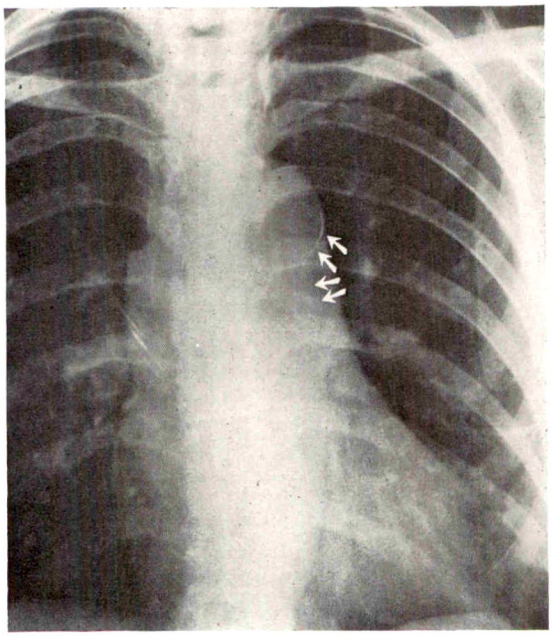


FIG. 1. Case I. A 41 year old female with calcified aortic arch. Also note curvilinear pulmonary artery calcification (arrows) extending to the region of the ductus arteriosus.

tensive calcification of the pulmonary artery, aortic arch and region of the patent ductus (Fig. 3, *A* and *B*).

Cardiac catheterization, angiography and dye-dilution studies were interpreted to indicate right-to-left shunting through a patent ductus arteriosus.

CASE IV. G.D., a 56 year old female, had a history of heart murmur since infancy. She had developed poliomyelitis at the age of 8 years, followed by kyphoscoliosis. Recurrent episodes of hemoptysis had started at age 12 years. At 50 years of age, she developed congestive heart failure. Digitalis and diuretics were prescribed. At the age of 55 years, she was noted to be cyanotic. During a previous hospitalization, she was also noted to be polycythemic.

On physical examination, the patient was cyanotic with slight clubbing. P_2 was loud and an atrial gallop was audible: a decrescendo diastolic murmur was audible along the left sternal border. Kyphoscoliosis was also noted as was bilateral foot-drop, secondary to her previous poliomyelitis.

Electrocardiogram revealed right ventricular hypertrophy.

Roentgenograms of the chest (Fig. 4) disclosed extensive pulmonary artery and aortic arch calcifications as well as cardiomegaly.

At the time of cardiac catheterization, the catheter repeatedly crossed a patent ductus arteriosus and entered the aorta from the pulmonary artery. There was equilibration of pressures in the pulmonary artery and in the aorta. Arterial oxygen saturation was 77 per cent.

Angiocardiography was performed with injection into the right ventricle. The angiocardiogram was interpreted as revealing a bidirectional shunt, predominantly right-to-left, at the level of the patent ductus.

CASE V. L.M., a 54 year old female, was first told of a heart murmur at the age of 27 years, when congestive heart failure was first noted. She was placed on digitalis, but continued to complain of exertional dyspnea, orthopnea and intermittent edema. Despite these symptoms, however, she managed to carry 9 pregnancies to full-term delivery without complications.

Physical examination revealed the apical impulse to be in the sixth intercostal space, 2 cm. outside the midclavicular line. A left parasternal thrill was palpated. A typical machinery murmur was heard at the pulmonic area.

Electrocardiogram revealed left ventricular hypertrophy.

Roentgenograms of the chest disclosed the

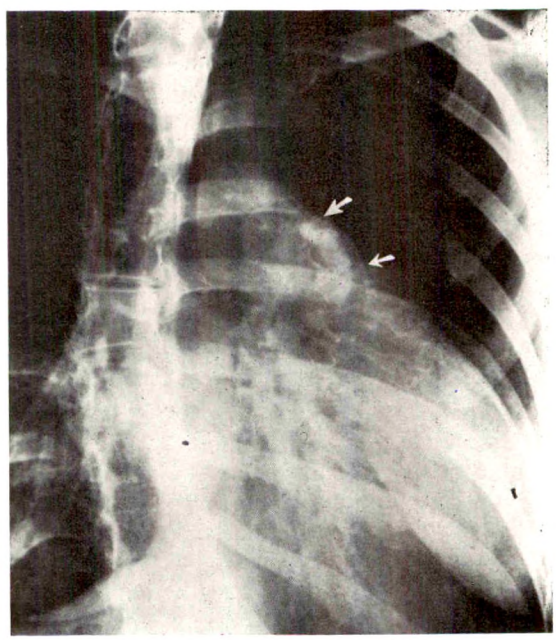


FIG. 2. Case II. Right anterior oblique Bucky view of the chest. Note curvilinear pulmonary artery calcification (arrows) extending to the aortic arch.

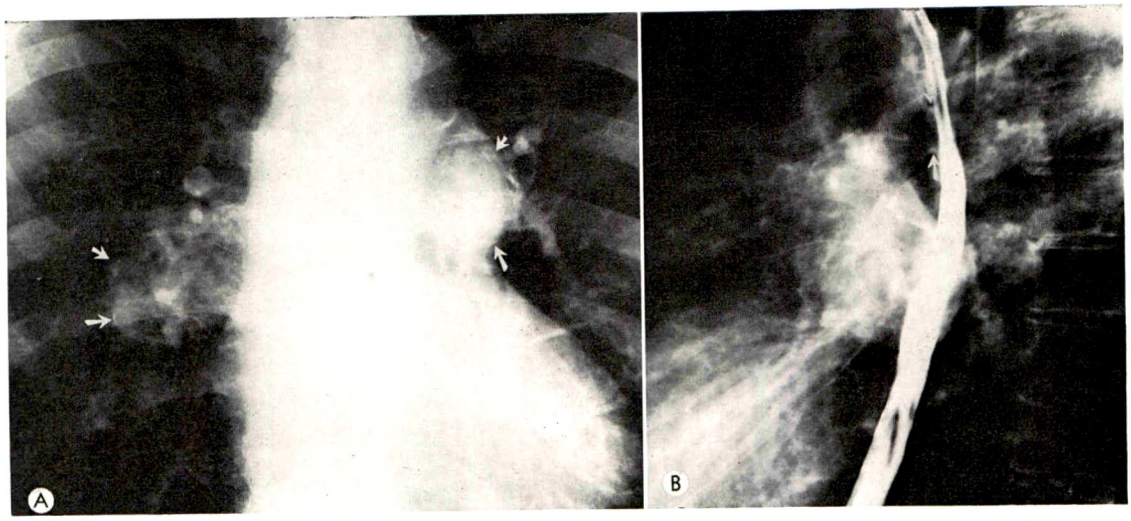


Fig. 3. Case III. (*A*) Frontal Bucky view of the chest. Note extensive calcification of the pulmonary artery and its branches (arrows) and aortic arch. (*B*) Esophagogram: lateral view. Note calcification in the region of the ductus arteriosus (arrows).

presence of cardiomegaly and markedly prominent pulmonary arteries. In addition, ringlike calcifications were noted in the pulmonary artery extending to the region of the ductus arteriosus and the aortic arch (Fig. 5).

Cardiac catheterization revealed left-to-right shunting at the level of the pulmonary artery. Angiocardiography confirmed left-to-right shunting at the level of the pulmonary artery.

The patient was operated upon and a patent ductus was ligated.

PULMONARY ARTERY CALCIFICATION

Timpanelli and Steinberg in 1961[25] described pulmonary artery calcification in 4 cases with patent ductus arteriosus with reverse blood flow, and noted that roentgen evidence of pulmonary artery calcification was very suggestive of this condition. However, in a later article, Steinberg[23] reported 3 cases of pulmonary artery calcification

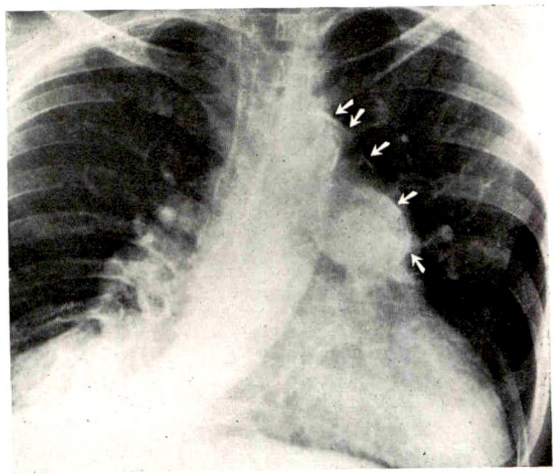


Fig. 4. Case IV. Curvilinear calcifications are seen in the pulmonary artery (arrows) and aortic arch.

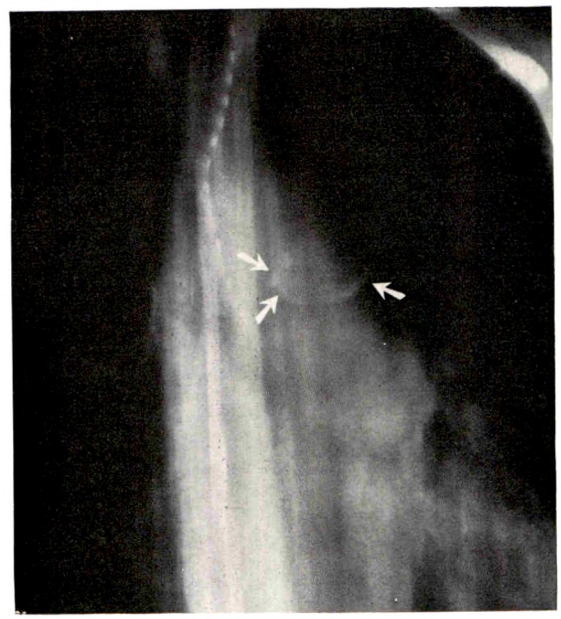


Fig. 5. Case V. Frontal tomogram. Note ring-like calcification in the pulmonary artery (arrows). The aortic arch is also calcified.

associated with Eisenmenger's syndrome without patent ductus arteriosus. Calcification of the pulmonary artery in Eisenmenger's syndrome was also reported by Goldfischer[14] and Mallamo *et al.*[17] Furthermore, calcification of the pulmonary artery can be seen, although rarely, in association with atheromatous disease of this vessel,[21] chronic pulmonary emphysema,[22] calcified thrombus[18] or metastatic tumor,[27] stenotic pulmonary valve,[10] chronic pulmonary schistosomiasis[12] and pulmonary artery aneurysm.[3]

AORTIC ARCH CALCIFICATION

Roentgenographic evidence of calcification of the aortic arch in young individuals before the fourth decade, particularly females, may be indicative of the presence of patent ductus arteriosus.[2,8,21,26] According to Dalith,[8] calcification of the aortic arch originates from single plaques in the aortic wall located at the entrance of the ligamentum arteriosum and at the origin of the left subclavian arteries. However, aortic arch calcification, even in young people, may be seen in conditions other than patent ductus arteriosus. These include traumatic aneurysm of the thoracic aorta,[19] aortic coarctation,[13,21] syphilitic aortitis, atheromatous occlusive disease and Takayashu's disease[1,24] and rheumatoid arthritis.[20]

PATENT DUCTUS CALCIFICATION

Calcification of an obliterated ductus arteriosus is not an infrequent occurrence in children.[6] However, calcification restricted solely to a patent ductus arteriosus itself is a very unusual finding.[21] Graham[15] described a case of a large calcified aneurysm of the ductus. Of 57 autopsied cases of patent ductus in adults, Keys and Shapiro[16] found only 1 instance of calcification restricted to the duct itself. The aortic end of the ductus was calcified in 3, the pulmonary artery was calcified in 1, and combined aortic and pulmonary artery calcification was seen in 1 case. In our Case III (Fig. 3, *A* and *B*), the roentgenograms suggest the presence of calcification in a patent ductus arteriosus.

DISCUSSION

The 5 currently reported cases illustrate the association between combined calcification of the pulmonary artery and aortic arch with patent ductus arteriosus (Fig. 1–5). The authors reviewed the published roentgenograms and descriptions of numerous articles in the last 3 decades reporting calcification of either pulmonary artery[3,5,10,12,14,17,18,22,23,25,27] or aortic arch in young people.[13,19–21,24] None of these reported cases showed combined pulmonary artery and aortic arch calcification, except in the presence of patent ductus arteriosus.[4,5,7,9,23,25] Therefore, it appears that roentgen evidence of combined pulmonary artery and aortic arch calcifications is most suggestive of the presence of patent ductus arteriosus.

There have been several reports emphasizing that pulmonary artery calcification may be indicative of reversal of blood flow in patent ductus arteriosus or congenital heart disease.[17,23,25] Several examples of pulmonary artery calcification and altered hemodynamics in patent ductus arteriosus were also illustrated in articles by Dammann *et al.*,[9] Ruskin and Samuel,[21] Cosh,[5] and Castleman and Kibbee.[4] This was also noted in our Cases III and IV. However, in our Cases I, II and V, only a left-to-right shunt was demonstrated through the patent ductus.

SUMMARY

Isolated calcification of either the pulmonary artery or aortic arch, particularly in the young, occurs in a small percentage of cases of patent ductus arteriosus.

It also occurs in a variety of other conditions; however, combined calcification of the pulmonary artery and aortic arch is highly suggestive of a patent ductus with or without reversal of flow.

Five illustrative cases are presented and the differential diagnosis is discussed.

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REFERENCES

1. Basu, A. K. Occlusive disease of aorta and its main branches. *Brit. J. Surg.*, 1961–1962, *49*, 148–156.
2. Blankenhorn, D. H. Relation of age and sex to diffuse aortic calcification in man. *J. Gerontol.*, 1964, *19*, 72–77.
3. Boyd, L. J., and McGavack, T. H. Aneurysm of pulmonary artery: review of literature and report of two new cases. *Am. Heart J.*, 1939, *18*, 562–578.
4. Castleman, B., and Kibbee, B. U. Editors. Case #46081. Case records of the Massachusetts General Hospital, 1960, *262*, 407–413.
5. Cosh, J. A. Patent ductus arteriosus with pulmonary hypertension. *Brit. Heart J.*, 1953, *15*, 423–329.
6. Currarino, G., and Jackson, J. H., Jr. Calcification of ductus arteriosus and ligamentum Botalli. *Radiology*, 1970, *94*, 139–142.
7. Dailey, F. H., Genovese, P. D., and Behnke, R. H. Patent ductus arteriosus with reversal of flow in adults. *Ann. Int. Med.*, 1962, *56*, 865–882.
8. Dalith, F. Calcification of aortic knob: its relation to fifth and sixth embryonic aortic arches; radiological and anatomical study. *Radiology*, 1961, *76*, 213–222.
9. Dammann, J. F., Jr., Berthrong, M., and Bing, R. J. Reverse ductus: presentation of syndrome of patency of ductus arteriosus with pulmonary hypertension and shunting of blood flow from pulmonary artery to aorta. *Bull. Johns Hopkins Hosp.*, 1953, *28*, 128–149.
10. Dinsmore, R. E., Sanders, C. A., Harthorne, J. W., and Austin, W. G. Calcification of congenitally stenotic pulmonary valve. *New England J. Med.*, 1966, *275*, 99–100.
11. Dominguez, P., Roitman, M., and Alzamora, R. Calcificación del infundibulum aortico en la persistencia del conducto arterioso. *An. Fac. Med. Lima*, 1960, *43*, 651–664.
12. Farid, Z., Greer, J. W., Ishak, K. G., Nagah, A. M., LeGolvan, P. C., and Mousa, A. H. Chronic pulmonary schistosomiasis. *Am. Rev. Tuberc. & Pulmon. Dis.*, 1950, *79*, 119–133.
13. Flynn, P. J., and Kattus, A. A. Coarctation of aorta with proximal aortic dilatation and calcific atheromatous degeneration corrected by endarterectomy. *J. Thoracic & Cardiovasc. Surg.*, 1959, *38*, 369–373.
14. Goldfischer, S. Abscess of brain in patient with ventricular septal defect: reversal of blood flow and calcification of pulmonary artery. *J.A.M.A.*, 1963, *184*, 896–898.
15. Graham, E. A. Aneurysm of ductus arteriosus, with consideration of its importance to thoracic surgeon. *Arch. Surg.*, 1940, *41*, 324–333.
16. Keys, A., and Shapiro, M. J. Patency of ductus arteriosus in adults. *Am. Heart J.*, 1943, *25*, 158–186.
17. Mallamo, J. T., Baum, R. S., and Simon, A. L. Diffuse pulmonary artery calcifications in case of Eisenmenger's syndrome. *Radiology*, 1971, *99*, 549–550.
18. McAlister, W. H., and Blatt, E. Calcified pulmonary artery thrombus. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1962, *87*, 908–910.
19. Meyer, J. E. Calcification in aortic arch of young man. *J.A.M.A.*, 1970, *214*, 545–546.
20. Munasinghe, D. R., and Rajasuriya, K. Calcification of aorta and main vessels in rheumatoid arthritis. *Am. Heart J.*, 1966, *71*, 813–817.
21. Ruskin, H., and Samuel, E. Calcification in patent ductus arteriosus. *Brit. J. Radiol.*, 1950, *23*, 710–717.
22. Salzman, E. Lung Calcification in X-ray Diagnosis. Charles C Thomas, Publisher, Springfield, Ill., 1968, pp. 92–99.
23. Steinberg, I. Calcification of pulmonary artery and enlargement of right ventricle: sign of congenital heart disease; Eisenmenger syndrome—pulmonary hypertension, increased pulmonary resistance, and reversal of blood flow. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1966, *98*, 369–377.
24. Steinberg, I. Calcified and dilated ascending aorta due to atheromatous occlusive disease simulating coarctation of aorta: report of case and theory of pathogenesis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1966, *98*, 840–843.
25. Timpanelli, A. E., and Steinberg, I. Calcification of pulmonary artery in patent ductus arteriosus with reversal of blood flow. *Am. J. Med.*, 1961, *30*, 405–417.
26. Weiss, E. Calcified plaque of aorta at entrance of patent ductus arteriosus: point in diagnosis. *Am. Heart J.*, 1931, *7*, 114–115.
27. Woo-Ming, M., Murray, S. M., Path, M. C., and Bateson, E. M. Calcified tumour-thrombus of renal carcinoma. *Brit. J. Radiol.*, 1966, *39*, 267–271.

ANGIOGRAPHIC DIAGNOSIS OF DISSECTING ANEURYSM OF THE AORTA*

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DISSECTING aortic aneurysms are related to degenerative disease of the media of the aorta. The cause of the initial rupture or tear in the intima and media of the aorta is not well understood. Forceful penetration of blood through the tear into the aortic wall results in dissection of the layers of the aorta and the development of a "false" channel. The usual location of the false channel is between the inner two-thirds and outer one-third of the media. Dissection may progress to involve part or all of the circumference of the aorta and extend for a variable distance along the length of the aorta. Blood within the false channel may either re-enter the true aortic lumen or may perforate the aortic wall externally.

Although dissecting aortic aneurysms have been reported in apparently healthy individuals without known precipitating factors, this disease entity is most frequently encountered in patients with hypertension and cystic medial necrosis.

DeBakey has divided aortic dissections into 3 types and his classification is useful in defining the origin and extent of dissections (Fig. 1). In Type 1, the dissection involves the entire aorta, originating in the ascending aorta near the aortic valve and extending to the external iliac arteries (Fig. 2, *A–D*; 3, *A–C*; and 4, *A–E*). In Type 2, the dissection is limited to the ascending aorta; this is commonly seen in Marfan's syndrome (Fig. 6). In Type 3 (Fig. 5, *A–C*) the dissection originates distal to the left subclavian artery and extends a variable length of the descending aorta, involving only distal aortic branches. In Types 1 and 2, the process originates within the first 5 cm. of the ascending aorta and may involve the coronary arteries as well as the major vessels originating from the aortic arch.

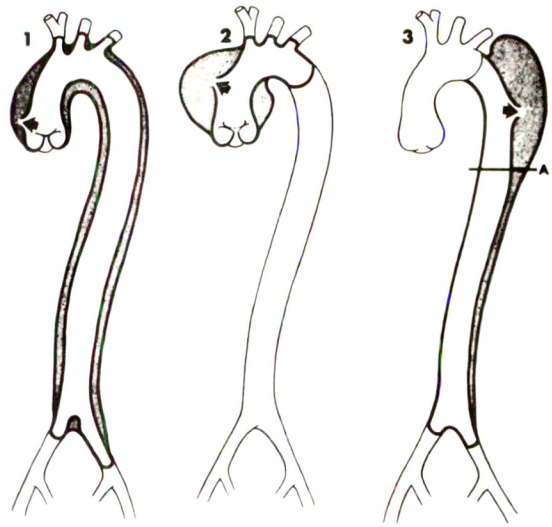


FIG. 1. Anatomic classification of dissecting aneurysm following DeBakey *et al.*[2] In Type 1 the dissection involves the ascending aorta, the aortic arch and extends to the descending aorta. In Type 2, the dissection is confined to the ascending aorta. Types 1 and 2 correspond to Group A of Daily *et al.*[1] In Type 3 the dissection originates distal to the left subclavian artery and the ascending aorta is not involved (Group B of Daily *et al.*[1]).

The clinical course, surgical therapy and prognosis of either Type 1 or 2 dissections differ greatly from those of the Type 3 variety, where the dissection does not involve the aorta proximal to the origin of the left subclavian artery.[1]

Clinically, acute dissections are usually characterized by the sudden onset of excruciating pain in the chest. The early mortality rate is high. DeBakey and colleagues[2] estimate that 25 per cent of patients survive for 2 weeks and only 10 per cent survive more than 6 months. Although the diagnosis of dissecting aortic aneurysm is suggested by the history and associated clinical findings, it is established with cer-

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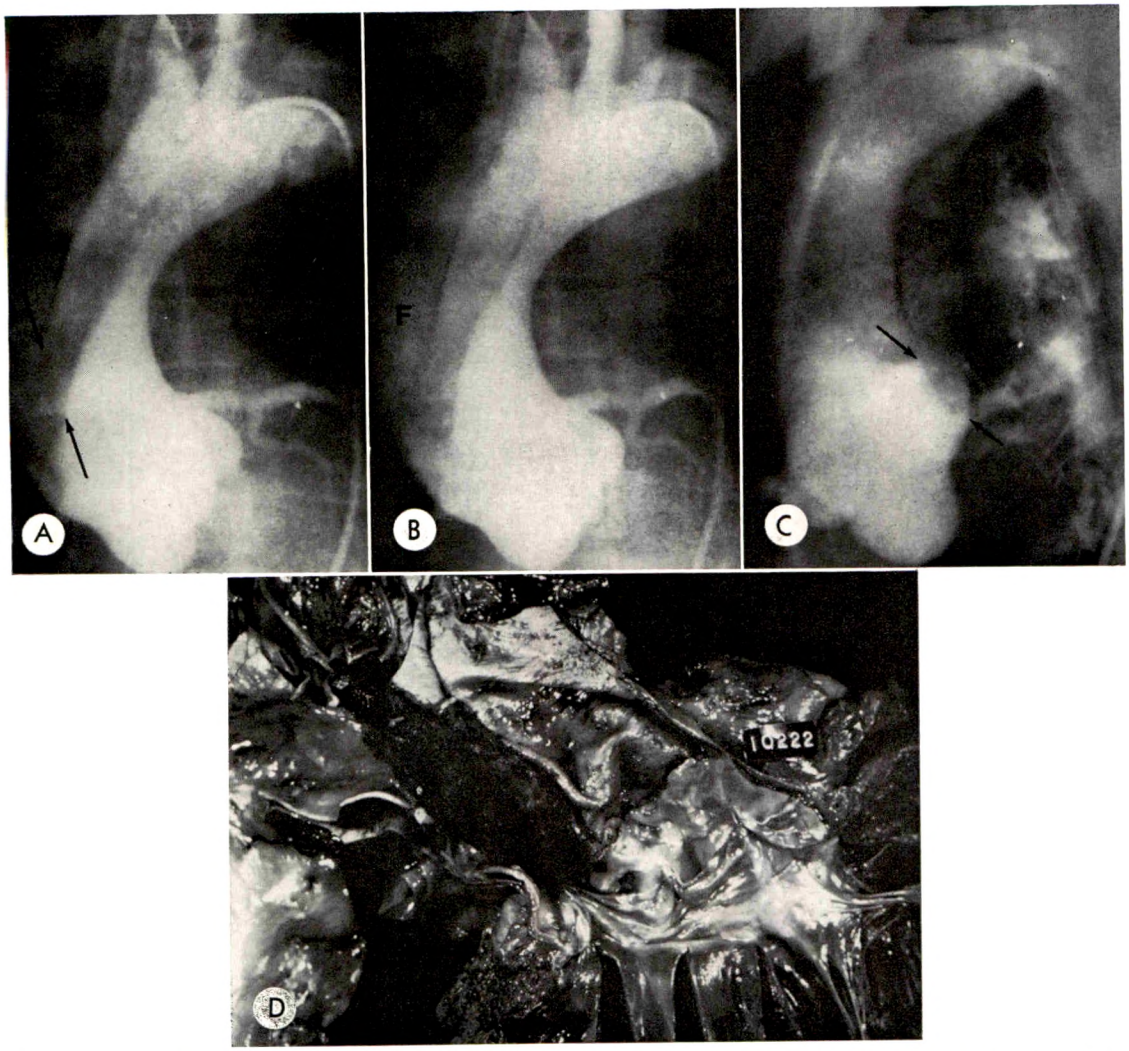


Fig. 2. (*A* and *B*) Anteroposterior aortograms showing a dissecting aneurysm, Type 1, with occlusion of the innominate artery. The catheter was introduced by Seldinger's technique into the true channel. The true channel is deformed on the right border with the proximal tear seen as a negative shadow between 2 arrows. Two seconds later in *B* the false channel "F" is also opacified, indicating the 2 directions of the blood flow. The upper arrow points to the occlusion of the innominate artery. (*C*) On the lateral aortogram the false channel "F" is seen posteriorly overlapping the true channel, and extending towards the aortic arch. (*D*) The oblique tear just above the aortic valve is demonstrated on the anatomic specimen. The true aortic lumen has been displaced to the left and the false channel partially filled with dark thrombus appears to the right on the specimen.

tainty only by aortography. Additional information afforded by aortography includes accurate definition of the extent of the lesion and elucidates important anatomic details regarding therapy.

METHOD OF STUDY

The initial aortic angiogram should be obtained following injection of the contrast medium into the ascending aorta in proximity of the sinuses of Valsalva. This first injection permits analysis of the morphology of the aortic sinuses, ascending aorta and aortic arch for delineation of the site of rupture as well as assessment of the degree of aortic insufficiency when present. A sec-

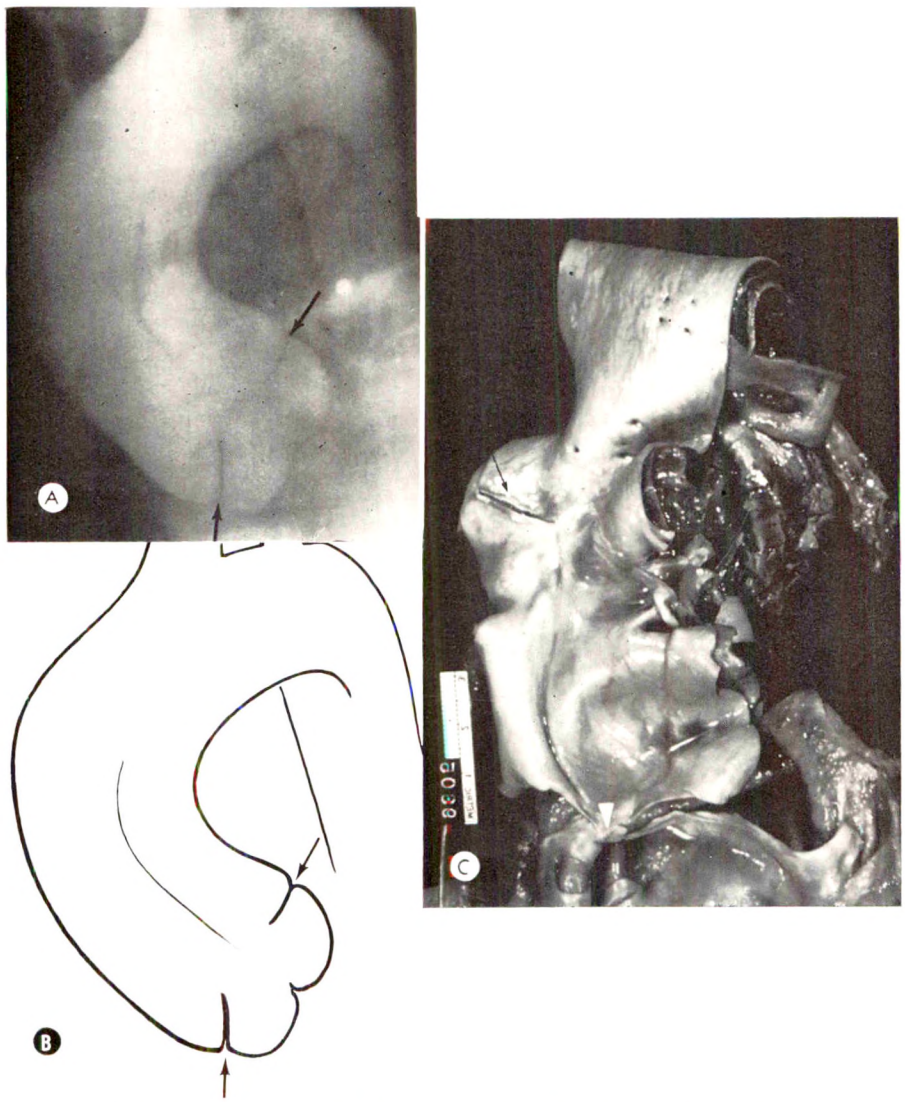


FIG. 3. (*A*) Aortogram, (*B*) diagrammatic sketch, and (*C*) anatomic specimen of a dissecting aneurysm, Type 2. The intimal tear appears as a horizontal or oblique radiolucent line immediately above the sinuses of Valsalva in the aortogram. It corresponds very neatly with the circumferential tear shown in the anatomic specimen (white marker), which is almost complete. The dissection continued past the level of a minor re-entry site (arrow on left of picture), and included the descending thoracic aorta beyond several intercostal arteries (upper border of picture).

ond injection of contrast material into the descending aorta is required for definition of the extent of the dissection and allows visualization of all distal aortic branches.

The catheter may be introduced into the aorta percutaneously from the femoral artery by the Seldinger technique[8] or from the arm via a brachial arteriotomy. In the majority of patients, the Seldinger approach is employed. However, in obese patients with very dilated aortas, it is sometimes difficult to obtain adequate concentration of contrast medium in the ascending aorta. In such individuals, passage of a short (80 cm.) catheter with a large internal diameter through a brachial arteriotomy allows one to obtain aortograms of high quality.

Biplane films in anteroposterior and lateral projections are exposed at a rate of 6 films per second for the first 2 seconds and 2 per second for the subsequent 5 seconds following injection of 75 ml. of contrast material within 2.5 seconds delivered by a power injector. An Elema Schönander biplane roll film changer and 2 M.S.I. General Electric x-ray generators are utilized in our laboratory. The exposure factors are: 1,200 ma. in each projection (anteroposterior and lateral); 85 to 100 kv.; and 15–30 msec. Careful collimation of secondary radiation, considering the size of the individual patient, is most important and cross grids, 8:1, covered with one sheet of lead foil 1/5,000 inch thick laminated in plastic, are utilized.

INDICATIONS

To confirm or exclude the diagnosis of aortic dissecting aneurysm, an aortogram is indicated in all suspected cases except in those patients who are unable to tolerate the examination. The procedure should be done as soon as possible, yet it should not be considered an emergency procedure. There are no data indicating that aortography *per se* results in a deterioration of the patient's clinical condition. However, in Types 1 and 2, when shock, hemopericardium, or hemothorax is present, aortography is contraindicated.

VERIFICATION OF CATHETER POSITION

Knowledge of the exact position of the catheter within the aorta prior to the injection of the contrast medium is critical. To verify the location of the catheter within the true aortic lumen, the following criteria are used in our laboratory (Table 1):

(a) Displacement of the catheter during cardiac injection is wide when it is in the true lumen, and absent or substantially reduced when the catheter has been placed in the false channel.

(b) Aortic blood pressure is recorded when the tip of the catheter is in the true lumen, whereas the pressure pulse is diminished or absent when the catheter is in the false channel.

(c) The injection of a small amount of contrast material by hand is helpful in verifying the position of the catheter. When the catheter lies within the true aortic lumen, the contrast material briefly outlines the normal aortic morphology including the sinuses of Valsalva and disappears rapidly. On the contrary, if the contrast medium remains for several seconds, and the sinuses of Valsalva and normal aortic morphology are not apparent, the catheter is within the false channel.

When the catheter has inadvertently been introduced into the false channel, a low pressure hand injection of a small amount of contrast material may be made without complication and will be useful in determining the exact morphology and extent of the false channel.

Table 1

DISSECTING ANEURYSM
VERIFICATION OF THE CATHETER POSITION

	True Channel	False Channel
Displacement During Cardiac Cycle	Wide in Systole and Diastole	Absent or Very Lessened
Blood Pressure	Systemic	Less Than Systemic
Blood Flow	Normal	Very Slow or Absent
Test Hand Injection of Contrast Medium	Contrast Medium Rapidly Washed Out	Contrast Medium Remains Several Seconds
Visualization of Sinus of Valsalva	Outlined With Normal Morphology	Not Outlined

ANGIOGRAPHIC APPEARANCE

The major goals of angiographic examination are determination of: (1) the location of the initial tear; (2) the extension of the dissection or false channel; (3) the deformation of the true channel; (4) the de-

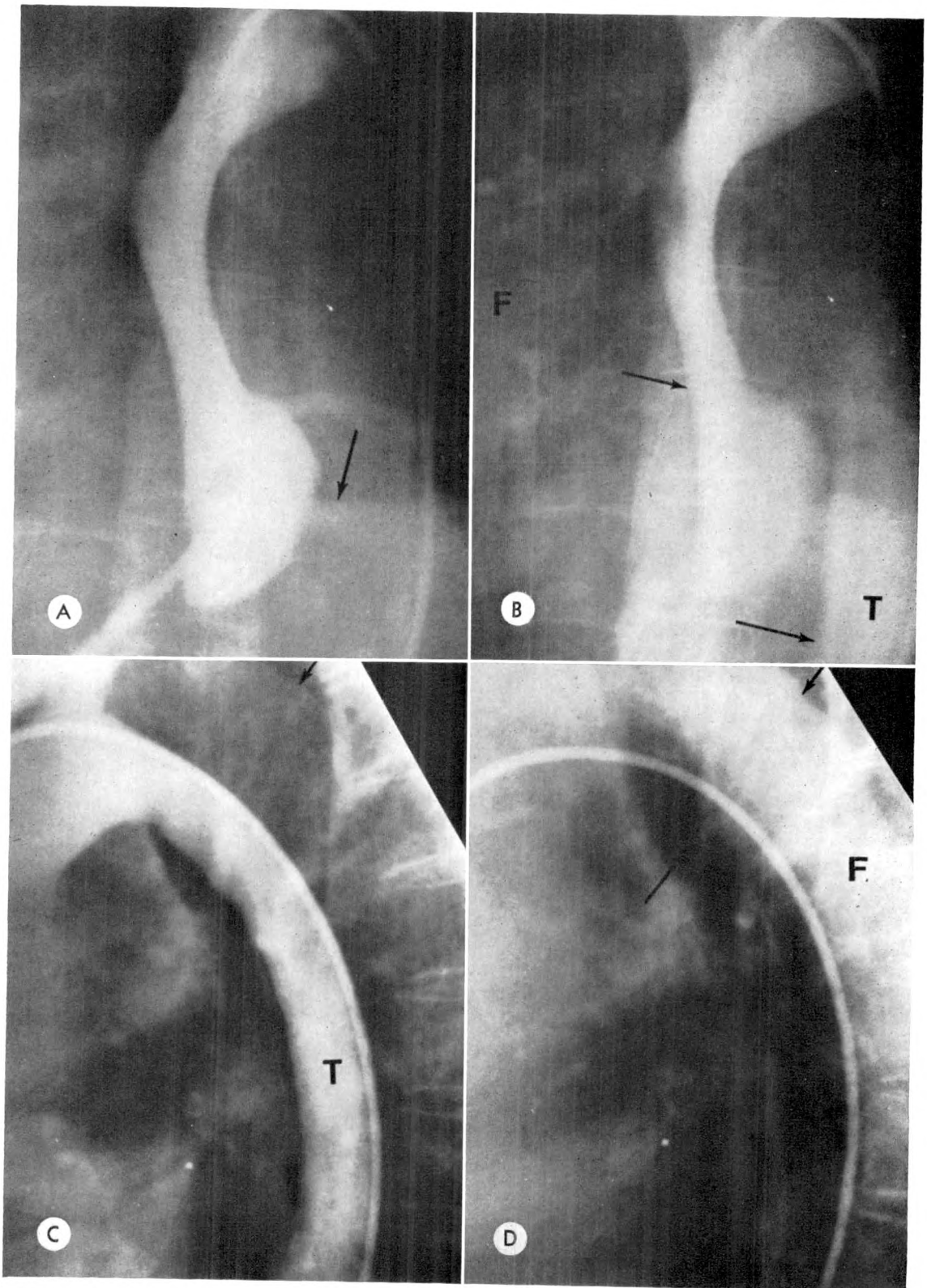


FIG. 4. (*A* and *B*) Anteroposterior aortograms made by the percutaneous Seldinger technique of a patient with a dissecting aneurysm, Type 1. The true channel of the ascending aorta is narrowed and deformed

gree of aortic insufficiency; and (5) the involvement of the aortic branches.

1. *The site of the rupture.* The usual location of the proximal intimal tear is in the ascending aorta above the coronary ostia within the first 5 cm. of the aortic valve: in Type 3 dissections it is usually located immediately distal to the left subclavian artery origin (Fig. 5, *A–C*). Angiographically the intimal tear is seen as a transverse radiolucent line of variable size just above the sinus of Valsalva. The tear may be oblique in relation to the axis of the aorta (Fig. 2, *A–D*). When the tear is not directly visualized, irregularities of the border of the aortic contour may indicate their location. Stein and Steinberg[10] have described a shadow-like ulcer on the surface of the true channel representing the tear, but this particular sign has not been observed in our laboratory. In some carefully performed aortographic examinations, it may be possible to determine the direction of the blood flow through the tear (Fig. 2, *A–D*).

2. *The extension of the dissection or false channel.* A major objective of the angiographic study is to visualize the false channel; *i.e.*, to determine the extent of the dissection. Complete visualization of the false channel with the initial injection of contrast material is helpful. This may be difficult when blood flows from the true channel into the false channel and re-enters through a distal tear. A second injection into the descending aorta is helpful in such instances. Identification of the distal tear is important prior to attempted surgical repair.

3. *The deformation of the true channel.* Sometimes the false channel is partially or totally occluded by clotted blood and cannot be demonstrated angiographically. There are indirect signs, however, which permit analysis of the extent of the lesion, the deformation of the aortic contour and the width of the aortic wall. The ascending aorta usually is deformed posteriorly and from the right side (Fig. 2, *A–D*; and 4, *A–E*), whereas the descending aorta is deformed posteriorly and from the left (Fig. 4, *A–E*). The thickness of the aortic wall may be useful in the assessment of the depth of the dissection. Normally the distance between the contrast medium columns of the aortic lumen and the lateral margin of aortic shadow is 2 to 3 mm.; when the distance is increased and surpasses 4 or 5 mm., dissection is indicated.[9]

4. *The degree of aortic insufficiency.* Aortic insufficiency occurs frequently in Types 1 and 2 and is important because of its surgical implications. Anatomically, it results from a loss of support of the aortic sinuses with prolapse of the leaflets due to the dissection, as seen in the pathologic specimen (Fig. 4*E*). Angiographically, the morphology of the sinuses of Valsalva is altered in that they are dilated, deformed and lower than usual. Contrast medium refluxes into the left ventricle. The degree of aortic insufficiency is judged by the speed of opacification and the density of the contrast medium in the left ventricle in relation to the density in the ascending aorta.[6]

5. *The involvement of the aortic branches.* Extension into the aortic branches may be the cause of such serious complications as: neurologic deficit with involvement of the carotid arteries; myocardial infarction when the coronary arteries are occluded or compromised; and/or severe arterial hypertension if the dissection includes the renal arteries. Absence of opacification is a sign of occlusion of aortic branches. A conical deformation indicating the extent of the dissection into the artery (Fig. 2, *A–D*), may sometimes be noted at the origin of the arteries. It is important to demonstrate

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by the dissection, and minor aortic valvular incompetence is depicted by the arrow in *A*. In *B* the descending aortic true channel, "T" is visualized. (*C* and *D*) Sequential lateral aortograms demonstrate opacification of the true channel "T" anteriorly and the false channel "F" posteriorly.

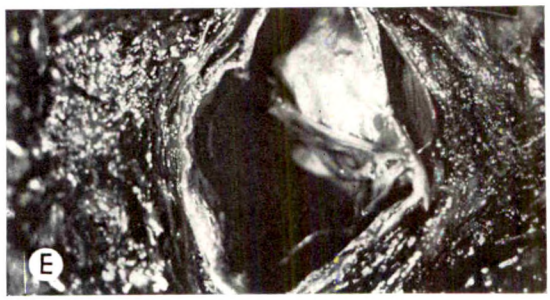


FIG. 4. (*E*) The specimen shows the relation of the dissection to the sinuses of Valsalva, and the resulting aortic valve deformity.

patency of the distal vessel prior to surgical intervention. These findings are well documented in the specimen illustrated in Figure 2*D*. Particular attention in excluding involvement of the coronary arteries, which may result in acute myocardial infarction, is warranted.

DISCUSSION

Dynamic changes can also be evaluated by the temporal sequence of the aortographic films with opacification of the true and false channels at different moments in time. Figure 4, *A–E*, shows the aortograms of a patient with dissecting aneurysm Type 1, with injection into the true channel and good opacification of the deformed ascending aorta, aortic arch and descending aorta. Several seconds later the contrast medium has been diluted in the true channel by admixture with blood. The false channel remains opacified with contrast material from the proximal part of the dissection located in the ascending aorta. This phenomenon is due to the different speed of blood flow in the true and false channels related probably to the small size of the proximal and distal tears.

The incidence of dissection in cystic medial necrosis is extremely high.[4] For the diagnosis of dissection in *cystic medial necrosis* a special angiographic approach is necessary. The usual appearance of the aorta in cystic medial necrosis is that of marked uniform, symmetric dilatation of the ascending segment involving the sinuses of Valsalva. The dilatation terminates abruptly a few centimeters proximal to, or at the origin of, the brachiocephalic trunk, and results in the classical chemical retort configuration of the aorta. The angiographic signs are different in appearance depending on the extent of the dissection. In those cases where the dissection is limited to the ascending aorta, the angiographic details

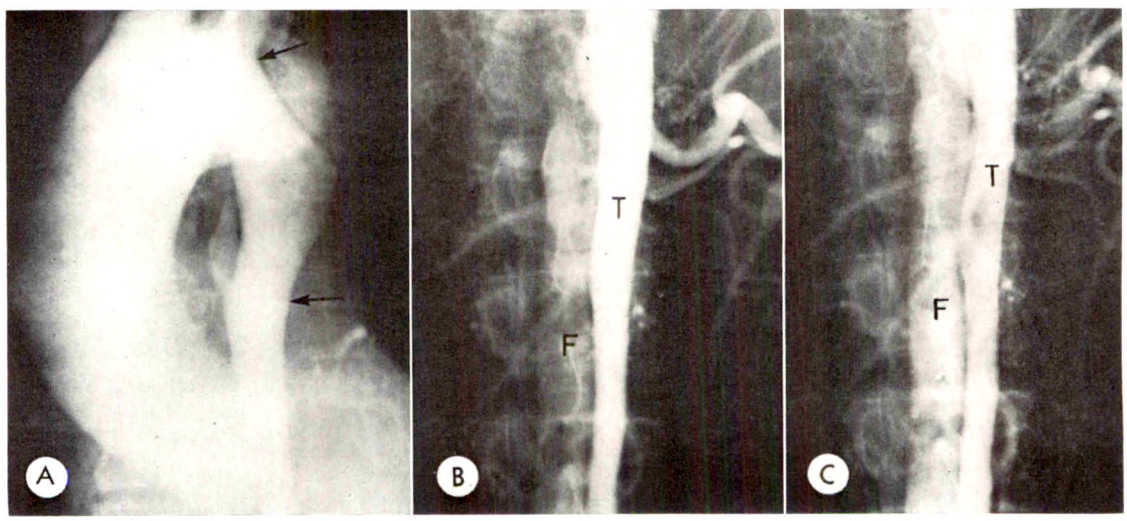


FIG. 5. (*A*) Anteroposterior aortogram showing a dissecting aneurysm, Type 3, beginning immediately distal to the left subclavian artery (upper arrow). The true channel to the right is considerably deformed and narrowed by a large false channel (lower arrow). (*B* and *C*) Subsequent views of the abdominal aorta show opacification of the true channel in *B* and both true and false channels in *C*. The right renal artery is occluded by the dissection.

are not easily identified. They are usually represented by thin radiolucent lines parallel to the aortic walls or diverging from the walls (Fig. 6). On the contrary, when the dissection extends beyond the ascending aorta, the angiographic signs are usually evident. The dilatation of the ascending aorta is asymmetric with distortion of its borders and of the sinuses of Valsalva with resultant aortic insufficiency. Some degree of dissection is thought to be present in every patient with moderate or severe aortic regurgitation.[4]

Although the roentgenologic signs of dissecting aortic aneurysm are usually evident, they are occasionally not readily apparent and misinterpretations may follow. These pitfalls in diagnosis have been well described by Shuford *et al.*[9] Simultaneous uniform opacification of both channels may simulate a normal aorta: however, a selective small injection in each channel can aid in the correct diagnosis. When the dissection is very localized, the lesion may not be visualized by the usual biplane angiograms and an additional injection in an oblique projection is required. With aortic insufficiency, the false channel may be simulated by a layering of contrast medium along the posterior wall of the descending aorta. Abnormally thick aortic walls in arteriosclerotic patients may suggest diagnosis of a dissection. Despite the existence of these pitfalls, diagnosis is possible when the examination is carefully performed. The diagnostic problems are reduced by obtaining good aortic opacification with high quality aortograms.

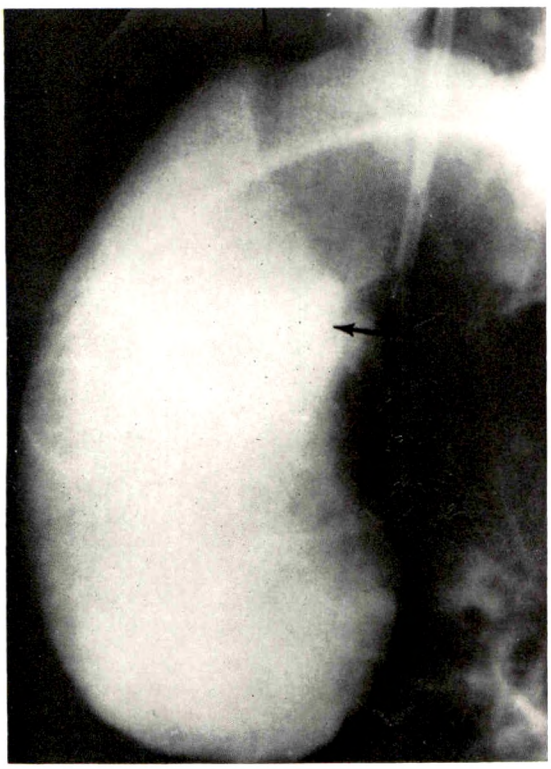


Fig. 6. Lateral view of an ascending aorta showing the characteristic deformity of cystic medial necrosis with a limited area of dissection, Type 2. The arrows point to the distal tear.

SUMMARY

Acute dissecting aortic aneurysm continues to be associated with a poor prognosis.

Angiographic study of aortic dissecting aneurysms is useful both in establishment of the diagnosis and in delineation of anatomic details important in management.

Aortography may be performed either by percutaneous Seldinger approach or through a brachial arteriotomy.

The importance of verifying the position of the catheter in the false or true channel, before injecting the contrast medium under high pressure, is emphasized.

Demonstration of the intimal tears, the extension of the dissection, the deformation of the true channel, the degree of aortic insufficiency and the degree of involvement of the aortic branches are the major goals of the angiographic examination.

The pitfalls in angiographic diagnosis, particularly in cases with cystic medial necrosis with dissection, are discussed.

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REFERENCES

1. Daily, P. O., Trueblood, H. W., Stinson, E. B., Wuerflein, R. D., and Shumway, N. E. Management of acute aortic dissections. *Ann. Thoracic Surg.*, 1970, *10*, 237–247.

2. DEBAKEY, M. E., HENLY, W. S., COOLEY, D. A., MORRIS, G. C., JR., CRAWFORD, E. S., and BEALL, A. C., JR. Surgical management of dissecting aneurysm involving ascending aorta. *J. Cardiovasc. Surg.*, 1964, *5*, 200–211.
3. DOW, J., ROEBUCK, E. J., and COLE, F. Dissecting aneurysms of aorta. *Brit. J. Radiol.*, 1966, *39*, 915–927.
4. EISEN, S., and ELLIOT, L. Roentgenology of cystic medial necrosis of ascending aorta. *Radiol. Clin. North America*, 1968, *6*, 437–449.
5. HIRST, A. E., JR., JOHNS, V. J., JR., and KIME, S. W., JR. Dissecting aneurysms of aorta: review of 505 cases. *Medicine*, 1958, *37*, 217–279.
6. HUNT, D., BAXLEY, W. A., BARCIA, A., KENNEDY, J. W., TERRENCE, P. J., WILLIAMS, J. E., and DODGE, H. T. The value of aortography on the evaluation of aortic insufficiency. Presented at 20th Annual Scientific Session: February 1971, 3–7.
7. KIRSCHNER, L. P., TWIGG, L., CONRAD, P. W., and HUFNAGEL, C. Retrograde catheter aortography in dissecting aortic aneurysms. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1968, *102*, 349–353.
8. SELDINGER, S. I. Catheter replacement of needle in percutaneous arteriography: new technique. *Acta radiol.*, 1953, *39*, 376–386.
9. SHUFORD, W. H., SYBERS, R. G., and WEENS, H. S. Problems in aortographic diagnosis of dissecting aneurysm of aorta. *New England J. Med.*, 1969, *280*, 225–231.
10. STEIN, H. L., and STEINBERG, I. Selective aortography: definitive technique for diagnosis of dissecting aneurysm of aorta. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1968, *102*, 333–348.
11. WHEAT, M. W., JR., HARRIE, P. D., MALM, J. R., KAISER, G., BOWMAN, F. O., JR., and PALMER, R. F. Acute dissecting aneurysms of aorta: treatment and results in 64 patients, *J. Thoracic & Cardiovasc. Surg.*, 1969, *58*, 344–351.

DYNAMICS OF BALLOONS IN INTRAAORTIC COUNTERPULSATION*

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IN HYPOTENSIVE states, counterpulsation with intraaortic balloons is sometimes ineffective for 2 reasons: (1) the aortic compliance $\Delta V/\Delta P$ increases with lowered arterial pressure resulting in a decrement of pulse pressure, if a fixed volume is given into the aorta; and (2) the decrease in the diameter of the aorta in low pressure states may lead to occlusion of the aorta by the balloon.

In aiming to optimize intraaortic balloon pulsation, cineangiographic investigations in models and in animal experiments were performed to obtain knowledge on the in-

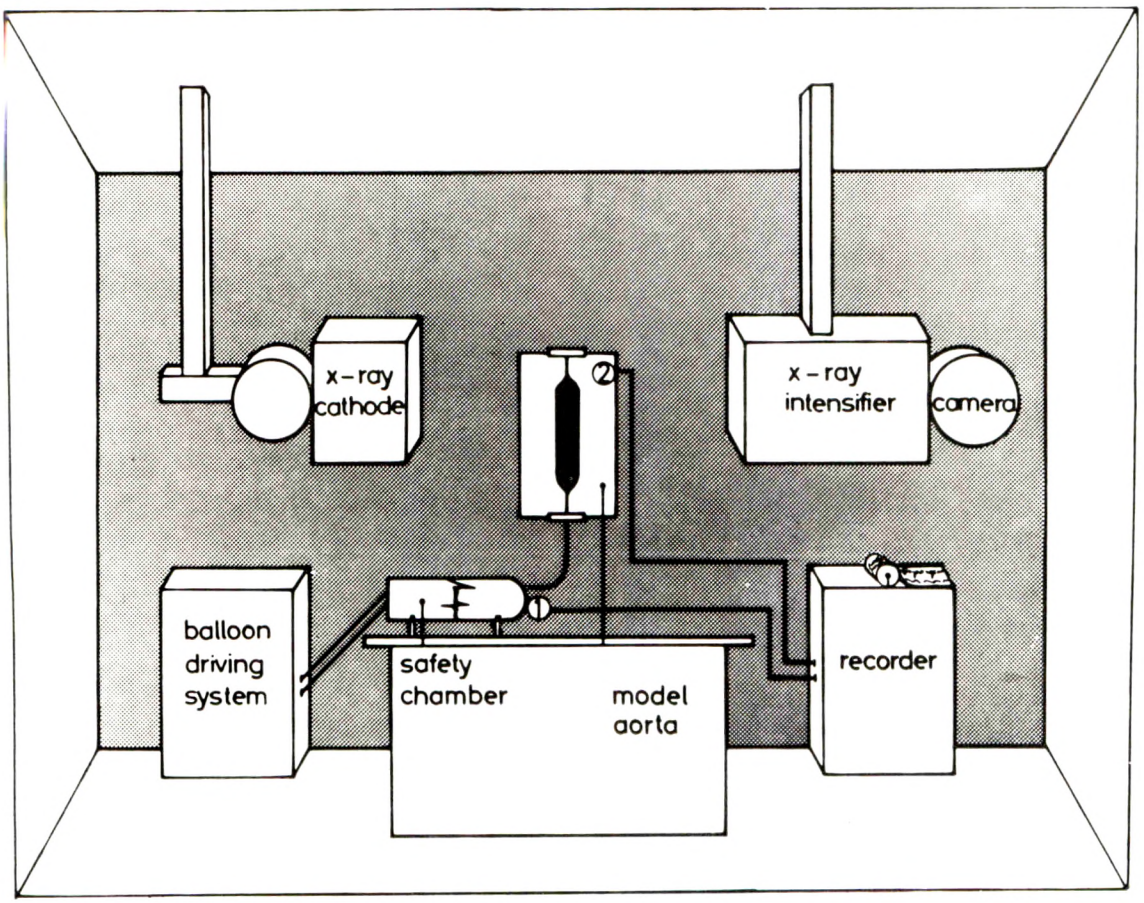


FIG. 1. *Experimental set-up for the model studies.*
The model aorta is connected to the safety chamber and the balloon driving system. Pressures in the safety chamber and the model aorta are recorded on a 6 channel printer. Cineroentgenograms were taken on 35 mm. film with 80 frames/sec.

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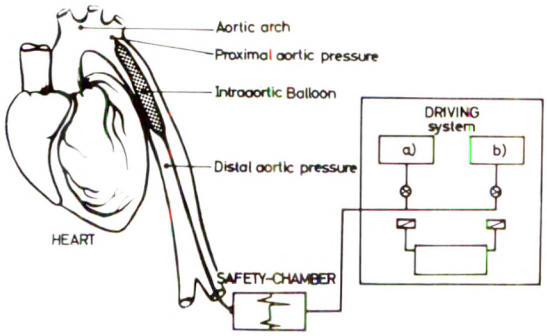


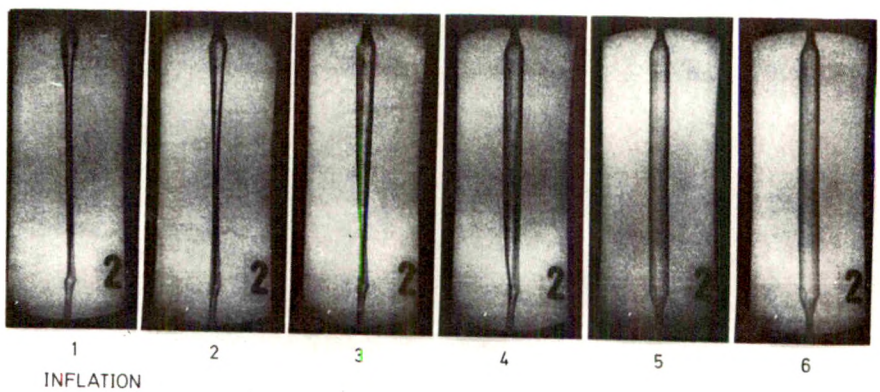
FIG. 2. *Experimental set-up for the animal studies.*
The intraaortic balloons are positioned in the descending aorta (straight balloons) or in the aortic arch (bent balloons; see Fig. 9 and 10) from the femoral artery connected to the safety chamber and the driving system. a) and b) represent the pressure and volume reservoirs, which are switched by solenoid valves to the safety chamber.

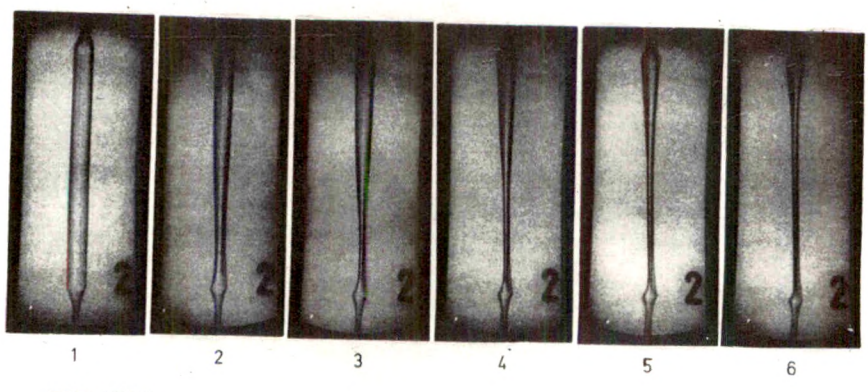
flating and deflating behavior of different balloon catheters. Bent balloons, which simulate the shape of the aortic arch[1,2] and straight balloons in the descending aorta were studied.

METHODS

MODEL STUDIES

The investigations were performed in a model of an aorta with pressure volume characteristics approximating those of a natural aorta within the pressure range to be considered. The model aorta consists of a Perspex case of 3,500 cm.^{3} volume, which is partially filled with water and air. The pressure inside the case and the air pad may be varied in order to simulate the compliance of a natural aorta. Details of





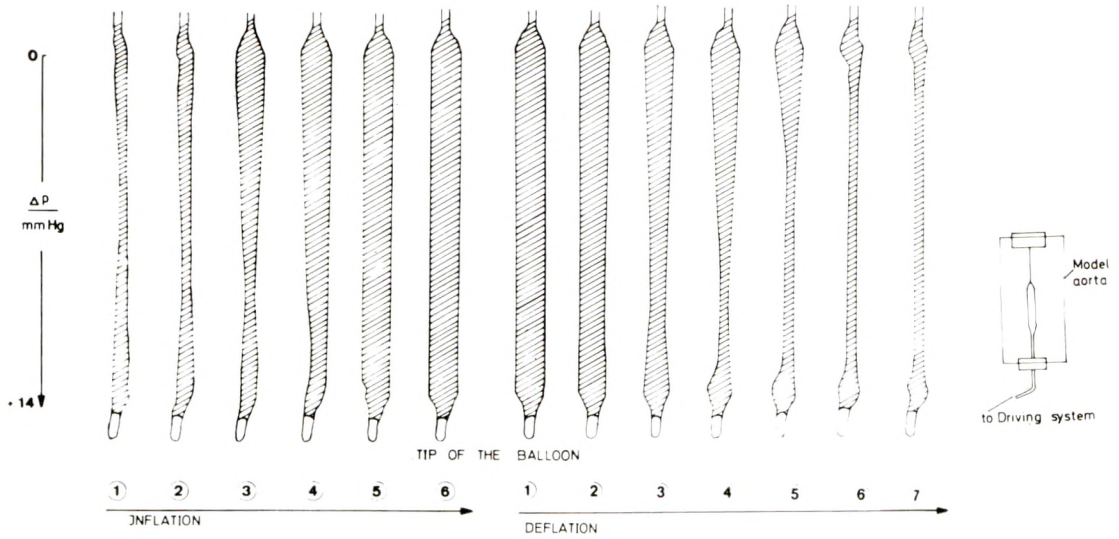


FIG. 4. *Drawings from cineroentgenograms of a straight balloon (28 ml.) in the model aorta in vertical position; 12.5 msec. time interval between each frame.*

The tip of the balloon is fixed at the bottom of the model, in reverse from the position in Figure 3. Under these conditions first gas filling can be seen at the end of the balloon (*2*). Deflation begins at the tip.

the model aorta have been described previously.[2] The model investigations were performed to study the influence of parameters determining balloon dynamics, such as pressure differences along the balloon, the velocity of inflation and the conditions governing the volume displacement in the aorta. Figure 1 shows the set-up for the model studies.

The balloons under study were fabricated from polyurethane with a length of 200 mm., a width of 15 mm., a volume of 28 ml. and a wall thickness of 100 μm. Pb O_2 was added to the polyurethane to make the balloon clearly visible on cineroentgenographic films. The balloons were connected to the driving system as described in details elsewhere.[3,4] Inflation and deflation of a 30 ml. balloon were completed within 58 msec. and 90 msec., respectively. Both phases were measured from the electrical signal triggering the solenoid valves of the driving unit. In the safety chamber and in the model aorta, pressures were measured with Statham* P 23 Db gauges to keep the experimental conditions constant. The recordings were performed with a paper speed of 500 mm./sec. on a 6 channel printer.† The cineangiograms were taken on 35 mm. film by a cine camera‡ with 80 frames/sec. The time

* Statham Instruments, Inc., California/USA.
† Oszillomink, Siemens, Erlangen/West Germany.
‡ Ariflex, Arnold & Richter, München/West Germany.

FIG. 3. *Cineroentgenograms of an inflation and deflation phase from a straight balloon (28 ml.) polyurethane with Pb O_2 in the model aorta in vertical position.*

As the tip of the balloon is fixed to the top of the model, the end *2* is under a hydrostatic pressure of 14 mm. Hg. The time interval between each frame is 12.5 msec. Inflation begins with a small distention of the tip of the balloon (Frame 2) proceeding to the end *2* (Frames 3–5). During deflation the gas first leaves the balloon end *2* (Frames 2 and 3). The balloon tip is deflated in the last frames (Frames 5 and 6).

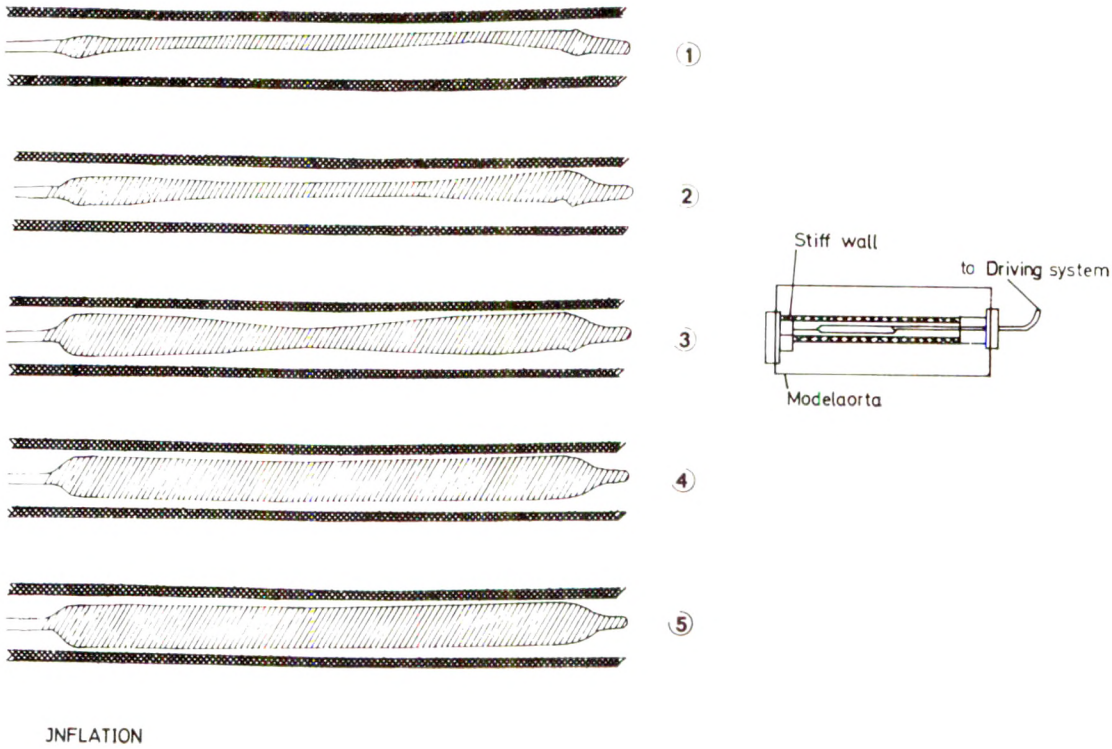


FIG. 5. *Influence of prevented radial volume displacement without pressure difference.*

Right. Experimental set-up: model aorta in horizontal position; straight balloon (28 ml.) surrounded by a stiff wall.

Left. Drawings from cineroentgenograms. Time interval between each frame is 12.5 msec. Typical balloon "trapping" is seen in Frame 3. The tip and the end of the balloon are inflated, whereas the middle is still collapsed.

between 2 pictures was 12.5 msec. For the evaluation of a single inflation or a deflation of the balloon, 5 to 7 frames were at disposal. A time code (cinechronocard§), simultaneously recorded with the pressure tracing and the films, served for synchronization of the angiograms and the pressure records. By projecting‖ the films on a screen, the aorta and the balloon were magnified to original size. The contours of the aorta and the balloon were copied on paper.

ANIMAL EXPERIMENTS

The animal experiments were performed on anesthetized dogs (Fig. 2). Straight balloons,¶ as used in the model studies, were placed from a femoral artery into the descending aorta; bent balloons were placed into the ascending aorta and the aortic arch. Pressures at the tip and the end of the balloons were taken from 8 F NIH** catheters of identical length introduced from a carotid artery and a femoral artery, respectively. R-wave triggered diastolic injections of contrast medium were used for the cineangiograms.

RESULTS

MODEL STUDIES

If the balloon is suspended in the model aorta in a vertical position, there is a hydrostatic pressure difference between the tip and the end of the balloon. This pressure difference can be varied by oblique posi-

§ Cinechronocard, Siemens, Erlangen/West Germany.
‖ Targe Arno®, Copenhagen/Denmark.
¶ Vygon, Breitscheid/West Germany.

** USCI, Glens Falls, New York/USA.

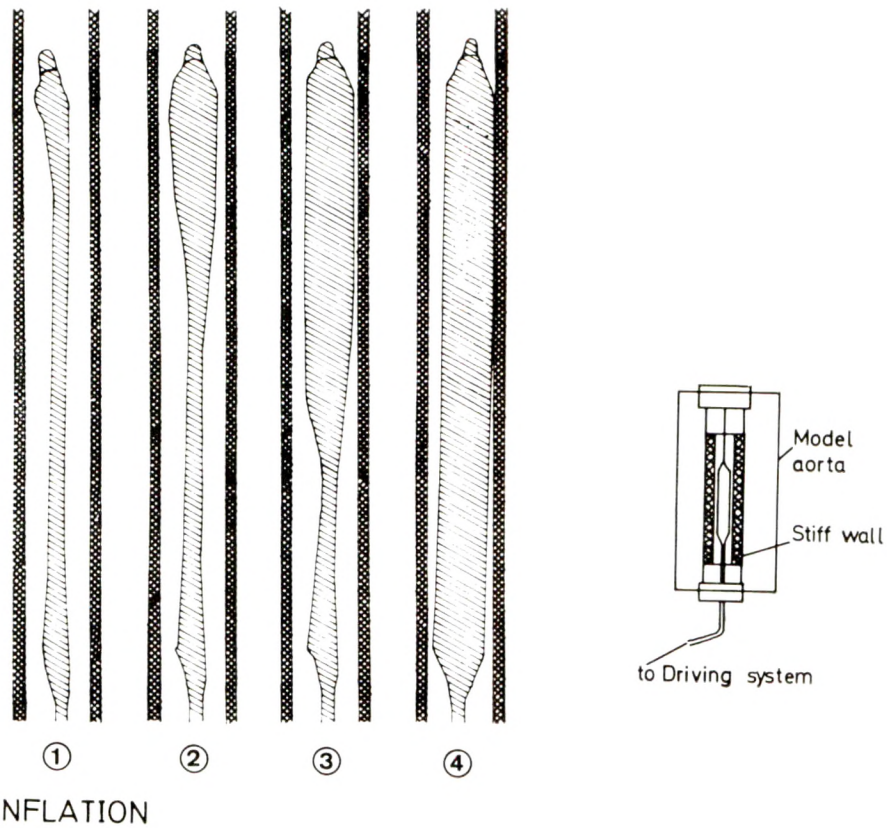


Fig. 6. *Influence of prevented radial volume displacement with hydrostatic pressure difference.*

Right. Experimental set-up: model aorta in vertical position; hydrostatic pressure on the end of the balloon 14 mm. Hg; straight balloon (28 ml.) surrounded by a stiff wall.

Left. Drawings from cineroentgenograms. Time interval between each frame is 12.5 msec. Inflation advances uniformly from the tip to the end of the balloon. Balloon "trapping" (Fig. 5) is not to be seen.

tioning or reversed by turning the model.

Figure 3 shows the inflation and deflation with the balloon tip suspended towards the top of the model. Under these conditions, the balloon end is under a pressure which is 14 mm. Hg higher than that at the tip. In Frame 2, the gas filling of the balloon begins at the tip, proceeds to the end of the balloon and is completed in Frame 5. The deflation is accomplished within 6 frames from the end to the tip. Thus, the part of the balloon which is deflated finally is the tip.

In a graph (Fig. 4), in which the contours of the balloon are drawn in accordance with the projected film, the reverse situation is shown: the balloon is suspended in the model with the tip towards the bottom. The pressure at the tip exceeds that of the end by 14 mm. Hg. The first distention of the balloon by gas now begins at the end; *i.e.*, the part of the balloon with the lowest hydrostatic pressure.

Figures 5 and 6 demonstrate the influence of prevented radial volume displacement. While in the above mentioned investigations (Fig. 3; and 4), a free distention of the balloon in the wide area of the model aorta was established, the free radial volume displacement is now prevented by surrounding the balloon with a stiff tube. The balloon is in a horizontal position (Fig. 5). In Frame 3, the tip and the end of the balloon are fully distended, but not the center. This occurs because the rapid gas filling of the balloon prevents the fluid sur-

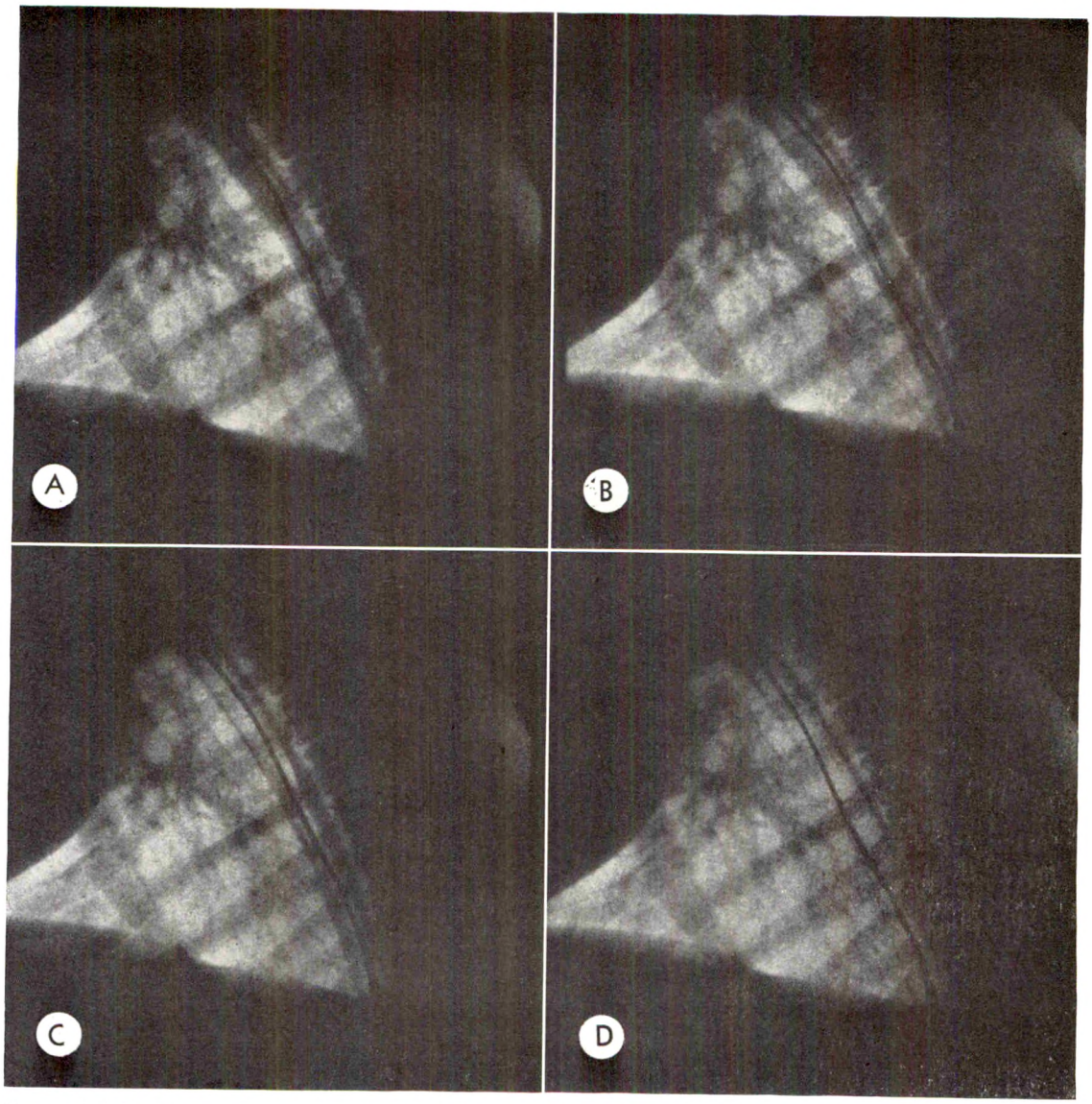


FIG. 7. (*A–D*) *Cineroentgenograms of an inflation phase of a straight balloon (28 ml.) in a dog (80 frames/sec.).* Inflation begins in *A* and *B* with a small distention of the balloon, proceeds in *C* and is completed in *D*.

rounding the balloon from being displaced as fast as it is in the model without the tube. However, this "trapping" effect[1] disappears if, under the same conditions, a pressure difference between tip and end of the balloon is established. The balloon is first inflated in that part where the lower pressure is exerted (Fig. 6).

Changes of inflation velocities ranging from 24 to 600 msec. resulted only in a delayed inflation of the balloon and did not lead to qualitative variations when compared with the described balloon dynamics.

ANIMAL EXPERIMENTS

Figure 7, *A–D*, shows a representative inflation procedure of a straight balloon (28 ml.) in a dog. It is obvious from Frame 1 that the first gas filling is seen around the central inlet. In Frame 2 the maximal distention of the balloon can be seen at the

tip proceeding, in Frame 3, to the end. Complete filling is established in Frame 4. Deflation proceeds in the reverse mode beginning at the end. The pressure tracings in the living animal, without balloon-action (Fig. 8, *A* and *B*), show that the pressure at the balloon end (distal aortic pressure) at the time of aortic valve closure exceeds that of the tip (proximal aortic pressure) by about 20 mm. Hg. From pressure records during balloon pulsation it is obvious that there is a pressure difference between the balloon tip and end during the diastole that is in the range of 15–40 mm. Hg at the beginning of the deflation.

The dynamics of an aortic arch balloon in the living animal are shown in Figures 9 and 10. In Frame 2, the first expansion of the balloon is visible in the aortic arch. Complete distention of the bent part

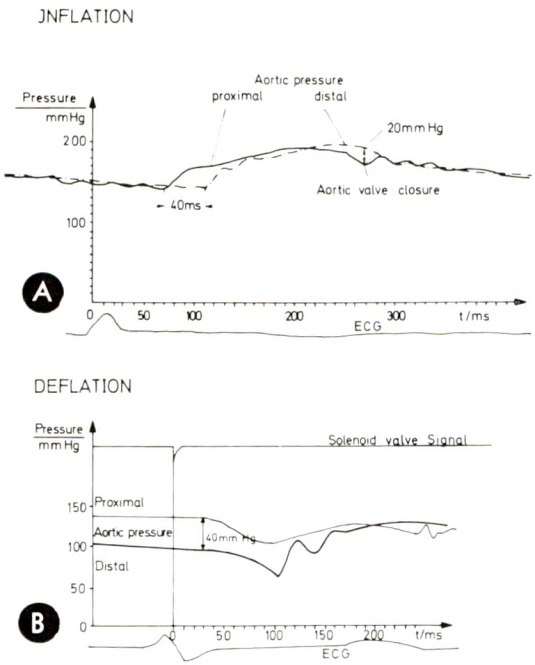


FIG. 8. *Pressure tracings in the proximal and distal aorta (A) without and (B) with balloon action.*

At the beginning of inflation (*A*), the pressure in the distal aorta exceeds that of the proximal aorta by 20 mm. Hg. Following diastolic increase, at the beginning of deflation (*B*), the proximal aortic pressure is 40 mm. Hg higher than the distal aortic pressure.

is attained in Frame 3, proceeding from there to the end. Pressure tracings are similar to those with the straight balloon. At the beginning of the filling process the pressure at the balloon end is 10–15 mm. Hg higher than at the tip.

The deflation of the aortic arch balloon is analogous to that of a straight balloon beginning at the tip, because the pressure during the whole deflation is higher at the tip than at the end (Fig. 10).

DISCUSSION

The investigations presented were performed to enlighten the cause of an insufficient diastolic increase with intraaortic balloon pulsation in low pressure states. In accordance with the model investigations in the living animal, the balloon dynamics are definitely dependent on the pressures in the aorta. At the time of aortic valve closure, the pressure at the end of the balloon exceeds that at the tip by 10 to 20 mm. Hg.[7] Thus the gas first fills the tip of the balloon. By this mechanism, in hypotensive states with reduced diameter of the aorta, the distention of the balloon tip may occlude the aorta without effective increase of blood pressure. Diastolic increase of blood pressure, following the expansion of the balloon, leads to a pressure increase in the proximal aorta of about 40 mm. Hg. Thus, gas first leaves the tip of the balloon. Because of the reversed pressure gradients, both deflation and inflation are first seen at the tip of the balloon in the proximal part of the aorta.

The trapping phenomenon described by Laird *et al.*[5] and Jones,[6] indicating simultaneous inflation of the balloon tip and end, is based on a model of an aorta which does not fit to the conditions *in vivo*. Their investigations were performed in horizontal positioned aortic models with stiff walls. Figure 5 shows that their results can be reproduced in our model with a stiff wall and in a horizontal position. However, balloon trapping disappears if pressure differences exist between the tip and

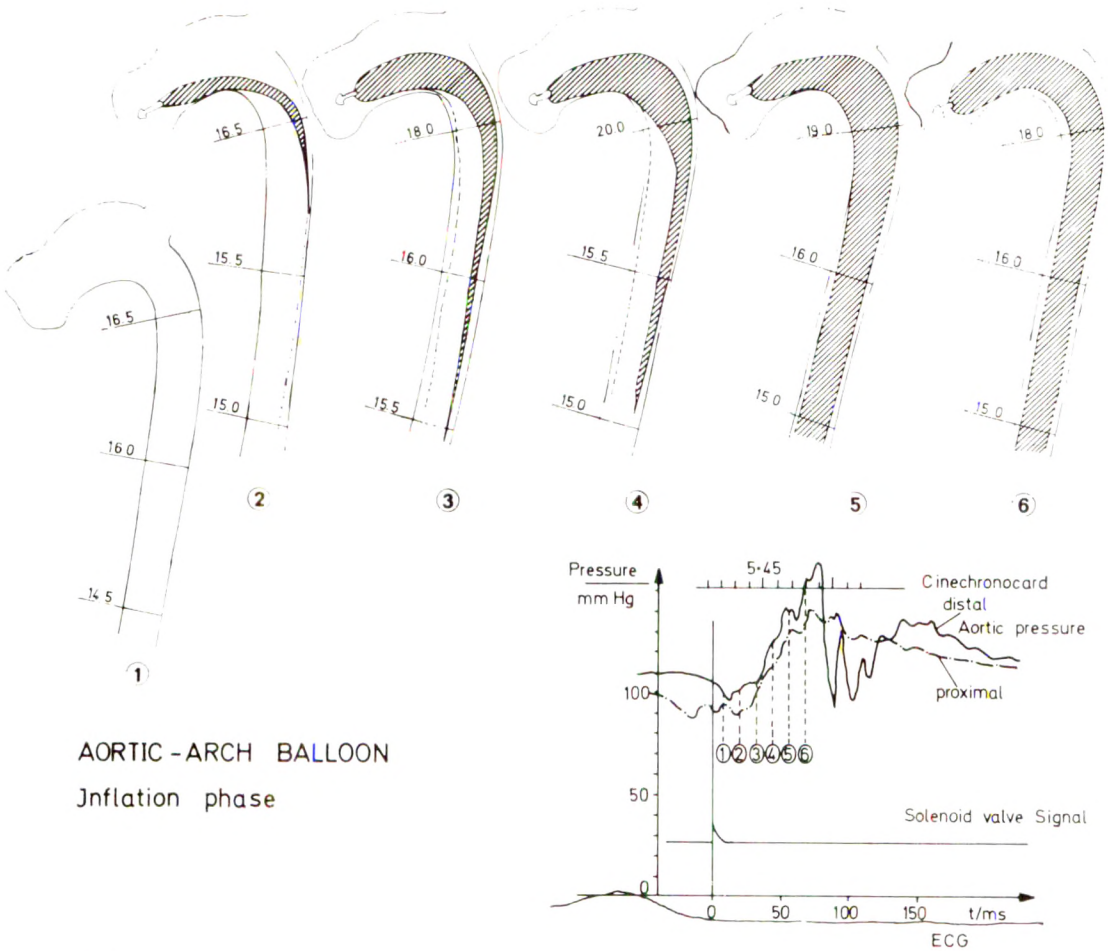


FIG. 9. *Inflation of an aortic arch balloon (28 ml.) in a dog.*

Top. Drawings from cineangiograms. Time interval between each frame is 12.5 msec. The pictures are taken at the time marked in the pressure tracings. Gas filling occurs first in the tip of the balloon (Frames 2 and 3), than advances to the end (Frames 4 and 5).

Bottom. Records corresponding to the cineangiograms. From above to below: cinechronocard signal for correlation of pictures to records, proximal and distal aortic pressures, measured at the tip and the end of the balloon; solenoid valve signal from the balloon driving system; ECG. Corresponding to Frame 2, the pressure is higher at the end of the balloon (distal aortic pressure) when inflation begins.

the end of the balloon. *In vivo* conditions, resulting in balloon trapping, will probably only occur if the balloons are relatively large compared with the diameter of the aorta and if the aortic elasticity is extremely low.

The dynamics of the aortic arch balloon are similar to those of the straight balloon. However, more effective diastolic increase is accomplished with pulsation in the aortic arch even in low pressure states.[1] The diameter of the aorta is largest in the ascending part and the distention of the bent part of the balloon is extremely rapid. In addition, there is only little dumping of the pressure wave, because diastolic increase occurs nearer the aortic valve.

SUMMARY

To optimize intraaortic balloon pulsation, the inflation and deflation mechanisms of different intraaortic balloons were

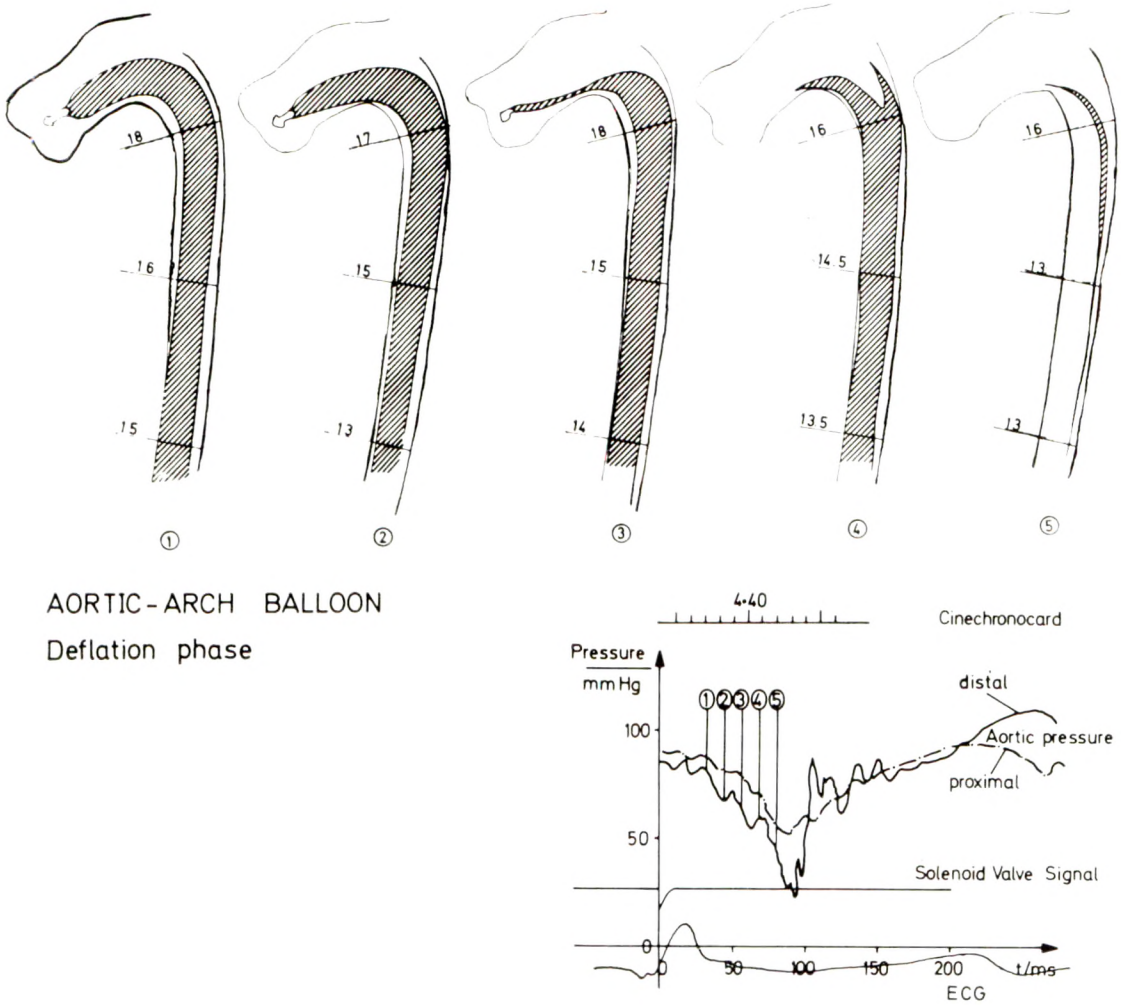


FIG. 10. *Deflation of an aortic arch balloon (28 ml.) in a dog.*

Top. Drawings from cineangiograms. Time interval between pictures is 12.5 msec. Gas first leaves the tip of the balloon.

Bottom. Records corresponding to the cineangiograms (see Fig. 9). Corresponding to Frame 2, the pressure is higher at the tip of the balloon (proximal aortic pressure) when deflation begins.

studied in a model aorta with pressure volume characteristics approximating those of a natural aorta and in anesthetized dogs by cineangiography. It could be demonstrated that inflation of straight balloons begins at the tip and proceeds from there to the end of the balloon. The distention of the aortic arch balloons is first observed in the aortic arch; inflation than proceeds to the end of the balloon. Deflation of both balloon types has the same direction as inflation, beginning at the tip.

From the model investigations it can be concluded that the balloon dynamics are definitely pressure dependent: inflation begins in that balloon part, where the pressure is lowest; deflation where the highest pressure is exerted. These results explain the behavior of balloons *in vivo*.

In the aorta of living dogs in the early diastole, the pressure nearest to the heart is less than in lower parts of the aorta. Therefore, gas filling is first accomplished at the balloon tip. However, following

diastolic increase in late diastole, the pressure difference between the proximal and distal aorta is reversed: deflation begins at the tip of the balloon.

When hypotension decreases the diameter of the aorta to that of the balloon, occlusion is produced at the balloon tip and only a minor or no pulse wave is allowed to run towards the heart. This is the reason for failure of intraaortic balloon pulsation in very hypotensive states and in those with a small diameter of the aorta.

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REFERENCES

1. Bleifeld, W., Bussmann, W. D., Meyer, J., and Effert, S. Improved cardiac assistance with aortic arch balloon. To be published.
2. Bussmann, W. D., Bleifeld, W., Irnich, W., Meyer, J., Meyer-Hartwig, K., and Effert, S. Vergleichende haemodynamische Untersuchungen bei Ballon-pulsation mit geradem (Aorta ascendens) und mit gebogenem Ballon (Aortenbogen). *Biomed. Technik*, 1971, *16*, 90–93.
3. Irnich, W., Meyer-Hartwig, K., and Bleifeld, W. Modelluntersuchungen zur intraaortalen Ballonpulsation. *Biomed. Technik*, 1971, *16*, 9–21.
4. Irnich, W., Bleifeld, W., Meyer-Hartwig, K., and Bisping, H. J. Die physiologischen und technischen Grundlagen der Ballonpulsationsmethode. *Ztschr. Kreisl. Fortsch.*, 1972, *61*, 339–349.
5. Laird, J. D., Jones, R. T., Austen, W. G., and Buckley, M. J. Observations of dynamics of intra-aortic balloon pump. Digest, 7th Int. Conf. Med. a Biol. Engn. Stockholm, 1967, *29–4*, 382.
6. Jones, R. T. Digest Fluid Dynamics of Heart Assist Devices, Agard-symposium on Flow Dynamics. *Neapel*, 1970, *12*, 1–9.
7. O'Rourke, M. F. Pressure and flow waves in systemic arteries and anatomical design of arterial system. *J. Appl. Physiol.*, 1967, *23*, 139–149.

TOTAL AORTOGRAPHY IN THE DIAGNOSIS OF TAKAYASU'S ARTERITIS*

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TAKAYASU'S disease is an arteritis of unknown etiology involving the aorta and its branches as well as the pulmonary arteries. Although first recognized as a clinical entity in Japan, more current reports from Asia,[5,26] Africa,[9,12] West European countries,[1,2,7,23,28] and the U.S.A.[3,4,6,13,17,24] indicate its world-wide occurrence. The disease is apparently much more common in the U.S.A. than is generally suspected.[24]

There appear to be several reasons for the infrequent recognition of this abnormality: (1) the impression still prevails that the syndrome is mainly confined to the Orient; (2) the full extent of vascular involvement in this abnormality has been recognized only in recent years;[9,20,21,23] (3) the symptomatology of Takayasu's arteritis is very variable (it has been described as "one of the great imitators of medicine"[28] and patients are frequently treated for a variety of complaints without the benefit of total aortography which is of primary diagnostic value); (4) finally, the disease is recorded under different names. These include Takayasu's arteriopathy or pulseless disease,[27] primary arteritis of the aortic arch,[3] brachiocephalic arteritis,[31] reversed coarctation,[8] subisthmic aortic stenosis of the descending aorta,[18] primary arteritis of the abdominal aorta,[5] atypical coarctation of the aorta,[12] and arteritis of the aorta and its major branches.[9]

The authors reviewed 8 cases of Takayasu's arteritis. These cases represent a good cross section of the diversity of anatomic involvement in this disease.

HISTORIC ASPECTS

In 1908 Takayasu, an ophthalmologist, first described peculiar eyeground changes in a young female, consisting of wreathlike anastomoses in the central vessels of the retina.[29] The attention of clinicians was subsequently drawn to absent pulsations in brachiocephalic vessels in association with ocular changes. Shimizu and Sano[27] described the clinical picture of obliterative brachiocephalic arteritis, leading to cerebral symptoms and arteriovenous communications in the retina. They named the syndrome "pulseless disease." Frövig and Löken,[7] and Ask-Upmark[1,2] subsequently reported similar cases from Scandinavia.

The very first reports had already recognized that the inflammatory process is not confined to the arch alone but may extend to involve the descending and abdominal aorta.

As angiographic and autopsy evidence accumulated, the full extent of the disease became apparent. Nasu,[21] in a review of 21 autopsies, found involvement of both the thoracic and abdominal aorta in the majority of cases. Occasionally the descending or abdominal aorta was coarcted. Stenosis or occlusion of the abdominovisceral and renal arteries was common.

The pulmonary arteries were similarly affected in the majority of cases.

Inada *et al.*,[11] Kozuka *et al.*,[15] Yamada *et al.*,[32] and Ueda *et al.*[30] provided convincing clinical, angiographic and pathologic evidence that the so-called "atypical coarctation of the aorta" represents a variable localization of Takayasu's arteritis with or without involvement of the arch.

The differential features of atypical coarctation from congenital isthmic coarctation have been outlined by several authors.[11,15,18–20]

Takayasu's disease is currently recognized as an arteritis of unknown etiology

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which may involve the aorta and any of the larger elastic arteries and which may progress to stenosis, occlusion or aneurysmal dilatation of the affected vessels.[9,20,23]

PATHOLOGIC ASPECTS

The characteristic angiographic findings correlate closely with the pathologic features of the disease.

In the early stages, the adventitia and media are infiltrated by lymphocytes and plasma cells.[7,13,21,27] Cuffing of the vasa vasorum may produce thickening and obliteration of the nutrient arteries. With progression, diffuse inflammatory tissue proliferates, leading to focal or diffuse disruption of the tunica elastica and muscularis of the media. Fibrous tissue replaces the functional elements of the vessel with thickening of the arterial wall.[19,21]

The intima is not directly involved in the inflammatory process. Fibrous intimal thickening is a secondary reactive phenomenon occurring at sites of arteritis.[21] The degree and character of intimal proliferation varies. In the aorta, at sites of severe narrowing, the intima may be markedly thickened with irregular infoldings into the lumen.[19,21] In the absence of severe narrowing, intimal hyperplasia may assume a more uniform distribution.[3] Our experience at aortography indicates that, except for segments of severe narrowing, the internal surface of the aorta tends to be smooth even when the contour of the vessel is grossly affected.

The histologic appearance will vary depending upon the stage and duration of the disease.[2,13] In more chronic cases fibrosis predominates,[9] with variable degrees of secondary postinflammatory deposition of calcium.[13,30]

Hypertension is common in Takayasu's arteritis due to renal involvement or coarctation and may accelerate the development of secondary atheromatosis.[2] Characteristically, however, atheromatosis is frequently limited to the segments of the aorta at and proximal to the site of coarctation.[19,26]

Arteritis of the aorta may extend into any of its branches, leading to stenosis or occlusion. The coronary arteries are usually not directly involved. Intimal thickening of the aorta, however, may narrow the coronary ostia[4,7,13] leading to anginal pain[20,23] and sometimes myocardial infarction.[3,4,23] Extension of aortitis into the aortic valves with contraction and insufficiency has been described.[4,13]

CLINICAL ASPECTS

In the majority of patients Takayasu's arteritis begins with acute systemic signs and symptoms.[20,23,28] After the generalized manifestations subside, stenosing arterial changes are responsible for the clinical symptoms of regional ischemia. Frequently the onset is insidious[20] or the disease assumes an intermittently relapsing course.[28]

Nonspecific findings in the early prepulseless phase are: fever, arthralgias, anorexia, vomiting, cough, palpitations etc.[20,23,28] Local tenderness over the affected arteries may appear.

Clinical evaluation is of eminent importance in the chronic stage of the disease because of the variability of the presenting symptoms.[28]

Involvement of the carotid arteries leads to a variety of cerebral and visual disturbances.[28]

Coarctation of the aorta is associated with hypertension in the upper trunk and decreased pulsations in the lower limbs closely resembling congenital isthmic coarctation.[19] Secondary to stenosis or occlusion of the renal arteries, renovascular hypertension is common.

Signs of heart disease are observed in the majority of patients[20,23] in the form of effort angina, myocardial infarction,[3,4,23] heart murmurs,[23] or cardiac failure secondary to hypertension. Cardiac murmurs without significant valvular disease are common.[23] Rare valvular involvement seems to be confined to the aortic valves, due to direct extension of aortitis.[4]

A close relationship between Takayasu's arteritis and autoimmune systemic dis-

orders has been suspected by several investigators.[6,13,23,28] Although the etiology of Takayasu's arteritis remains obscure, the concept of an autoimmune mechanism is currently favored. This, however, lacks definitive proof.[10]

Laboratory findings of elevated γ and $\alpha 2$ globulin fractions and sedimentation rate, although nonspecific, support the hypothesis of autoimmune disease.[10,13,17,20,23,24]

MATERIAL AND METHOD

Aortography was performed in 8 cases of Takayasu's arteritis. Apparently due to greater awareness, 5 of these cases were identified within the last year.

All patients were females ranging from 18 to 75 years of age. All were residents of New York City. Six were natives of Puerto Rico, 1 was born in Haiti and 1 in Guatemala.

The aim of arteriography was to visualize the entire aorta and all its branches. The site of puncture is frequently dictated by the location and degree of vascular obliterative changes. For example, in the presence of coarctation, an axillary puncture for visualization of the arch and descending aorta usually needs to be supplemented by a femoral puncture for satisfactory demonstration of the abdominal aorta.

REPORT OF CASES

Case 1. This 38 year old Puerto Rican female was admitted with a history of blurred vision in her left eye and increased pulsations in her neck since childhood. Dyspnea on exertion, palpitations, frequent headaches and hypertension had been present for 14 years.

At physical examination the blood pressure in the right arm fluctuated between 180 to 300 mm. Hg systolic and 50 to 60 mm. Hg diastolic, whereas the blood pressure in the left arm was 90/60 mm. Hg. Left eyeground examination revealed central fragmentation and beading of arterioles and venules. The retinal background was pale with only a few thin vessels. An ejection systolic murmur was present over the pulmonic area.

Significant laboratory findings included: an elevated sedimentation rate of 75 mm./hr.; nonreactive V.D.R.L.; total protein, 7.6 gm. per cent, with 53 per cent albumin. Electrophoresis disclosed an elevation of $\alpha 2$ globulin fraction.

At roentgenography calcification outlined the arch, the descending aorta and in a patchy fashion the abdominal aorta down to the bifurcation. At cardiac catheterization no valvular abnormalities were found. At retrograde aortography, widespread changes of arteritis were shown (Fig. 1, *A* and *B*).

The patient is being followed in the cardiac clinic and is maintained on antihypertensive drugs with gratifying symptomatic relief.

Case 11. This 26 year old Puerto Rican female was admitted with headaches and dizziness of several months' duration. Hypertension had been known for at least 2 years. Except for a past history of cervical lymphadenopathy since childhood, she was apparently in good health.

Significant physical findings were confined to severe hypertension in the left arm of 220/140 mm. Hg with unobtainable blood pressure in her right arm. Enlarged nontender lymph nodes were palpated in the left cervical region. Loud murmurs were heard over the base of the heart.

Laboratory studies revealed: a rapid sedimentation rate of 70 mm./hr.; total protein of 6.5 gm. per cent, with 53 per cent albumin. Electrophoresis disclosed elevation of γ and $\alpha 2$ globulins. V.D.R.L. was nonreactive.

Biopsy of the cervical lymph nodes was reported as tuberculous lymphadenitis.

Chest roentgenography demonstrated a small area of linear calcification in the arch.

At cardiac catheterization, no valvular abnormalities were found. The main pulmonary artery pressure was 80 mm. Hg with a drop to 50 mm. across the right and left pulmonary arteries indicating a functionally significant bilateral stenosis. Pulmonary arteriography demonstrated arteritic involvement of the pulmonary arteries (Fig. 2*C*). At aortography multiple sites of occlusion and aortic wall involvement were shown (Fig. 2, *A* and *B*).

The patient refused surgical intervention and is being treated medically.

Case 111. This 24 year old Puerto Rican female was first hospitalized in 1963 at the age of 18 years. Since childhood she complained of

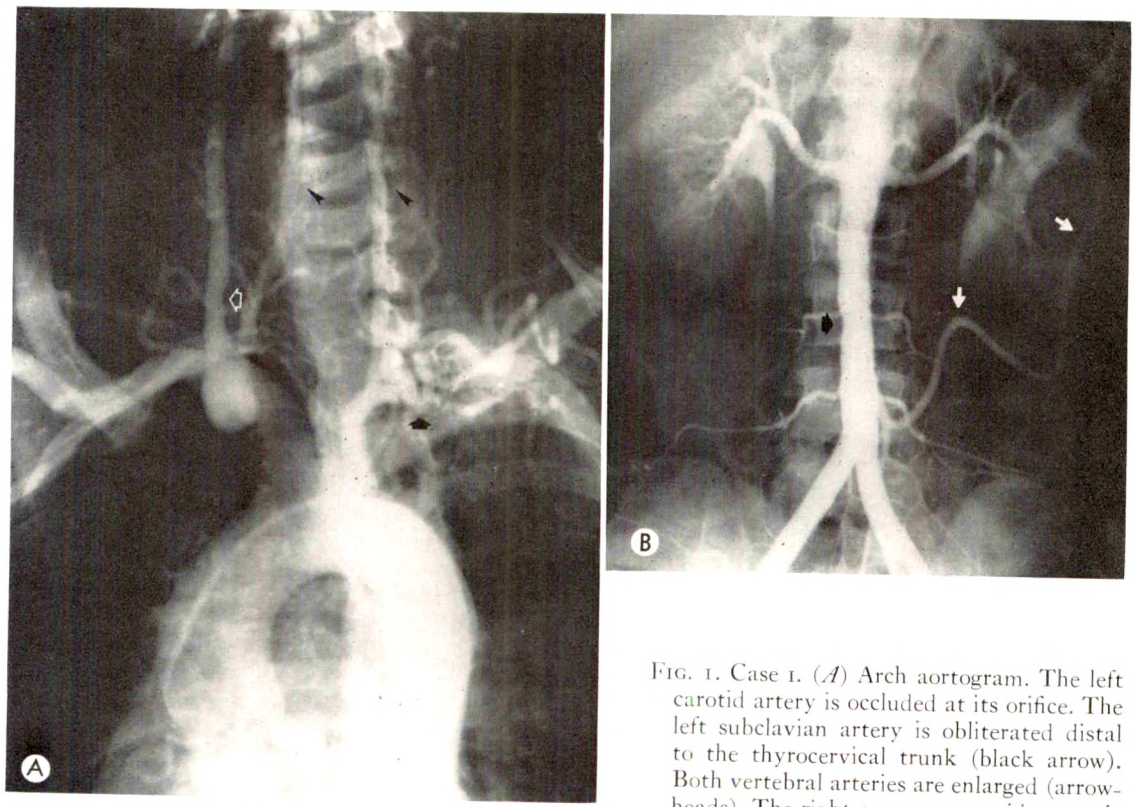


FIG. 1. Case I. (*A*) Arch aortogram. The left carotid artery is occluded at its orifice. The left subclavian artery is obliterated distal to the thyrocervical trunk (black arrow). Both vertebral arteries are enlarged (arrowheads). The right common carotid artery is slightly narrowed (white arrow). At thoracic aortography the lower descending aorta was moderately narrowed with smooth borders. The upper abdominal aorta assumed again a normal caliber. (*B*) Low abdominal aortogram. On plain roentgenograms, spotty calcification outlined the descending and abdominal aorta. The infrarenal abdominal aorta is narrowed with slight constriction (black arrow). Isolated changes of this nature can be easily overlooked. A large left colic artery (white arrows) proceeds to supply the superior mesenteric artery which was occluded 2 cm. from its origin.

dyspnea on effort, intermittent claudication in the legs, hot flushes and headaches.

Significant findings at admission were: hypertension of 190/110 mm. Hg equal in both arms and hypotension in her lower limbs with systolic pressure recorded at 80 mm. Hg. A systolic murmur was heard along the left sternal border.

The laboratory data were within normal limits.

Heart catheterization showed no abnormalities. Anticipating an isthmic coarctation of the congenital variety, contrast material was injected at the orifice of the left subclavian artery. A diffuse marked narrowing of the descending aorta was shown (Fig. 3).

The patient was operated on in 1964 and a thoraco-abdominal aortic bypass procedure was performed. Since the operation the patient became completely asymptomatic. When last seen in 1969 her blood pressure was 125/80 mm. Hg.

CASE IV. This 36 year old Guatemalan female was first hospitalized in 1958 at the age of 25 years with a several year history of hypertension headaches, dizzy spells, dyspnea on exertion and coldness of the feet.

Significant physical findings at the first admission were hypertension nearly equal in both arms of 170/110 mm. Hg and feeble femoral pulses. A systolic murmur along the right sternal border and a diastolic blowing murmur over the left one were present. A clinical diagnosis of congenital aortic coarctation, adult type, was made and the patient was explored. At operation it was realized that the coarctation was atypical in type with the area of severe narrowing in the region of the diaphragm. A hard calcified arch and a cylindrical calcified aneurysm of the left subclavian artery were palpated.

A few weeks later, a side-to-side thoraco-abdominal aortic bypass was performed using a

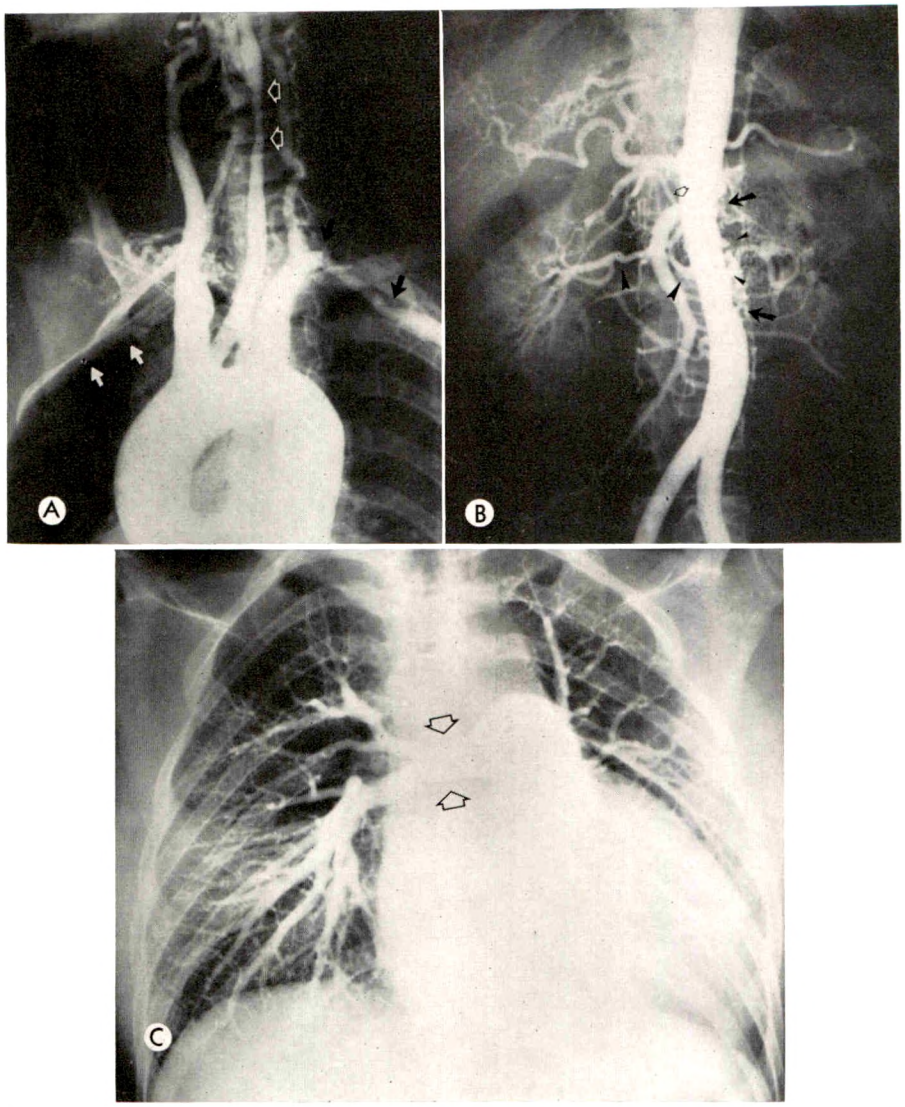


Fig. 2. Case 11. (*A*) Arch aortogram. The arch is slightly narrowed. The right subclavian artery is completely occluded distal to the thyrocervical trunk. Rich collateral circulation composed of the upper 3 intercostals and branches of the thyrocervical trunk revascularized the axillary artery. Coiling of the second intercostal artery produced a very fine notching of the adjacent rib (white arrows). The left common carotid artery is constricted proximal to its bifurcation (white open arrows). The constricted segment is well demarcated with minimal irregularity of its contour and sharp luminal surface. Both vertebral arteries are of large caliber. Constriction of the left subclavian artery extends from the thyrocervical trunk to the first rib, with post-stenotic dilatation (black arrows). (*B*) Abdominal aortogram. Segmental constriction of the mid abdominal aorta (black arrows). A short segmental narrowing of the superior mesenteric artery can be identified distal to its origin (black open arrow). The left kidney has a double blood supply with the upper vessel occluded and the lower severely stenosed (small arrowheads). A rich collateral plexus is revascularizing the left kidney, which was only slightly smaller than the right. The right renal artery is constricted throughout its length commencing about 5 mm. from its root (large arrowheads). The inferior mesenteric artery is occluded at its origin and was subsequently revascularized through the internal pudendal-rectal anastomosis. (*C*) Pulmonary arteriogram. The main pulmonary artery trunk is enlarged with smooth narrowing of the right main pulmonary artery (arrows). The vessels in the right upper lobe are irregular and sparse and there is marked decrease in perfusion to the left lower lobe with paucity of vessels indicating obstruction. On late roentgenograms of the arch aortography, enlarged bronchial arteries perfused these regions.

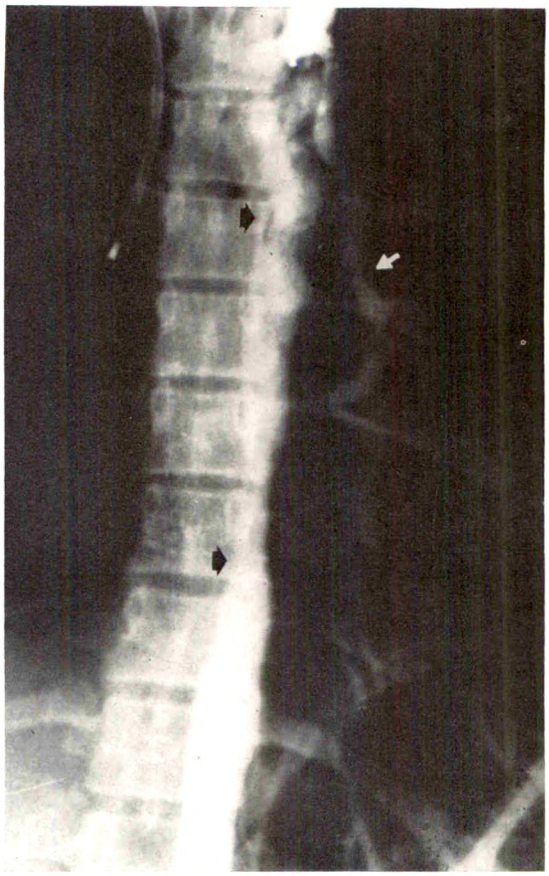


FIG. 3. Case III. Thoracic aortogram with the catheter tip at the orifice of the left subclavian artery. Diffuse marked narrowing of the descending aorta with a grossly undulating contour. This elongated "atypical coarctation" tapers off gradually with the point of maximal narrowing located a few cm. above the diaphragm (black arrows). Distally the thoracic and abdominal aorta resume a normal appearance. A large left mammary artery (white arrow) communicates with the inferior epigastric artery and through the musculophrenic branches with the inferior phrenic artery creating a prominent collateral channel.

nylon graft. The lumen of the abdominal aorta was also reported narrowed.

Following the operation the patient's clinical status remained essentially unchanged; the bypass was assumed to be not patent.

Serial electrocardiograms obtained over several months were interpreted as compatible with the evolutional changes of an anterior wall infarction.

When readmitted in 1968 her clinical status had remained essentially unchanged. In addition, recent claudication appeared in the lower limbs.

Significant laboratory findings were: elevated sedimentation rate of 45 mm./hr.; white blood cell count of 5,000–6,000, with persistent eosinophilia of 10 to 14 per cent.

At chest roentgenography linear calcification outlined the isthmus and the cylindrical dilatation of the left subclavian artery.

Cardiac catheterization was performed through the right brachial artery. The catheter could be passed into the ascending aorta only with extreme difficulty. Mild aortic regurgitation was present. The blood pressure in the aortic root was recorded at 220/135 mm. Hg.

A thoracic and abdominal aortogram was obtained and showed multiple segments of aortic involvement with coarctation of the descending portion (Fig. 4). The left subclavian artery was stenosed and the right subclavian artery never visualized during arteriography. Considering the difficulties encountered in passage of the catheter it was apparently stenosed and temporarily obstructed by the catheter. A bilateral subclavian obstruction induced a blood pressure of virtually equal levels in both upper extremities. The patient refused surgical intervention.

She is being followed in the cardiac clinic and treated medically.

CASE V. This 18 year old Puerto Rican female was admitted for evaluation of hypertension with a 2 year history of headaches, blurring of vision, dizzy spells and occasional syncope. The past history was otherwise unremarkable.

The blood pressure in her right arm was 180/110 mm. Hg and 120/70 in her left arm. A systolic murmur was recorded over the base of the heart.

The laboratory findings were within normal limits, except for an elevated sedimentation rate of 60 mm./hr.

Chest roentgenogram demonstrated indentation of aortic contour in the region of the isthmus with a prominent aortic knob and ascending aorta (Fig. 5*A*). A translumbar aortogram (Fig. 5*B*) and a retrograde thoracic aortogram (Fig. 5*C*). demonstrated widespread involvement of the aorta and its branches.

The subsequent surgical management of this patient will be the subject of a separate report.

CASE VI. This 52 year old Negro female from

Haiti presented with a history of several years of headaches, palpitations, precordial pain unrelated to effort and mild dyspnea on exertion. Hypertension was first discovered 20 years prior to admission.

The pertinent physical findings were equal hypertension in both upper extremities and ranging from 185/80 mm. Hg to 230/80 mm. Hg. The femoral pressures were equal and recorded at 130 mm. Hg systolic. An ejection systolic murmur over the left sternal border was present. At lateral chest roentgenography a pencil-like calcification outlined the descending aorta.

Thoracic and abdominal aortography was obtained. A diffuse narrowing of the descending aorta was shown (Fig. 6).

The coarcted segment was excised and replaced with a dacron graft. The postoperative course was uneventful. Her blood pressure stabilized at 130/80 mm. Hg and she was relieved of all her symptoms.

The pathologic report was as follows: "Portion of the descending thoracic aorta measuring 11 cm. in length, showing segmental atherosclerosis gradually diminishing proximally. There is subintimal calcification and severe focal medial fibrosis. Surgical ends are essentially normal. Such segmental changes are not typical of ordinary atherosclerosis, but would rather suggest degenerative changes secondary to some previous vascular disease."

Case vii. This 75 year old Puerto Rican female was admitted because of congestive heart failure. The patient was hospitalized for the first time 6 years previously for fever of unknown origin. Hypertension was recorded at that time. Gradually progressive dyspnea and orthopnea developed several weeks prior to admission. Except for pneumonitis in childhood the patient claimed to have enjoyed good health.

At physical examination a loud systolic murmur was best heard over the apex of the heart. The blood pressure, equal in both arms, was recorded at 200/110 mm. Hg. The femoral pulses were of a good quality.

Laboratory data of note included: elevated sedimentation rate of 50 mm./hr.; total protein of 7.7 gm. per cent, with 50.5 per cent albumin; and nonreactive V.D.R.L. Protein electrophoresis disclosed marked elevation of $\alpha 2$ globulin and moderate elevation of $\alpha 1$ fraction.

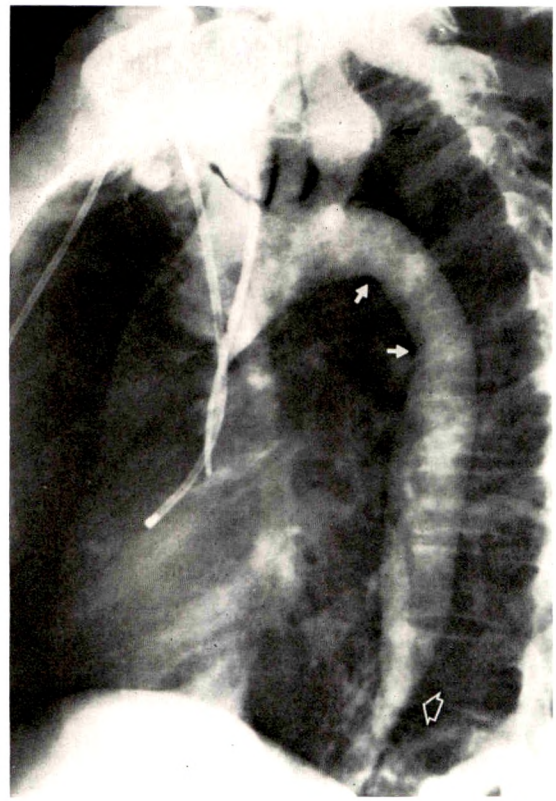


Fig. 4. Case iv. Thoracic aortogram. The ascending aorta and the arch are dilated. A moderate segmental narrowing with smooth contours and luminal surface is present in the isthmic region (white arrows). The aorta widens again distally only to become markedly constricted over several cm. before reaching the diaphragm (white open arrow). At the site of narrowing the aortic lumen is irregular and shaggy. Post-stenotic dilatation of the upper abdominal aorta was present. The vessel, however, was moderately narrowed more distally, suggesting diffuse arteritic involvement. Calcification outlines the aneurysm of the proximal left subclavian artery (black arrow). Distally the vessel maintained a wide caliber and was then severely stenosed over the first rib with post-stenotic dilatation. The right subclavian artery through which the catheter was advanced never visualized indicating stenosis.

Roentgenography showed calcification of the aortic arch and the abdominal aorta. Aortography revealed extensive involvement of the descending and abdominal aorta with obstructive changes of the renal and visceral branches (Fig. 7, *A–C*).

Under treatment the patient gradually improved and was discharged to the home care service.

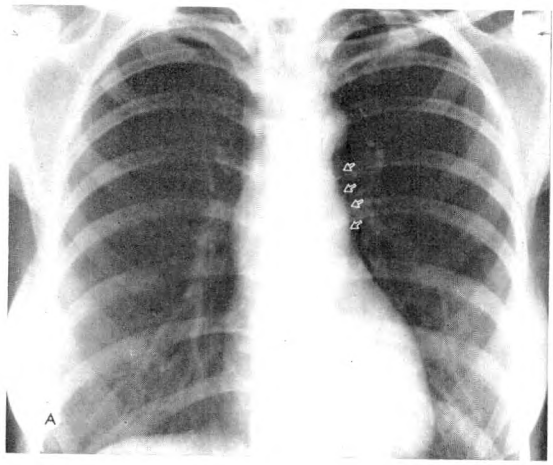
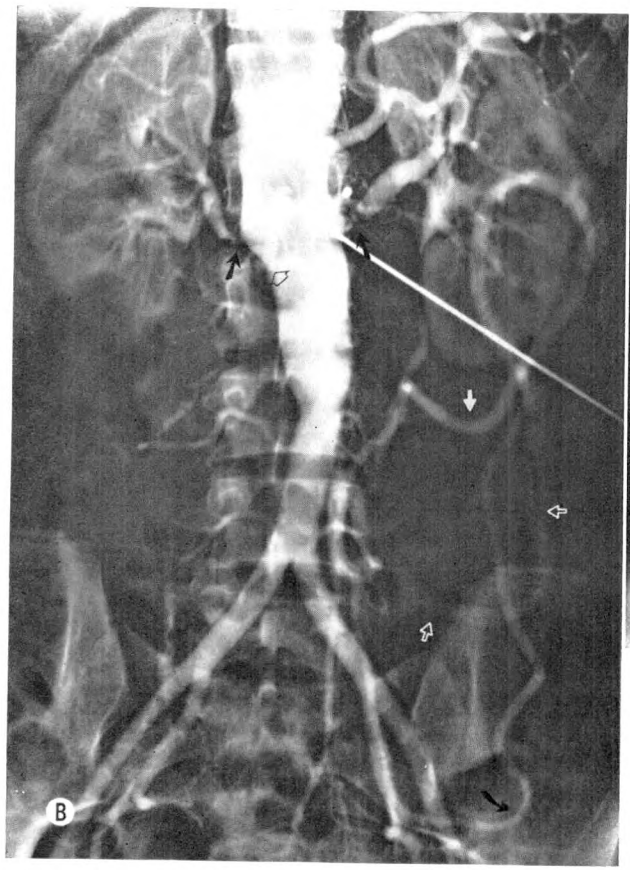


FIG. 5. Case v. (*A*) Roentgenogram of the chest demonstrates indentation of the aortic contour in the region of the isthmus (white arrows), with a prominent aortic knob and ascending aorta. At thoracic aortography a waist-like constriction measuring about 4 cm. in length was present in the isthmic region. The ascending aorta was dilated. The left subclavian artery was occluded at its root with revascularization of the distal vessel through the left vertebral artery.



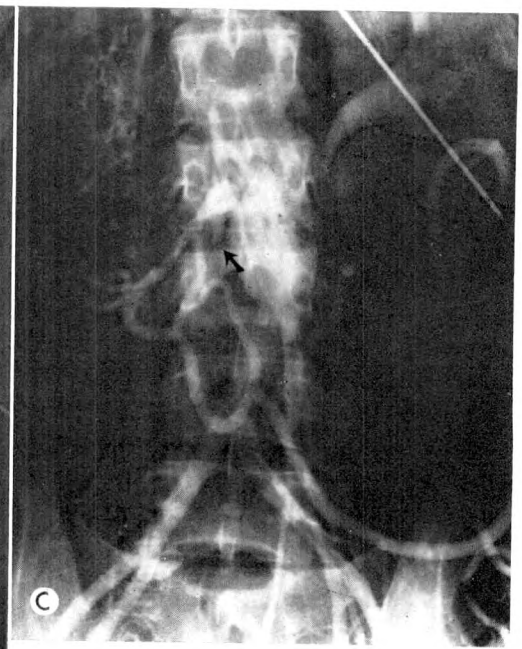


FIG. 5. (*B*) Translumbar aortogram demonstrates diffuse dilatation of the abdominal aorta, with multiple outpouchings, extending from the diaphragmatic hiatus to just proximal to the origin of the inferior mesenteric artery. Both renal arteries are stenosed at their orifices with post-stenotic dilatations (black arrows). The superior mesenteric artery is occluded about 2 cm. from its origin (black open arrow). The left colic artery (white closed arrow) and the marginal artery (black bowed arrow) join at the splenic flexure to supply the superior mesenteric artery via the middle colic artery (white open arrows). (*C*) Retrograde thoracic aortogram. The visualized superior mesenteric artery is again occluded distally after giving off a few jejunal branches (arrow). In sequential roentgenograms the opacification of the remaining superior mesenteric artery was accomplished by a large right branch of the middle colic artery. The ileocolic artery and the remainder of the vessel were subsequently visualized

Case VIII. This 31 year old Puerto Rican female entered the hospital for evaluation of hypertension. She had enjoyed good health until the age of 18 years, when she developed headaches, dizziness and palpitations. Hypertension was first recorded at that time.

Physical examination revealed a blood pressure of 180/80 mm. Hg, equal in both arms, and diminished femoral pulses. A loud systolic murmur was heard along the left sternal border.

Laboratory findings were: increased erythrocyte sedimentation rate of 36 mm./hr.; total protein 7.2 gm. per cent, with 47 per cent albumin. Electrophoresis disclosed marked elevation of γ globulin and mild elevation of β and $\alpha 2$ globulins. V.D.R.L. was nonreactive.

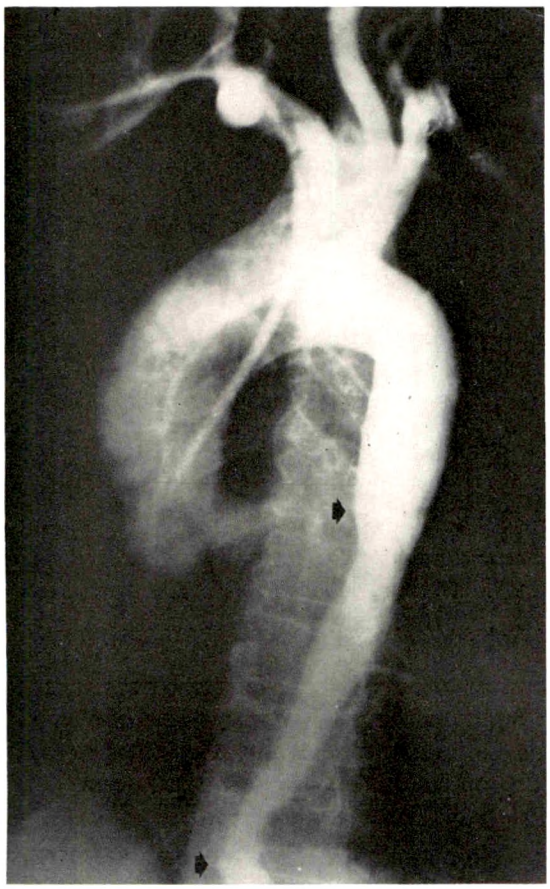


Fig. 6. Case VI. Thoracic aortogram shows that the ascending aorta and the arch are moderately dilated, whereas the descending portion is diffusely narrowed and tapers off gradually towards the diaphragm (arrows). This "rat-tail" narrowing has a grossly undulated contour. A shaggy luminal margin in the region of the severe constriction suggests secondary atherosclerosis. Moderate post-stenotic dilatation of the abdominal aorta was characterized by an abrupt transition to a normally appearing vessel. The aortic branches were not affected.

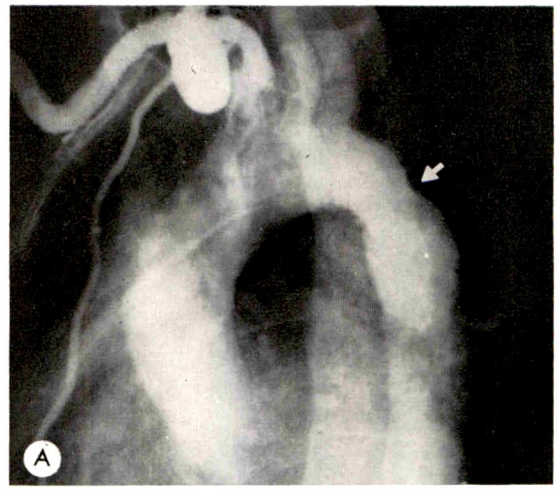


Fig. 7. Case VII. (*A*) Thoracic aortogram. Segmental narrowing of the isthmus (arrow). The brachiocephalic vessels are not affected.

At cardiac catheterization, no valvular abnormalities were noted. Aortography disclosed diffuse narrowing of the descending and abdominal aorta with a short segmental coarctation at the level of the diaphragm (Fig. 8, *A* and *B*).

At operation the wall of the coarcted region was markedly thickened with a fibrotic protrusion partially obstructing the lumen. Some atherosclerosis was present; however, it was not a prominent feature. After an endarterectomy a dacron patch graft was inserted over a distance of about 6 cm. in order to widen the aorta. A biopsy of the aortic wall was obtained. Following operation there was moderate symptomatic improvement with the blood pressure stabilized at 160/80 mm. Hg.

Specimens from endarterectomy showed nonspecific fibrosis and some atherosclerosis. Fragments of the aortic wall revealed fibrosis with a few elastic fibers and decreased amount of recognizable smooth muscle. There were foci of inflammatory cells composed of polymorphonuclear cells and lymphocytes.

ROENTGENOGRAPHIC AND ARTERIOGRAPHIC FINDINGS

Conventional roentgenography may frequently reveal vascular abnormalities sup-

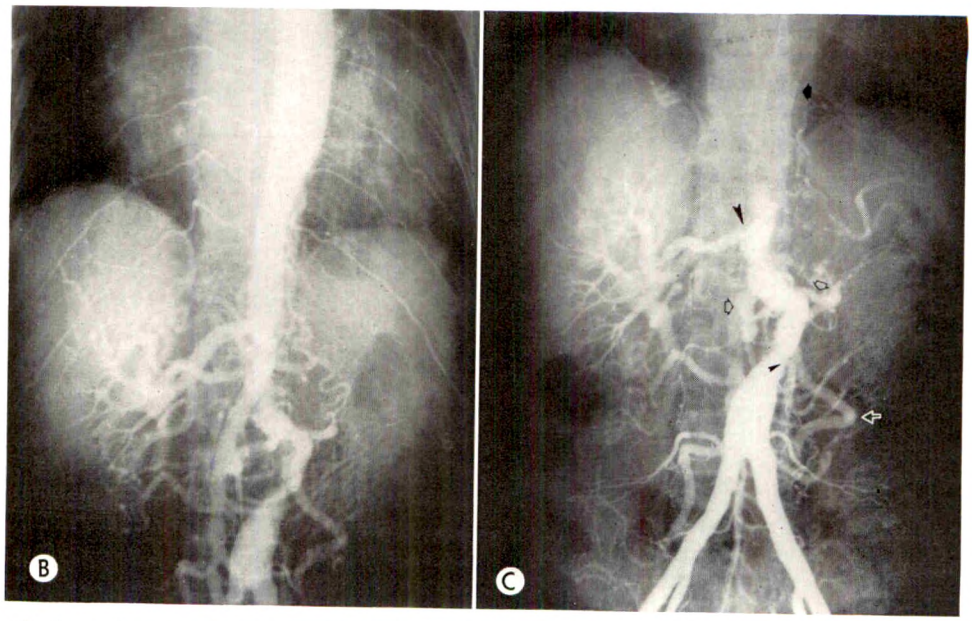


FIG. 7. (*B*) Smooth narrowing of the distal descending aorta is associated with marked coarctation of the abdominal part. The contour of the abdominal aorta is irregularly twisted with shaggy margins suggesting secondary atherosclerosis. (*C*) Below the level of coarctation, the distal abdominal aorta, the bifurcation, and the iliac arteries are unusually well preserved with smooth luminal margins. The right renal artery (large arrowhead) and the inferior mesenteric artery (small arrowhead) are stenosed at their orifices. The left renal artery is occluded at its orifice. A large left branch of the middle colic artery (black open arrows) and the left colic artery (white arrow) create a prominent collateral channel contributing to the supply of the inferior mesenteric artery. The narrowed descending aorta is outlined by linear calcifications (black closed arrow).

porting the clinical impression of Takayasu's arteritis.

Roentgenograms of the chest may show the ascending aorta to be prominent or dilated, whereas the descending aorta may display irregularity of its contour (Fig. 4). In coarctation of the aorta, rib notching may occasionally be observed[19] (Fig. 2*A*). Roentgenograms of the abdomen may disclose diminution in size of one or both kidneys, while intravenous urography will indicate its ischemic nature.

In the aorta, scarring, the sequela of arteritis, leaves its angiographically recognizable imprint in several patterns. A summary of regional involvement is outlined in Table 1.

The most common findings were solitary or multiple segmental narrowings. Both the linear extent and the degree of narrowing are variable. Frequently, a localized constriction is clearly demarcated with sharp luminal surface (Fig. 2*B*). Skipped areas of involvement are common. A localized narrowing of the isthmic region, for instance, may be associated with segmental constriction or aneurysmal dilatation of the more distal portions of the aorta (Fig. 5, *A* and *B*). Occasionally the entire descending and abdominal aorta is diffusely narrowed with superadded segmental constriction (Fig. 8, *A* and *B*). Dilatation of the ascending aorta is common.

An outstanding feature of thoracic coarctation is the predilection for the segment of severe constriction to be located at or slightly above the diaphragm (Fig. 3; 4; 6; and 8, *A* and *B*). In the elongated form of coarctation, the aorta has a wavy contour and tapers off gradually towards the diaphragm (Fig. 3). Despite gross irregularity of the wall, the proximal portion of the involved segment tends to be sharply delineated, whereas the constricted segment is

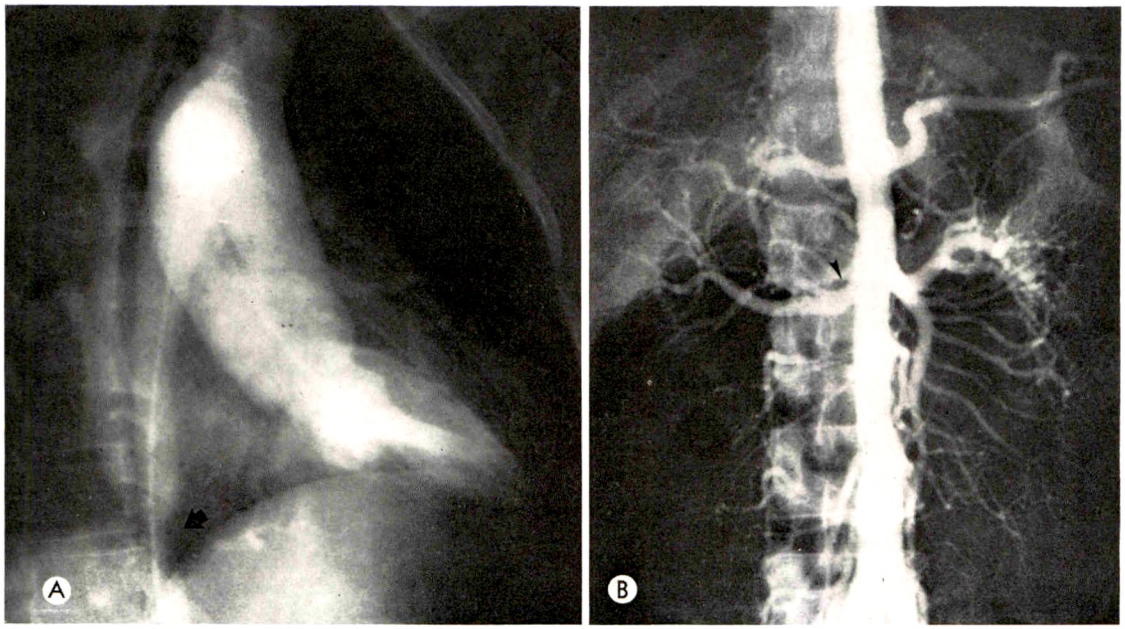


Fig. 8. Case VIII. (*A*) Left ventriculogram in right anterior oblique projection. Small hypertrophic left ventricular chamber. Marked dilatation of the ascending aorta and the arch is probably secondary to aortitis. Diffuse narrowing of the descending aorta with a short segmental constriction at the level of the diaphragm (arrow). (*B*) Diffuse narrowing of the abdominal aorta with grossly irregular contours. The origin of the right renal artery is slightly narrowed (arrow). The distal abdominal aorta, the bifurcation, and the iliac arteries appear normal.

Table I

SUMMARY OF REGIONAL INVOLVEMENT

Case No.	Sex and Age (yr.) at Admission	Ascending Aorta	Arch	Descending Aorta	Abdominal Aorta	Subclavian Artery (Left)	Subclavian Artery (Right)	Carotid Artery (Left)	Carotid Artery (Right)	Celiac Trunk	Superior Mesenteric Artery	Inferior Mesenteric Artery	Renal Artery (Right)	Renal Artery (Left)	Calcification
I	F 38			N	N	O		O	N		O				+
II	F 26		N		N	S	O	S			N	O	S	O	+
III	F 18	D	D	C											
IV	F 25	D		C	N	S	S						O		+
V	F 18	D	D	N	D	O					O		S	S	
VI	F 52	D	D	C											+
VII	F 75			N	C							S	S	O	+
VIII	F 31	D	D	N	N								N		

N = narrowing; C = coarctation; D = dilatation; S = stenosis; O = occlusion; + = calcification.

shaggy in outline (Fig. 4; and 6). This is especially true in older hypertensive patients in whom the appearance of secondary degenerative atherosclerosis can be anticipated. High blood pressure proximal to severe narrowing and the hemodynamic stress at the site of constriction are the accelerating factors. Distal to the site of coarctation, there is frequently an abrupt transition to a normally appearing aorta.

In a 75 year old patient, the abdominal aorta was severely contracted and twisted with irregular margins indicating secondary atherosclerosis (Fig. 7*B*). The distal abdominal aorta and the bifurcation, commonly involved in ordinary atherosclerosis[17] were conspicuously spared (Fig. 7*C*). This observation indicated a different etiologic process. Corroborative evidence of Takayasu's aortitis was further provided in this patient by the appearance of the descending aorta and the isthmic region (Fig. 7, *A* and *B*).

Recognition of aortic involvement is of decisive diagnostic importance. Aortography uncovered evidence of unsuspected aortitis in all patients. In some cases, angiographic findings were limited to the aorta only (Fig. 3; 6; and 8, *A* and *B*).

Review of the literature[9,11,12,15,17–19] and the characteristic aortographic features as illustrated in our cases produce a highly distinctive appearance of the diseased aorta. It has to be emphasized, however, that Takayasu's disease may apparently affect the major arterial branches without angiographically detectable involvement of the aortic wall.[20,23]

Arteritis of the aorta and its branches has been described in association with rheumatoid arthritis,[25] systemic lupus erythematosus,[16] and ankylosing spondylitis.[22] These rare reports lack sufficient arteriographic documentation to permit a valid comparison with Takayasu's arteritis.

Although premature atherosclerosis may cause occlusion or stenosis of aortic branches, it cannot duplicate the appearance of the aorta produced by Takayasu's disease.

Aortography disclosed unsuspected stenosis or occlusion of branch vessels in the majority of the patients. The characteristic sites and the appearance of occlusive arterial changes may frequently indicate the nature of the disease.

The subclavian arteries were frequently involved. Except for 1 instance where the subclavian artery was occluded at its orifice, the proximal aspect of the vessel remained patent. The site of occlusion was typically just beyond the thyrocervical trunk and the site of stenosis over the first rib (Fig. 1*A*; 2*A*; and 4).

A unique situation arises in the presence of bilateral subclavian artery obstruction in association with coarctation of the aorta. In such a case, a direct recording of the aortic root pressure may be the only means to obtain the actual level of the patient's hypertension (Case IV).

In 1 instance the left carotid artery had a long constriction of its distal portion (Fig. 2*A*). The affected area was sharply demarcated with minimal degree of irregularity of its lumen. This type of segmental stenosis is unique and when present is highly suggestive of Takayasu's arteritis.[6,31] In the presence of occlusion of the carotid arteries, the vertebral arteries tend to be large, serving as collateral channels (Fig. 1*A*; and 2*A*). In general, obliterative disease of the carotid arteries is confined to the proximal aspect of the vessels, but it may extend as far as the base of the skull,[8] and their outline is frequently markedly irregular.

The renal arteries were significantly affected in about a half of the patients. The involvement was usually bilateral. Occlusion of the renal arteries was observed at or in proximity to their orifices (Fig. 2*B*; and 7, *B* and *C*). Renal artery stenosis manifested itself as a short segmental constriction at the ostium with or without poststenotic dilatation (Fig. 5*B*; and 7*C*) or as a long narrowing throughout the entire length of the main renal artery (Fig. 2*B*). Due to excellent collateral circulation, function was preserved in all but the oldest patient.

Most of the patients with involvement of the abdominal aorta had associated obliterative disease of the renal and/or visceral branches.

Gastrointestinal complaints are uncommon in the chronic stage of Takayasu's arteritis[20,23] despite the frequently encountered obliteration of visceral branches.

The celiac trunk is reported to be only rarely involved in Takayasu's disease and none was involved in this series.

Stenosis or occlusion of the superior mesenteric artery was common (Fig. 2*B*; and 5*B*). When the vessel was occluded, excellent collateral flow reconstituted the distal portion. On one occasion the superior mesenteric artery was occluded at 2 sites; each occlusion was bridged sequentially by collaterals (Fig. 5, *B* and *C*).

Occlusion or stenosis of the inferior mesenteric artery was observed in 2 instances (Fig. 2*B*; and 7*C*).

Occlusive arteritis of Takayasu's type is apparently an insidious and gradual process. This feature combined with the relatively young age of the patients permits the development of the most abundant and variable collateral circulation as ever demonstrated angiographically. Detailed description of collateral pathways is, however, beyond the scope of this paper.

Pulmonary arteriography was performed in 1 patient (Fig. 2*C*) with evidence of involvement of the main and smaller peripheral arteries. It appears certain that arteritis of the pulmonary vessels is more common than generally appreciated.[14,21] In the severe form, pulmonary arteritis may lead to occlusion of the main branches.[14] Aneurysmal dilatation of the pulmonary arteries has been described.[9]

Total aortography is the definitive method for demonstration of the extent and sites of vascular abnormalities. As a diagnostic test it is of paramount importance. In each instance it uncovered evidence of unsuspected involvement of the aorta and its branches.

Angiographic diagnosis is rarely difficult. The characteristic sites of involvement, their configuration and multiplicity, allied with the clinical profile, indicate the nature of the disease.

SUMMARY

Takayasu's arteritis may affect all parts of the aorta leading to narrowing, coarctation or dilatation. The pulmonary arteries are involved in similar fashion.

Associated stenosis or occlusion of the subclavian, carotid, renal or abdominovisceral arteries is common.

The angiographic findings in 8 patients are illustrated and the clinical features described. The clinical symptoms are very variable and the laboratory findings are nonspecific.

Total aortography is of primary diagnostic importance, providing information not obtainable by any other method.

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REFERENCES

1. Ask-Upmark, E. On "pulseless disease" outside of Japan. *Acta med. scandinav.*, 1954, *149*, 161–178.
2. Ask-Upmark, E., and Fajers, M. C. Further observation on Takayasu's syndrome. *Acta med. scandinav.*, 1956, *155*, 275–291.
3. Barker, W. N., and Edwards, E. J. Primary arteritis of aortic arch. *Circulation*, 1955, *11*, 486–492.
4. Cheitlin, D. M., and Carter, B. P. Takayasu's disease. *A.M.A. Arch. Int. Med.*, 1965, *116*, 283–288.
5. Danaraj, J. T., and Ong, H. W. Primary arteritis of abdominal aorta in children; causing bilateral stenosis of renal arteries and hypertension. *Circulation*, 1959, *20*, 856–863.
6. Falicov, E. R., and Cooney, F. D. Takayasu's arteritis and rheumatoid arthritis. *A.M.A. Arch. Int. Med.*, 1964, *114*, 594–600.
7. Frövig, G. A., and Löken, C. A. Syndrome of obliteration of arterial branches of aortic arch due to arteritis. *Acta psychiat. et neurol. scandinav.*, 1951, *26*, 313–337.
8. Griffin, H. M. Reversed coarctation with vasomotor gradient: report of cardiovascular anomaly with symptoms of brain tumor. *Proc. Staff Meet. Mayo Clin.*, 1939, *14*, 561–565.

9. GOTSMAN, M. S., BECK, W., and SCHRIRE, V. Selective angiography in arteritis of aorta and its major branches. *Radiology*, 1967, *88*, 232–248.
10. HIRSCH, M. S., AIKAT, B. K., and BASU, A. K. Takayasu's arteritis: report of five cases with immunologic studies. *Bull. Johns Hopkins Hosp.*, 1964, *115*, 29–64.
11. INADA, K., SHIMIZU, H., and YOKOYAMA, T. Pulseless disease and atypical coarctation of aorta with special reference to their genesis. *Surgery*, 1962, *52*, 433–443.
12. JOFFE, N. Aortitis of obscure origin in African. *Clin. Radiol.*, 1965, *16*, 130–140.
13. JUDGE, D. R., CURRIER, D. R., GRACIE, A. W., and FIGLEY, M. M. Takayasu's arteritis and aortic arch syndrome. *Am. J. Med.*, 1962, *32*, 379–392.
14. KOZUKA, T., NOSAKI, T., SATO, K., and IHARA, K. Aortitis syndrome with special reference to pulmonary vascular changes. *Acta radiol.* (Diag.), 1968, *7*, 23–52.
15. KOZUKA, T., NOSAKI, T., SATO, K., and IHARA, K. Roentgenologic diagnosis of atypical coarctation of aorta. *Acta radiol.* (Diag.), 1966, *4*, 497–507.
16. LESSOF, H. M., and GLYNN, E. L. Pulseless syndrome. *Lancet*, 1959, *1*, 799–801.
17. MCKUSICK, A. V. Form of vascular disease relatively frequent in Orient. *Am. Heart J.*, 1962, *63*, 57–64.
18. MILLOY, F., and FELL, H. E. Elongate coarctation of aorta. *A.M.A. Arch. Surg.*, 1960, *80*, 759–765.
19. MORRIS, C. G., DEBAKEY, E. M., COOLEY, A. D., and CRAWFORD, E. S. Subisthmic aortic stenosis and occlusive disease. *A.M.A. Arch. Surg.*, 1960, *80*, 87–104.
20. NAKO, K., IKEDA, M., KIMATA, S., NIITANI, H., MIYAHARA, M., ISHIMI, Z., HASHIBA, K., TAKEDA, Y., OZAWA, T., MATSUSHITA, S., and KURAMOCHI, M. Takayasu's arteritis. *Circulation*, 1967, *35*, 1141–1155.
21. NASU, T. Pathology of pulseless disease. *Angiology*, 1963, *11*, 225–242.
22. PALOHEIMO, J. A., JULKUNEN, H., SILTANEN, P. A., and KAJANDER, A. Takayasu's arteritis and ankylosing spondylitis. *Acta med. scandinav.*, 1966, *179*, 77–85.
23. PALOHEIMO, J. A. Obstructive arteritis of Takayasu's type. *Acta med. scandinav.*, 1967, Suppl. 468, 1–42.
24. RIEHL, L. J., and BROWN, W. J. Takayasu's arteritis. *Arch. Neurol. & Psychiat.*, 1965, *12*, 92–97.
25. SANDRING, H., and GUNNAR, W. Aortic arch syndrome with special reference to rheumatoid arthritis. *Acta med. scandinav.*, 1961, *170*, 1–19.
26. SEN, K. P., KINARE, G. S., KULKARNI, P. T., and PARULKAR, B. G. Stenosing aortitis of unknown etiology. *Surgery*, 1962, *51*, 317–325.
27. SHIMIZU, K., and SANO, K. Pulseless disease. *J. Neuropath. & Clin. Neurol.*, 1951, *1*, 37–47.
28. STRACHAN, R. W. Natural history of Takayasu's arteriopathy. *Quart. J. Med.*, 1964, *33*, 57–69.
29. TAKAYASU, M. Case with peculiar changes in retinal vessels. *Acta Soc. Ophth. Jap.*, 1908, *12*, p. 554.
30. UEDA, H., ITO, I., OKADA, R., INOUE, G., YAMADA, H., MATSUYAMA, K., SAITO, S., and GONDAIRA, T. Aortic arch syndrome with special reference to pulseless disease and its variants. *Jap. Heart J.*, 1963, *4*, 224–232.
31. WICKBOM, I. Arteriography in brachiocephalic arteritis. *Acta radiol.*, 1957, *48*, 321–329.
32. YAMADA, H., HARUMI, K., OHTA, A., NOMURA, T., OKADA, R., and ISHII, M. Aortic arch syndrome with cardiomegaly and aortic calcification. *Jap. Heart J.*, 1961, *2*, 538–548.

ANGIOGRAPHY IN ERGOTISM*

REPORT OF TWO CASES AND REVIEW OF THE LITERATURE

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ERGOT intoxication, or ergotism, is a condition that has existed for centuries. Originally it occurred in epidemic form as a result of ingestion of rye (cereal or bread), that had become contaminated with the ergot fungus, *Claviceps purpurea*.[5] The disease, typified by either gangrene or convulsions, was of such wide spread proportions in the Middle Ages, that European hospitals designated entire wards for its treatment.[5]*

The epidemic form of the disease has been rare in the 20th century due to introduction of quality food controls in most countries. The last reported epidemic occurred in France in 1951 when a baker, attempting to circumvent a grain tax, bought a load of contaminated, bootleg flour. The bread he made from such flour resulted in a local disaster: "Over two hundred people were more or less simultaneously affected and many ran delirious in the streets. Four people died. Ducks, chickens, dogs, cats, goats and even fish to whom bread had been given sickened and died."[5]

Today, the preponderance of cases of ergotism occurs in sporadic form as the result of ingestion of ergot-derivative drugs, used primarily for the treatment of either migraine headache or postpartum hemorrhage.

Recently, the authors have had occasion to see 2 patients with ergotism that developed secondary to ergotamine tartrate ingestion, taken to ward off symptoms of migraine headache. The angiographic findings of these 2 patients will be presented and compared to those already reported in the literature.

* These patients, so afflicted, described their gangrenous pains as "fire-like" and, since the St. Anthony's Hospitals treated so many of them, ergotism came to be known as "St. Anthony's fire."[5]

REPORT OF CASES

CASE I. *History*. E.F.K. is a 43 year old white female admitted to St. Joseph's Infirmary on November 25, 1967 with a 24 hour history of cold, numb and tingling sensations in her right foot and ankle. She described a 1 week history of aching cramps and tightness in her right calf upon walking and, occasionally, even at rest. The patient had a 1 year history of headache, diagnosed 4 years ago as "tension headache," following an evaluation which included normal skull series, electroencephalogram, bilateral carotid arteriograms and cerebrospinal fluids. The patient had been on ergotamine tartrate suppositories for 2 weeks for "migraine headache."

Physical Examination. The patient had both a cold right foot and a cold lower one-third of the right leg. Pulses were palpable in the right and left femoral arteries, where bruits were heard, but no pulses were palpable distal to that in the right leg. The presence of left popliteal and dorsalis pedis pulses was doubtful.

Angiography. On November 27, 1967, translumbar aortography was performed which showed bilaterally symmetric irregularities, minimal in degree, involving both external iliac arteries. This was said to resemble "fibromuscular hyperplasia, but could be stationary arterial waves." There was a 4 to 5 cm. narrowing of the right distal superficial femoral and proximal popliteal arteries. The left posterior tibial artery was not well seen. On December 2, 1967, repeat angiography, following subsidence of the patient's symptoms, showed a normal distal right superficial femoral artery. The previously described changes, compatible with a fibromuscular hyperplasia, were still present (Fig. 1, *A–C*).

Clinical Course. Treatment, consisting of elevation of the head of the bed, heparinization and discontinuation of the ergotamine tartrate, was begun; the patient completely recovered.

CASE II. *History*. I.N.M. is a 50 year old white female who was first admitted to St.

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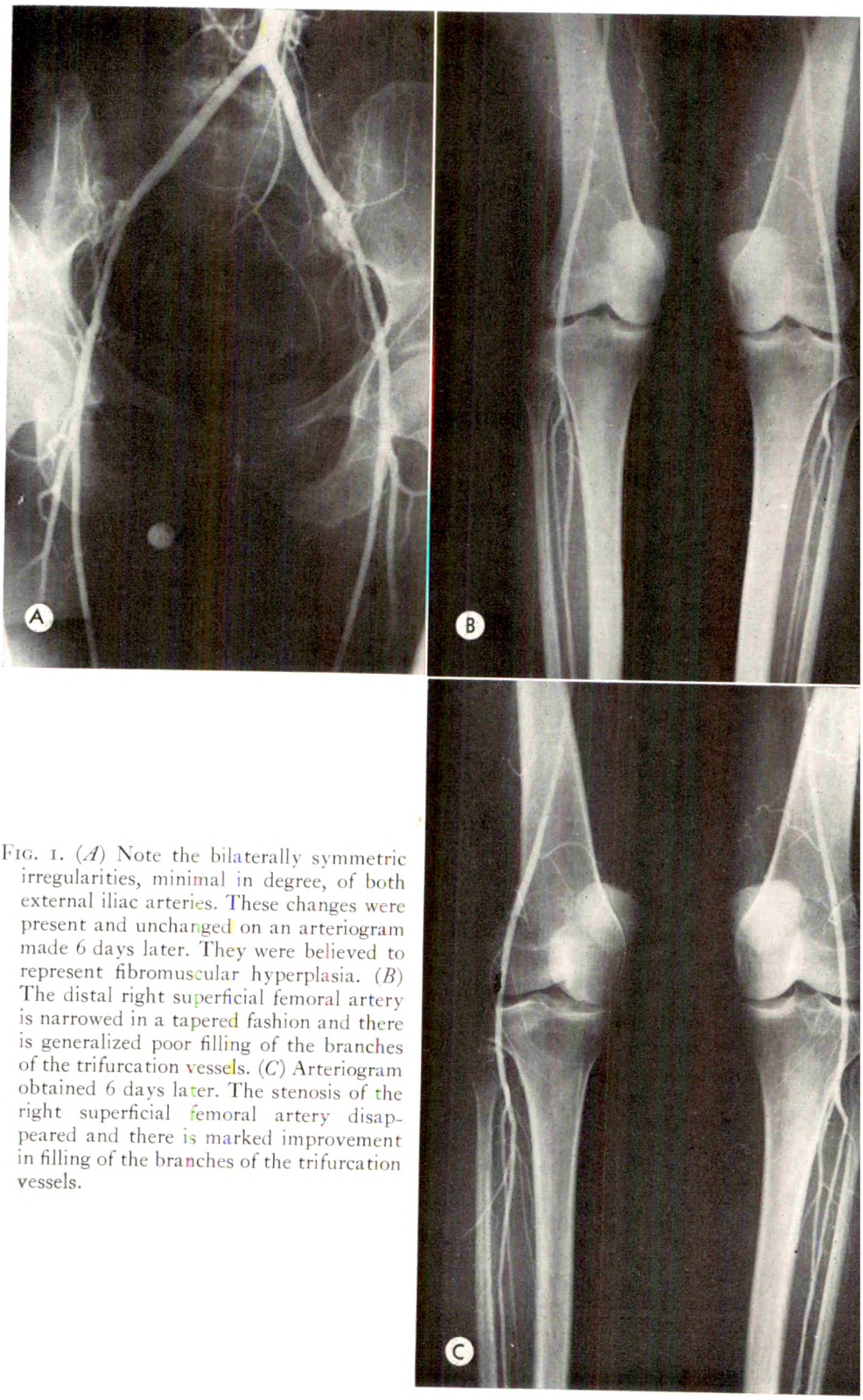


FIG. 1. (*A*) Note the bilaterally symmetric irregularities, minimal in degree, of both external iliac arteries. These changes were present and unchanged on an arteriogram made 6 days later. They were believed to represent fibromuscular hyperplasia. (*B*) The distal right superficial femoral artery is narrowed in a tapered fashion and there is generalized poor filling of the branches of the trifurcation vessels. (*C*) Arteriogram obtained 6 days later. The stenosis of the right superficial femoral artery disappeared and there is marked improvement in filling of the branches of the trifurcation vessels.

Joseph's Infirmary on June 16, 1969 with a 48 hour history of painful, cold, and numb feet which had a bluish discoloration. This was accompanied by paresthesias of both hands. The patient had been hospitalized elsewhere 2 days previously, at which time the clinical suspicion was vasospasm secondary to Sansert (methylsergide) ingestion for migraine prophylaxis. She had been treated there with low spinal anesthesia on 2 different occasions. The patient, who had a 10 year history of migraine headache, had been taking Sansert daily for the past 2 or 3 years.

Physical Examination. Examination revealed cold feet and legs with marked discoloration and mottling. Gangrene of the feet and toes developed. Only femoral pulses were present. Patient could not move her right leg at the time of admission.

Angiography. Angiography performed on the day of admission showed marked narrowing of both superficial femoral arteries and of all the peripheral vessels. The right was more severely involved than the left. The trifurcation vessels were threadlike on the left and nonvisible on the right. There was also some question of decreased caliber of the aorta, the common iliac and the common femoral arteries (Fig. 2, *A–D*).

Therapy. Antibiotic and heparin therapy were initiated. Bilateral dilatation of the femoral, popliteal and tibial arteries along with bilateral lumbar sympathectomy was performed on June 17, 1969. Gangrenous changes persisted in the right toes and left heel in spite of treatment. Soft tissue infection and osteomyelitis were a continued problem in the right foot, such that multiple procedures involving incision and drainage of the right foot were performed. These were ineffective and a two-stage Symnes amputation became necessary in late 1970. The left foot so far has been saved, but the patient continues to have osteomyelitis involving the os calcis; she was last seen on September 2, 1971 for this; future amputation may

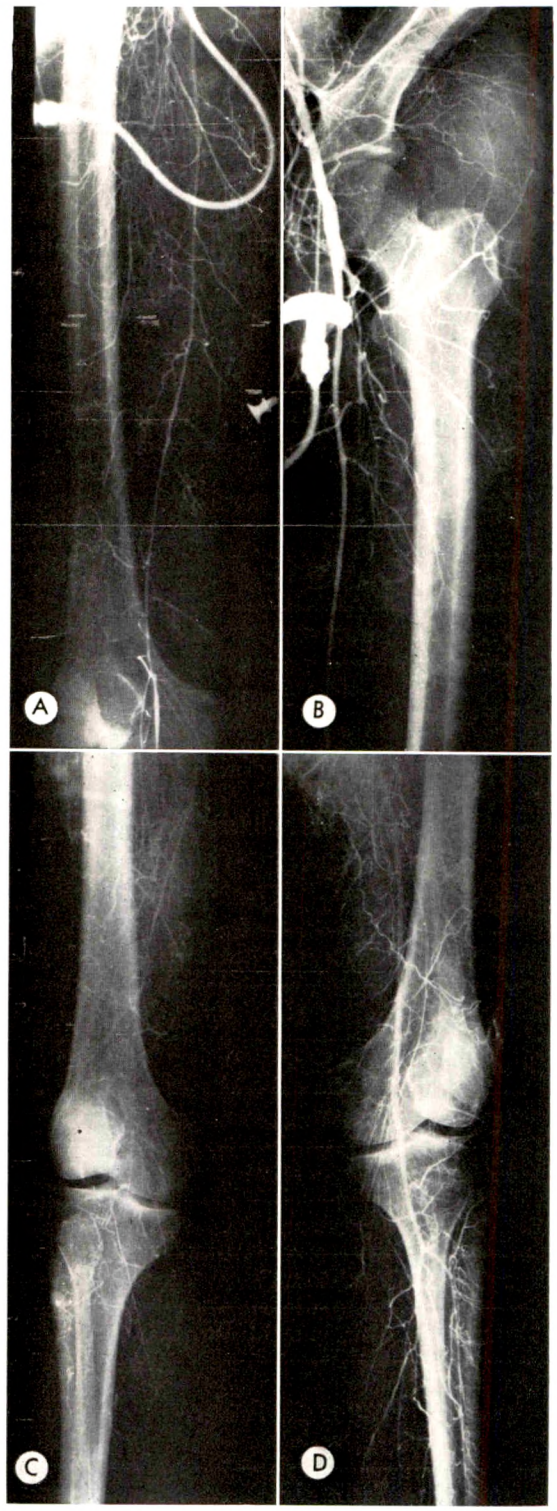


Fig. 2. Views of (*A*) right and (*B*) left thighs show marked narrowing of both superficial femoral arteries and all peripheral branches. The trifurcation vessels are threadlike on the left (*D*) and nonvisible on the right (*C*). Multiple tiny collateral vessels are present about the left knee.

become necessary. The patient's clinical course has been complicated by viral hepatitis (thought to be secondary to her numerous blood transfusions), and by urinary tract infection.

Comment. These 2 cases are typical of ergotamine tartrate intoxication in both their clinical symptoms and their angiographic findings. They demonstrate the extremes of the clinical picture from total recovery to gangrene and its ramifications.

CLINICAL FEATURES

Nineteen reports of the angiographic findings in patients with ergotism appear in the literature (Table 1). The 2 cases in this report bring the total to 21. The average age of the patients is 38.4 years. Only 4 patients are men; this reflects the higher incidence of migraine headache in women. The drug taken in every case was ergotamine tartrate and the indications for the

TABLE 1

SUMMARY OF FINDINGS IN CASES COLLECTED FROM THE LITERATURE

Year	Author	Location of Symptoms		Angiographic Findings		
		UE	LE	Spasm	Collaterals	Thrombi
1936	Yater and Cahil[32]		+	+	+	
1961	Young and Humphries[33]	+		+		
1962	Johnsson[18]		+	+		
			+	+	+	
1965	Engle and Silvertssen[7]		+	+	+	
		+	+	+		
1965	Kramer *et al.*[21]	+		+	+	+
1966	Glazer *et al.*[10]		+	+		
1966	Tator and Heimbecker[28]	+		+		
1967	Fagerberg *et al.*[8]	+	+	+	+	
1967	Pader[24]		+	+		
1968	Ahlgren *et al.*[1]		+	+	+	
1968	Haynes and Davis[13]		+	+	+	
1969	Bertho *et al.*[3]	+		+		
1970	Fedotin and Hartman[9]		+	+		
1970	Sutton and Preston[25]		+	+		
			+	+		
1970	Yao *et al.*[31]		+	+		
1971	Syme[27]		+	+	+	

UE = upper extremity; LE = lower extremity.

drug therapy were: migraine or "cluster" headache in 19; pruritus of jaundice in 1; and an unknown cause in 1. The symptoms in this group were due to schemia: pallor, coldness, numbness and pain. Three patients had gangrenous changes at the time initially seen; 2 of these patients eventually needed amputations and the third patient had gangrenous changes which regressed following therapy. Of these 21 patients, 15 had only lower extremity involvement, 3 had only upper extremity involvement and 3 had both upper and lower extremities involved (Table 1).

The symptoms were of acute onset in 11 cases, subacute (between 1 and 3 months) in 2, and chronic in 7. Drug dosage varied from as little as 10 mg. to very large quantities taken over a 20 year period. The drug had been taken for less than a 1 month period by 6 patients and for more than a year by 14 patients. Of these 14, 5 had recently increased their dosage of the drug. One case had no mention of the duration of drug therapy.

ANGIOGRAPHIC FINDINGS

Three findings may be noted angiographically, in these patients: (1) arterial spasm; (2) collateralization; and (3) thrombi formation.[21]

1. The arterial spasm is usually bilateral and symmetric with abrupt or smooth areas of narrowing The narrowing may be quite "thread-like" and is usually more severe distally. Total occlusion may occur. In the lower extremities, the spasm may begin in the common iliac arteries, but is more common in the superficial femoral, the deep femoral, the popliteal and the trifurcation arteries. The areas of spasm may be focal rather than generalized, but this is less common. In any case, the vasospasm may be reversible and usually responds to drug withdrawal, although other measures may be necessary.

2. Collateral formation is more common in patients who have taken the ergot drugs chronically. Of the 2 patients who developed collaterals while having been on ergot drugs for only short periods of time, 1 was 64 years of age and developed gangrene requiring amputation,[31] while the other, 54 years of age, developed infarction of the biceps muscle.[21] Collaterals may disappear following withdrawal of the medication.[8,13]

3. Thrombi formation has been rarely reported.[21,28] It is believed to be related to a combination of factors: (a) vascular stasis distal to the areas of spasm; and (b) direct toxic effect upon the endothelium of the vessels.[21] The thrombi present as small filling defects within the vascular lumina. It is not known if thrombi formation is reversible once therapy is instituted.

DISCUSSION

1. SYMPTOMS

Most cases of ergotism today occur in individuals who are taking ergot-derivative drugs for migraine headache, but cases have been reported in obstetric patients taking the drugs for postpartum hemorrhage or for abortion inducement. Recently, accidental poisoning of neonates occurred in a newborn nursery, where vitamin K and ergonovine maleate vials were stored side by side: the vitamin K was mistakenly given to 5 mothers and the ergonovine maleate (0.20 mg.) to 5 infants.[6] The children developed skin lesions with purpura, cyanosis and blebs of the extremities, groin and genitalia. Four recovered and 1 died. Young children have taken overdoses of these drugs; in such cases, peritoneal dialysis, along with other therapeutic measures may be particularly effective.[19]

Ergotism, the sporadic type, may occur in 1 of 3 temporal forms: (1) as the result of *chronic* ingestion of therapeutic dosages of ergot drugs; (2) as the result of *acute* ingestion of *excessive* amounts of ergot drugs; or (3) as the result of *acute* ingestion of *small* amounts of ergot drugs in individuals hypersensitive to them.

The symptoms of ergot intoxication may appear soon after ingestion; however, patients may take the medications for many years before symptoms occur. It is more

common for the overdosed or hypersensitive patients to develop the symptoms immediately or acutely. As little as 10 mg. of ergotamine tartrate, given over a 6 day period, has caused ergotism in 1 hypersensitive individual.[9] Contrast this with patients who have taken 1 to 2 mg. tablets or suppositories of ergotamine tartrate for 5 to 10 years, without ever developing symptoms. It is true, however, that most patients who have taken ergot drugs on a chronic basis and who develop the symptoms of ergotism, have had a recent increase in dosage of the medication. Nonetheless, individual response to these drugs varies greatly.[18]

Maximal "safe" dosages of ergotamine tartrate have been described as: (1) orally, 6 mg. per attack; (2) intramuscularly, 2 mg. or 2 ml. per week; and (3) rectally, 4 suppositories (2 mg./per week or 2 suppositories per attack, if not more than 2 are taken a week).[32]

II. PHARMACOLOGY

Ergot drugs have 2 principal pharmacologic effects: as an alpha-adrenergic blocker and as a vasoconstrictor, mainly a peripheral vasoconstrictor.[8]

Hence, either neurologic or circulatory symptoms can be clinically present in ergotism. The neurologic symptoms consist of convulsions and/or coma. The circulatory symptoms are a result of the vasoconstriction and are principally located in the extremities, where the ischemia can lead to coldness and numbness and can be followed by gangrene if medication is not stopped. Lower extremities are much more commonly affected than upper extremities. Occasionally, arterial systems other than those located in the extremities can be involved (see the discussion on Systemic Changes).

III. AGGRAVATING CONDITIONS

Certain conditions aggravate the vasoconstrictive effects of ergot drugs: febrile states; sepsis; malnutrition; thyrotoxicosis; pregnancy; hepatic disease; renal disease; hypertension; coronary artery disease; and peripheral vascular disease.[15] At one time, ergotamine tartrate was given to patients for the pruritus associated with jaundice.[20,29] It soon became evident, however, that this should not be done, since liver failure seemed to worsen the possibility of gangrene development in these patients.

A case of an unusual nature was reported recently:[4] an 18 year old pregnant Nigerian female developed pregangrene of the extremities following ingestion of a native root given her by a witch doctor. Samples of the root revealed it to contain alkaloids with ergot-like properties. Obstinacy on the part of the witch doctor prevented complete study of the root contents. Pregnancy may have heightened the effects of the "drug" in this patient.

IV. DRUGS THAT POTENTIATE THE EFFECTS OF ERGOT

Certain other drugs may potentiate the harmful effects of ergot. Oral contraceptives have been implicated in numerous cases.[8] Also, smoking is said to aggravate the vasospastic qualities of these drugs.[8] Recently, a case report implicated the broad spectrum antibiotic, triacetylolandomycin, in the precipitation of acute ergotism.[14] In theory, TAAM may cause "a mild cholestatic and hepatitic reaction" and, since ergotamine tartrate undergoes some metabolic change in the liver, the ergotamine tartrate was metabolized less effectively than normal in this patient, thus creating a higher serum level. Perhaps, in future cases of ergotism, potentiation of ergot effect by other drugs should be considered more carefully.

V. SYSTEMIC EFFECTS OF ERGOT DRUGS

It was mentioned earlier that systemic as well as peripheral ischemic changes may be found in patients taking ergot-derivative drugs. The other arterial system most commonly reported involved after the iliac and subclavian is the coronary.[11] Both angina and myocardial infarction have been reported. Most of the cases of infarction have had predisposing disease—hy-

perthyroidism, syphilitic aortitis, aortic valve deformities, atherosclerosis of the coronary ostia and angina—but at least 1 case of well-documented infarction developed in a patient with no known predisposing disease.[11] The case was that of a 35 year old white female who was given a 2.0 mg. dose of ergotamine tartrate orally and 0.5 mg. of the same intramuscularly for migraine headache. Twenty minutes later she developed classical symptoms of coronary insufficiency. Subsequent electrocardiographic and enzymatic changes confirmed the clinical impression of acute myocardial infarction.

Other systems can be involved with resultant organ gangrene—carotid,[25] renal,[9] etc.[12] Of particular note is the case reported by Fedotin and Hartman[9] in which a patient developed the classical angiographic findings of ergotism in the lower extremities. In addition, he developed changes within the renal arteries which were indistinguishable from fibromuscular hyperplasia. An arteriogram, obtained 10 days later, showed the changes within the renal arteries to have reverted completely to normal. This case brings to mind Case 1 in this report, which demonstrated changes inseparable from those of fibromuscular hyperplasia within the external iliac arteries. An arteriogram obtained 6 days later showed these changes still present. Whether these changes were due to the presence of an associated fibromuscular hyperplasia or to an abnormality caused by the ergotism is not known. It would have certainly been interesting to have an even later arteriogram on this patient.

SUMMARY

Two cases of ergotism with peripheral ischemic changes, well demonstrated by angiography, are reported.

These cases are compared symptomatically and angiographically to the other cases in the literature.

Three angiographic findings appear to be present within the extremity arteries of patients with ergotism: (1) arterial vasospasm; (2) collateralization; and (3) thrombi formation.

The vasospasm and collateralization may be reversible with adequate treatment.

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REFERENCES

1. Ahlgren, I., Haeger, K., and Nylander, G. Imminent gangrene of leg after ergot poisoning. *Angiology*, 1968, *19*, 354–361.
2. Allen, E. V., Barker, N. W., and Hines, E. A., Jr. Raynaud's phenomenon and allied vasospastic conditions. In: Peripheral Vascular Diseases. W. B. Saunders Company, Philadelphia, 1962, p. 148.
3. Bertho, E., Ratte, J., and Jean, J. D. Iatrogenic ergotism; report of case. *Angiology*, 1969, *20*, 455–459.
4. Colbert, D., and Lysaght, F. Threatened gangrene in pregnancy caused by root alkaloids. *J. Irish M. A.*, 1970, *63*, 446–471.
5. Devitt, R. E., Chater, E., and Colbert, D. S. Ergot poisoning. *J. Irish M. A.*, 1970, *63*, 441–445.
6. Edwards, W. M. Accidental poisoning of newborn infants with ergonovine maleate; a lesson applicable to all delivery rooms. *Clin. Pediat.*, 1971, *10*, 257–260.
7. Engle, I., and Silvertssen, E. Ergotism due to therapeutic doses of ergotamine tartrate. *Am. Heart J.*, 1965, *70*, 665–670.
8. Fagerberg, S., Jorulf, H., and Sandberg, C. G. Ergotism, arteriospastic disease and recovery studied angiographically. *Acta med. scandinav.*, 1967, *182*, 769–772.
9. Fedotin, M. S., and Hartman, C. Ergotamine poisoning producing renal arterial spasm. *New England J. Med.*, 1970, *283*, 518–520.
10. Glazer, G., Myers, K. A., and Davies, E. R. Ergot poisoning. *Postgrad. M. J.*, 1966, *42*, 562–568.
11. Goldfischer, J. D. Acute myocardial infarction secondary to ergot therapy; report of case and review of literature. *New England J. Med.*, 1960, *262*, 860–863.
12. Grollman, A. Pharmacology and Therapeutics. Fifth edition. Lea & Febiger, Philadelphia, 1962, p. 315.
13. Haynes, C. D., and Davis, M. E., Jr. Ergotism: report of case with localized arteriographic changes in femoral vessels. *Angiology*, 1968, *19*, 199–202.
14. Hayton, A. C. Precipitation of acute ergotism by triacetylolandomycin. *New England M. J.*, 1969, *69*, 42–50.

15. HEMINGWAY, D. L. Ergot poisoning with multisystem involvement. *Mil. Med.*, 1965, *130*, 1103–1106.
16. HUDGSON, P., and HART, J. A. Acute ergotism; report of case and review of literature. *M. J. Australia*, 1964, *2*, 589–591.
17. INEL, J., and MUNE, O. Peripheral arterial insufficiency in ergotism; review of literature and foot plethysmographic studies in ergotamine poisoning. *Vasc. Dis.*, 1967, *4*, 159–166.
18. JOHNSSON, K. A. Angiography in two cases of ergotism. *Acta radiol.*, 1962, *57*, 280–284.
19. JONES, E. M., and WILLIAMS, B. Two cases of ergotamine poisoning in infants. *Brit. M. J.*, 1966, *2*, 466–467.
20. KATZ, A. F., and MASSRY, S. G. Arteriospasm after ergotamine tartrate in infectious hepatitis. *A.M.A. Arch. Int. Med.*, 1966, *118*, 62–64.
21. KRAMER, R. A., HECKER, S. P., and LEWIS, B. I. Ergotism; report of case studied arteriographically. *Radiology*, 1965, *84*, 308–311.
22. LIEBER, E. Galen on contaminated cereals as cause of epidemics. *Bull. Hist. Med.*, 1970, *44*, 332–345.
23. LONDON, M., MAGORA, F., and ROGEL, S. Acute ergot poisoning; case report. *Angiology*, 1970, *21*, 565–567.
24. PADER, E. Leriche syndrome in patient with prolonged and continuous use of ergot derivatives; review of vascular effects of ergot. *Vasc. Dis.*, 1967, *4*, 380–388.
25. PROHULT, J., FORSBERG, O., and HELLSTROM, R. Case of multiple arterial thromboses after oral contraceptives and ergotamine. *Acta med. scandinav.*, 1967, *181*, 453–456.
26. SUTTON, D., and PRESTON, B. J. Angiography in peripheral ischemia due to ergotism; report of two cases. *Brit. J. Radiol.*, 1970, *43*, 776–780.
27. SYME, J. Ergotamine-induced peripheral arterial spasm: clinical and angiographic diagnosis. *Australasian Radiol.*, 1971, *15*, 45–51.
28. TATOR, C. H., and HEIMBECKER, R. O. Unilateral arm ischemia due to ergotamine tartrate. *Canad. M. A. J.*, 1966, *95*, 1319–1321.
29. THOMPSON, W. S., JR. Prolonged vasoconstriction due to ergotamine tartrate; report of case with recovery, with objective evaluation of vascular findings. *Arch. Int. Med.*, 1950, *85*, 691–698.
30. WHELTON, M. J., ALLAWAY, A., and STEWART, A. Ergot poisoning in acute hepatic necrosis. *Gut*, 1968, *9*, 287–289.
31. YAO, S. T., GOODWIN, D. P., and KENYON, J. R. Case of ergot poisoning. *Brit. M. J.*, 1970, *3*, 86–87.
32. YATER, W. M., and CAHIL, J. A. Bilateral gangrene of feet due to ergotamine tartrate used for pruritus of jaundice; report of case studied arteriographically and pathologically. *J.A.M.A.*, 1936, *106*, 1625–1631.
33. YOUNG, J. R., and HUMPHRIES, A. W. Severe arteriospasm after use of ergotamine tartrate suppositories. *J.A.M.A.*, 1961, *175*, 1141–1145.

VALUE OF OBLIQUE PROJECTIONS IN TRANSLUMBAR AORTOGRAPHY*

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THE treatment most likely to benefit patients with arteriosclerotic insufficiency to the lower limbs is surgical widening of stenotic or occluded segments of the arterial tree. The success of such operative therapy, which may consist of grafting, vein by-pass or endarterectomy, depends on the accurate display of the arterial system of the legs.

When there is occlusion or stenosis of the superficial femoral artery, the profunda femoris artery maintains the circulation to the lower limbs through collaterals. In this situation, should a stenosis or occlusion of the origin of the profunda femoris artery occur, the collateral circulation may become inadequate. Operative reconstruction of the profunda femoris artery may, however, lead to relief of symptoms.[4,6,11]

Buttock claudication due to bilateral stenoses or occlusions of the internal iliac arteries is rare as the sole manifestation of arterial insufficiency and is usually associated with leg claudication due to generalized peripheral vascular disease. Localized surgery to these arteries alone is thus rarely indicated. Buttock claudication may, however, be confused with pain due to neurologic or orthopedic diseases, so that the demonstration of stenosed internal iliac arteries may be important in the differential diagnosis.

For these reasons, it is important to show the origins of both the profunda femoris arteries and the internal iliac arteries clearly.

As the internal iliac arteries arise posteromedially and the profunda femoris arteries posterolaterally, the origins of these arteries are often partly obscured in the conventional frontal projection.

This paper compares the display of the origins of the superficial femoral, the profunda femoris, the internal iliac, and the external iliac arteries in the left and right anterior oblique projections (L.A.O. and R.A.O.) with that obtained in the conventional posteroanterior projection (P.A.).

A simple method of obtaining oblique views with translumbar aortography is described.

MATERIAL

Fifty consecutive patients with intermittent claudication due to arteriosclerosis were examined by translumbar aortography in posteroanterior and both anterior oblique projections (Fig. 1, *a–c*). Ten of these patients were excluded from the series either because the aorta or the iliac arteries on one side were totally occluded or because one or more of the 3 series of roentgenograms was technically unsatisfactory.

METHOD

The patients are first examined in the conventional posteroanterior projection. An A.O.T. serial film changer with moving table top is used and the whole of the arterial system to the lower limbs demonstrated, using 60 ml. of sodium iothalamate 70 per cent.[11]

Five films are then exposed in each of the anterior oblique projections, the field including the lower abdominal aorta and the bifurcation of the femoral arteries. Twenty-five ml. of contrast material is injected for each series. The films are exposed at the same rate as for the posteroanterior run.

Five ml. of contrast medium is injected to check the needle position for the P.A. view, and similar injections are made after turning the patient for each oblique projection to ensure that the needle has not be-

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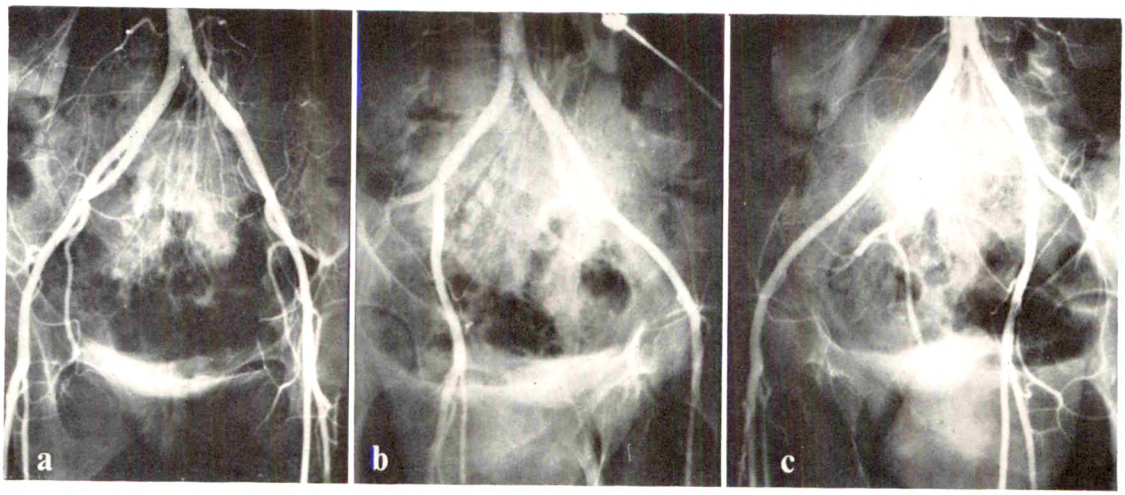


FIG. 1. (*a*) Posteroanterior (P.A.) projection. Note overlapping of origin of left internal iliac artery and both profunda femoris arteries. (*b*) Right anterior oblique (R.A.O.) projection. The origins of the left internal iliac and right profunda femoris arteries are clearly shown. The origins of the right internal iliac artery and left profunda femoris artery are obscured. (*c*) Left anterior oblique (L.A.O.) projection. The origins of the right internal iliac artery and left profunda femoris and superficial femoral arteries are clearly shown. The left internal iliac artery origin is obscured. The origin of the right profunda femoris artery is slightly overlapped.

come displaced during positioning: a total of 125 ml. of contrast material is used in all.

To obtain the oblique projections, the patient is turned onto the left or right side on a balsa wood block of triangular cross section. The block, 120 cm. long, is placed beneath the patient to support the shoulders and pelvis (Fig. 2; and 3). Corrugated rubber strips along the edges of the under surface of the block prevent its slipping on the table top. It was found from previous experience before the main series of patients was examined, that an obliquity of about 30° gave the best over-all demonstration of the origins of the iliac and femoral artery

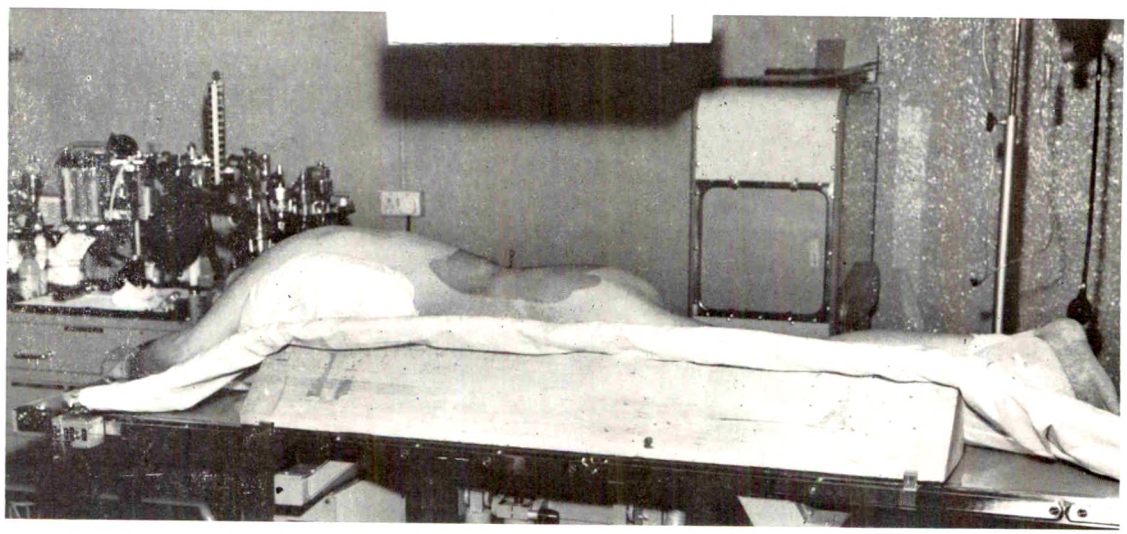


FIG. 2. Patient positioned for R.A.O. projection. Note triangular balsa wood block beneath the patient and translumbar needle in position.

bifurcations. This degree of obliquity is obtained with a block with a 45° slope.

RESULTS

The roentgenograms from each of the 3 series were read and the demonstration of the origins of the internal and external iliac arteries, and the origins of the profunda femoris and superficial femoral arteries was graded as diagnostic or non-diagnostic. If there was any doubt, the demonstration was considered non-diagnostic. The presence of stenoses or occlusions at the origins of these arteries was recorded for each series.

For both left and right internal and external iliac arteries and for the profunda femoris and superficial femoral arteries, the number of origins diagnostically or non-diagnostically shown in each projection was totalled and expressed as a percentage (Table 1; Fig. 7).

The internal artery was demonstrated significantly better in the contralateral anterior oblique projection (*left* 90 per cent, *right* 78 per cent; Fig. 1, *b* and *c*; and Fig. 4*b*) than the frontal projection (*left* 65 per cent, *right* 53 per cent). The number of diagnostic demonstrations in the ipsilateral anterior oblique projections were considerably less (*left* 23 per cent, *right* 23 per cent; Fig. 1, *b* and *c*). There was little difference in the demonstration of the origins of the external iliac arteries in the 3 projections, being shown well in all (*left*—R.A.O. 93 per cent, P.A. 93 per cent, L.A.O. 90 per cent; *right*—R.A.O. 80 per cent, P.A. 88 per cent, L.A.O. 100 per cent; Fig. 1, *a–c*).

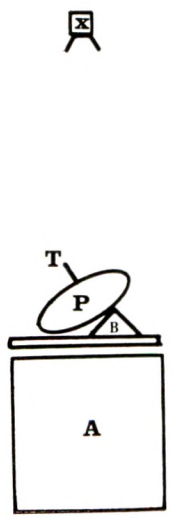


Fig. 3. Diagram to show position of patient and x-ray equipment for oblique translumbar aortography. X = x-ray tube. T = translumbar needle. P = patient. B = balsa wood block. A = A.O.T. serial film changer.

Table 1

DIAGNOSTICALLY DEMONSTRATED ARTERY ORIGINS IN POSTEROANTERIOR AND OBLIQUE PROJECTION EXPRESSED AS PERCENTAGES

Artery origin	P.A. (per cent)	R.A.O. (per cent)	L.A.O. (per cent)
Left internal iliac	65	90	22.5
Right internal iliac	52.5	22.5	77.5
Left external iliac	92.5	92.5	90
Right external iliac	87.5	80	100
Left profunda femoris	37.5	25	95
Right profunda femoris	40	77.5	42.5
Left superficial femoral	65	55	95
Right superficial femoral	62.5	97.5	62.5

The origin of the profunda femoris artery was shown best in the ipsilateral oblique (*left* 95 per cent, *right* 78 per cent; Fig. 1, *b* and *c*; Fig. 5*b*; and Fig. 6*b*) and poorly in the contralateral oblique (*left* 25 per cent, *right* 43 per cent; Fig. 1, *b* and *c*) and posteroanterior projections (*left* 38 per cent, *right* 40 per cent; Fig. 1*a*; Fig. 5*a*; and Fig. 6*a*).

The demonstration of the origins of the superficial femoral arteries showed a similar pattern to the profunda femoris, but there was less difference between the projections (*left*—R.A.O. 55 per cent, P.A. 65 per cent, L.A.O. 90 per cent; *right*—R.A.O. 98 per cent, P.A. 63 per cent, L.A.O. 63 per cent).

Thus the left anterior oblique projection (L.A.O.) is the best projection to show the right internal iliac artery and left profunda

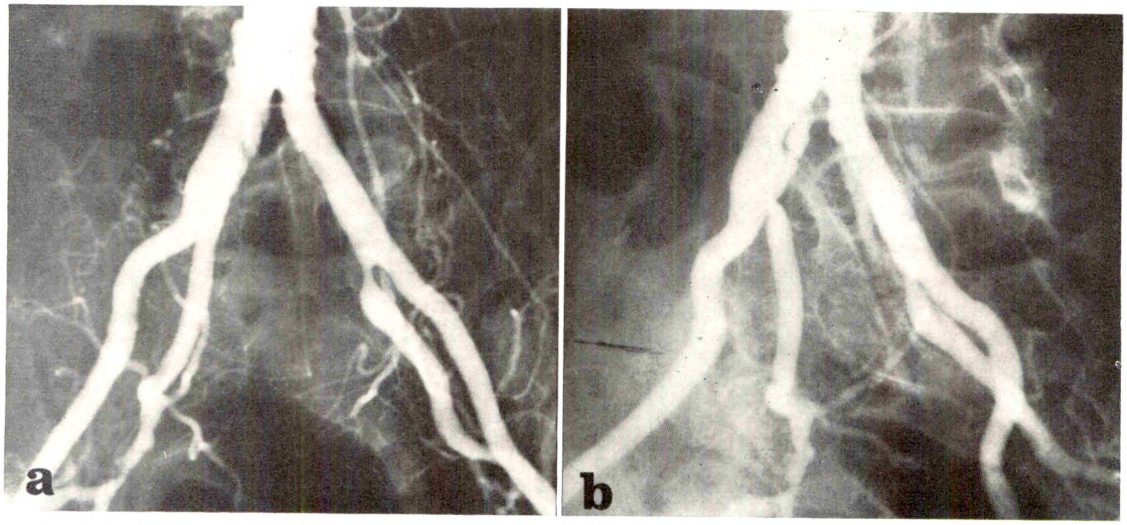


FIG. 4. (*a*) P.A. projection. The origin of the right internal iliac artery is obscured. There is an obvious stenosis of the left internal iliac artery. (*b*) L.A.O. projection. Note stenosis at the origin of the right internal iliac artery.

femoris origins and the right anterior oblique (R.A.O.) the best to show the left internal iliac artery and the right profunda femoris origins. Similarly, the right anterior oblique (R.A.O.) projection is the best view to show the origin of the left in-

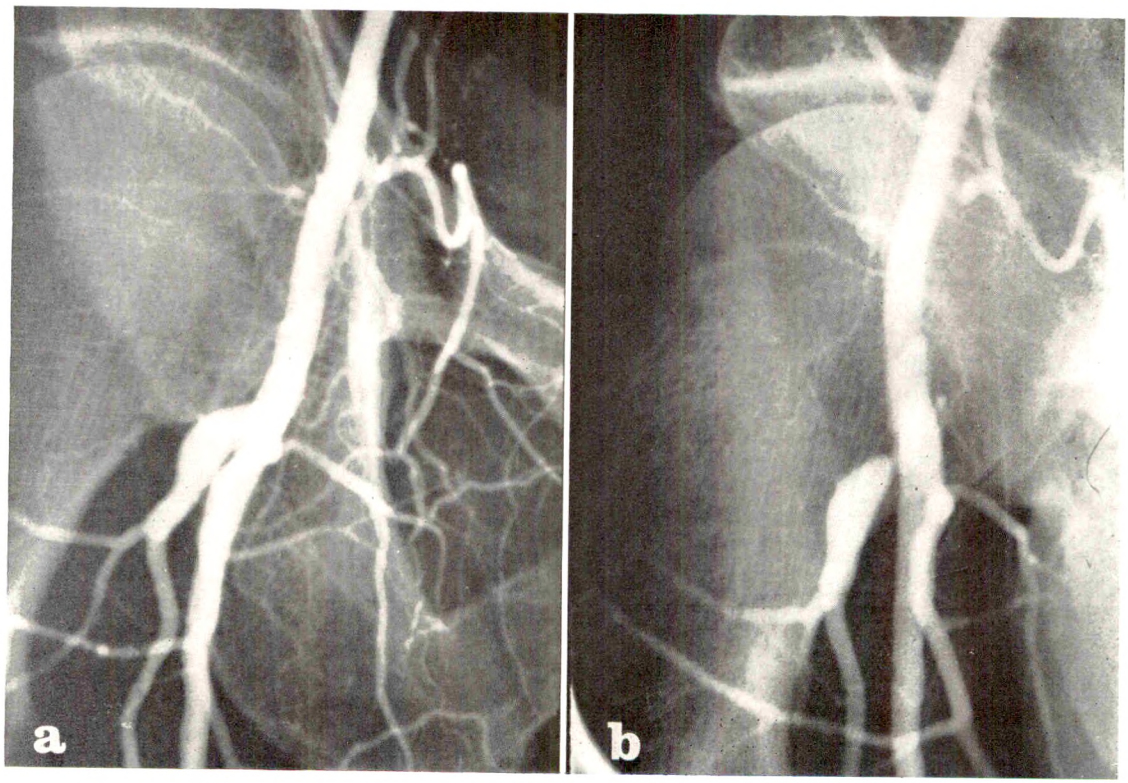


FIG. 5. (*a*) P.A. projection. The origin of the profunda femoris artery is obscured. (*b*) R.A.O. projection. Note stenosis at the origin of the profunda femoris artery.

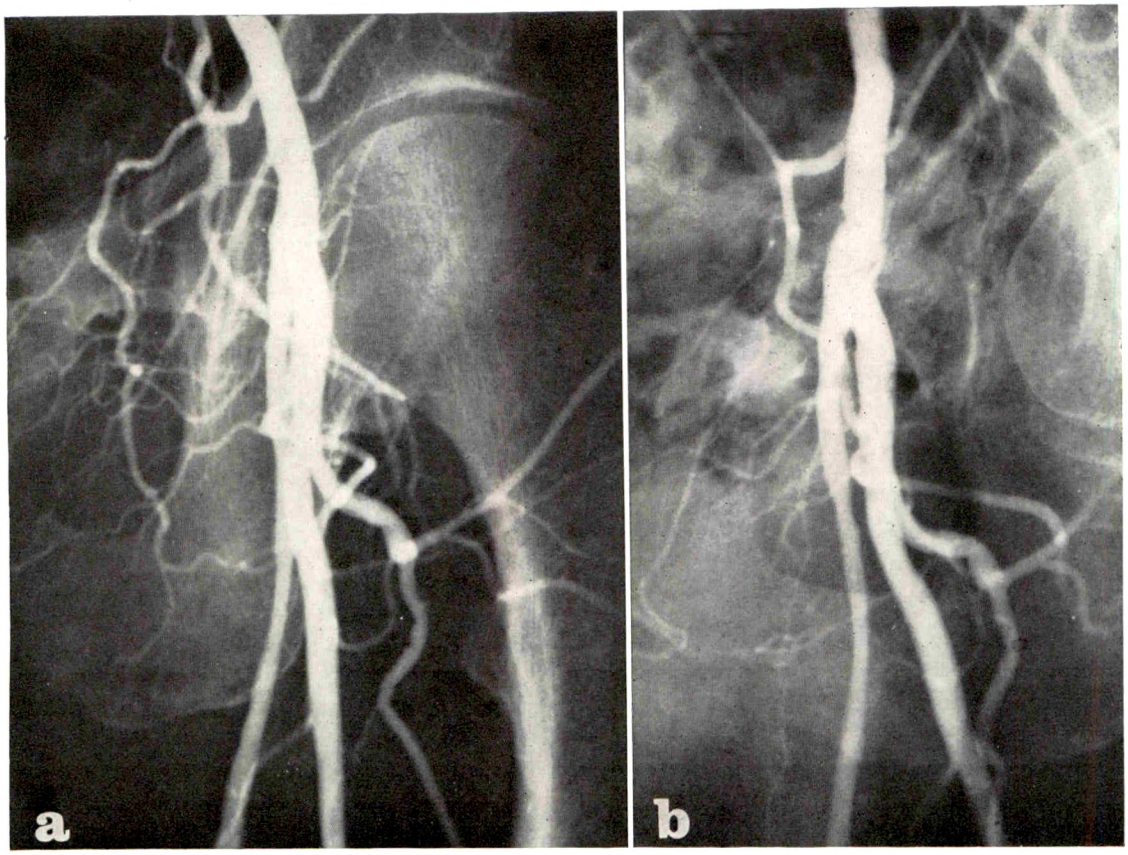


Fig. 6. (*a*) P.A. projection. The origins of the superficial femoral and profunda femoris arteries are partly overlapped. (*b*) L.A.O. projection. Note that the origins of the superficial femoral and profunda femoris arteries are clearly shown.

ternal iliac artery and the right profunda femoris artery.

These results are summarized in Table 1 and Figure 7.

STENOSES

The number of stenoses at the origin of the internal and external iliac arteries and the superficial femoral and profunda femoris arteries were also added up, but the numbers are too small to be meaningfully expressed as percentages.

In the left internal iliac artery 8 stenoses were shown in the straight projection, while the R.A.O. roentgenograms showed these 8 and an additional 2. In the right internal iliac artery 5 stenoses were shown on the P.A. roentgenograms, while 8 were visualized by the use of the L.A.O. projection. The 3 stenoses of the left external iliac arteries were shown diagnostically in all 3 projections. There were no right external iliac stenoses in the series.

No stenoses were shown in the left profunda femoris arteries in the P.A. projections, but 2 were revealed by the L.A.O. views. In the right profunda femoris artery only 1 stenosis was demonstrated in the P.A. projection, while 3 were shown by the use of the R.A.O. view.

There were no superficial femoral artery stenoses in the series.

Thus more stenoses at the origins of the internal iliac and profunda femoris arteries were shown in oblique projections than in the conventional straight views.

These results are shown in Table II.

There were 2 total occlusions at the origins of the left internal iliac artery, 1 at the origin of the left superficial femoral ar-

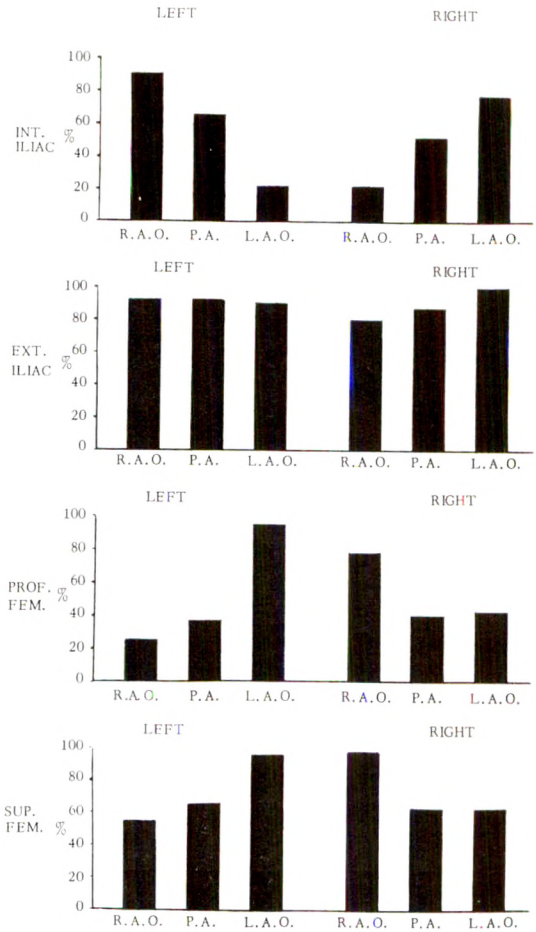


FIG. 7. Histogram showing number of diagnostic demonstrations of the origins of the internal iliac, external iliac, profunda femoris and superficial femoral arteries expressed as percentages.

tery and 1 at the origin of the external iliac artery. These were all diagnostically shown in the 3 projections and have not therefore been listed separately.

DISCUSSION

We have found translumbar aortography safer to use in patients with arteriosclerotic disease of the aorta and its branches than femoral catheterization, which can lead to occlusion in narrowed and diseased arteries. Nevertheless, the conclusions drawn from this paper would apply equally to both techniques except that since femoral artery catheterization is carried out with the patient supine, the positions used would be anteroposterior and left and right posterior oblique.

No complications due to either the translumbar technique or to the contrast medium were encountered in this series.

The total volume of contrast medium used was about 125 ml. in each patient. It must be admitted that the use of a large volume must increase the concentration of contrast medium entering the visceral and spinal arteries. With our technique the needle puncture was made below the renal arteries and none of our patients had a total aortic occlusion. Most of the serious complications, such as renal failure[1–3,7,9] and paraplegia[5,8] occurred with the older contrast media (acetrizoate and diodone) and few complications of this nature have been reported using the more modern media.[7]

This slightly increased risk of complications from the larger volume of contrast material needed for additional oblique projections is outweighed by the greater diagnostic information obtained concerning the bifurcations of the common iliac and common femoral arteries.

TABLE II

NUMBER OF STENOSES SHOWN ON STRAIGHT AND OBLIQUE ROENTGENOGRAMS

Artery origin	Straight	R.A.O.	L.A.O.	No. missed on P.A.
Left internal iliac	8	10	4	2
Right internal iliac	5	2	8	3
Left external iliac	3	3	3	—
Right external iliac	—	—	—	—
Left profunda femoris	—	—	2	2
Right profunda femoris	1	3	3	2
Left superficial femoral	—	—	—	—
Right superficial femoral	—	—	—	—

Moving a patient with a translumbar needle in the back could lead to displacement of the needle, which would not be the case with the catheter method. In none of our patients, however, did the needle become displaced during positioning, as the needle appeared to be held firmly in the muscles of the lumbar region.

This series, therefore, indicates that the use of oblique projections in translumbar aortography is safe. The best view to show the origin of an internal iliac artery is the contralateral anterior oblique, and to show the origin of the profunda femoris artery the ipsilateral anterior oblique. More stenoses at the origins of these arteries were shown with the use of the appropriate oblique than the conventional frontal projection.

It is, therefore, suggested that both anterior oblique projections should be used in addition to the posteroanterior view in all patients undergoing translumbar aortography for arteriosclerotic vascular disease.

SUMMARY

Left and right anterior oblique projections in addition to a posteroanterior view of the bifurcations of the common iliac and common femoral arteries were used at translumbar aortography in 40 consecutive patients with arterial insufficiency to the lower limbs.

The origins of the profunda femoris arteries were shown diagnostically in the appropriate oblique projection in a higher percentage of patients (77.5–95 per cent) compared with the conventional frontal view (37.5–40 per cent).

The origins of the internal iliac arteries were shown diagnostically in the appropriate oblique more frequently (77.5–90 per cent) than with the straight projection (52.5–65 per cent).

The external iliac artery origin was shown diagnostically in all 3 projections.

Stenoses were shown more often in an oblique than in the straight projections.

Total occlusions were equally well shown in the 3 projections.

The profunda femoris origin is best shown in the ipsilateral anterior oblique and the internal iliac artery, especially in the contralateral oblique projection.

No complications were encountered in the series.

It is suggested that oblique projections of the common iliac and common artery bifurcations should be used routinely in translumbar aortography carried out for the investigation of arteriosclerotic disease of the lower limbs.

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REFERENCES

1. Abrams, H. L. Radiologic aspects of operable heart disease: hazards of retrograde thoracic aortography. *Radiology*, 1957, *68*, 812–823.
2. Abrams, H. L. Angiography. J. & A. Churchill, Ltd., London, 1961, pp. 163–171.
3. Ansell, G. National survey of radiological complications: interim report. *Clin. Radiol.*, 1968, *19*, 175–191.
4. Beales, J. S. M., Adcock, F. A., Frawley, J. S., Nathan, B. E., McLachlan, M. S. F., Martin, P., and Steiner, R. E. Radiological assessment of disease of profunda femoris artery. *Brit. J. Radiol.*, 1971, *44*, 854–859.
5. Djindjian, R., and Faure, C. Spinal cord complications of aortography. *J. belge de radiol.*, 1967, *50*, 207–213.
6. Frawley, J. E., Rosengarten, D. S., and Martin, P. Profundaplasty. *Angiopatias* (Brasil), 1970, *10*, 41.
7. Grainger, R. G. Complications of cardiovascular radiological investigations. *Brit. J. Radiol.*, 1965, *38*, 201–215.
8. Hare, W. Damage to spinal cord during translumbar aortography. *J. Fac. Radiologists*, 1957, *8*, 258–266.
9. McAfee, J. G. Survey of complications of abdominal aortography. *Radiology*, 1957, *68*, 825–838.
10. Martin, P., Renwick, S., and Stephenson, C. On surgery of profunda femoris artery. *Brit. J. Surg.*, 1968, *55*, 539–542.
11. Thomas, M. L., and Fletcher, E. W. L. Large volume translumbar aortography in aortic occlusion. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *109*, 541–548.

A REFINEMENT IN TRANSLUMBAR CATHETERIZATION: USE OF THE J-TIP MOVABLE CORE GUIDE WIRE*

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DURING the past 2 years we have performed 157 translumbar aortographies for various indications, but mostly for peripheral vascular disease. We have followed the technique described by Amplatz[1] and Stocks *et al.*,[4] using the needle, teflon sleeve and guide wire combination. The recommended guide wire has a curved tip with a radius of 15 mm. and does not have a movable core* (Fig. 1*A*).

Not infrequently, however, we have encountered some difficulty in advancing the guide wire cephalad from our point of entry in the aorta. The problem is mostly due to arteriosclerotic plaques and kinks in the aorta and sometimes the wire will move caudad or enter the celiac or superior mesenteric artery.

We have solved this problem by using a 50 cm. long, 0.035 inch, 3 mm. radius, J-tip movable core guide wire* (Fig. 1*B*). The use of the safety J-tip guide wire was described by Judkins *et al.*,[3] for tortuous and atheromatous vessels and the movable core, J-tip guide wire by one of us (S. H. C.[2]) for passing a catheter above an abdominal aortic aneurysm from the femoral route.

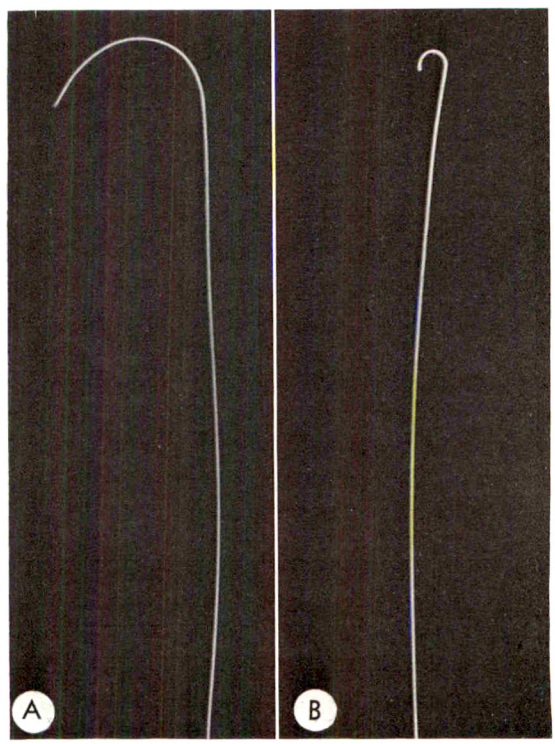


FIG. 1. (*A*) A 15 mm. radius fixed core guide wire recommended for use with the teflon sleeve technique of translumbar aortography. (*B*) A 3 mm. radius movable core guide wire which we have found to be more satisfactory in most cases.

TECHNIQUE

The curved tip of the 0.035 inch J-tip movable core guide wire is straightened by either pushing the inner core wire to the end or by the use of a small plastic tubing which is supplied with the wire. This is done to facilitate the insertion of the tip into the metal hub of the teflon sleeve. Following puncture of the aorta, the pointed stylet is removed and the metal cannula with its teflon sleeve is adjusted to obtain a good pulsatile back flow. The metal cannula is then removed leaving the teflon sleeve in the aortic lumen. The special guide wire is threaded into the teflon sleeve and advanced cephalad gently under fluoroscopic control. If an obstruction is encountered the core of the wire is pulled back further, so that the leading part of the wire becomes very flexible. The more the inner core is pulled out the more flexible the leading end of the guide wire becomes. This feature is very useful in overcoming tortuosity and by-passing plaques which may otherwise

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prevent the advance of a less flexible guide wire.

In some instances the difficulty in advancing the guide wire is not due to arteriosclerotic plaques or aortic tortuosity. The teflon sleeve which is slightly shorter than the metal cannula may still lie intramurally even though the tip of the cannula is already in the aortic lumen. To remedy this, we recommend that the metal cannula with the teflon sleeve, but without the sharp pointed stylet, be advanced gently until the tip hits the opposite aortic wall. The metal cannula is then removed and the teflon sleeve adjusted until a good pulsatile back flow of blood is obtained. The J-tip movable core guide wire is then inserted as described above.

SUMMARY

We have found the use of the J-tip movable core guide wire a further improvement in the technique of catheter translumbar aortography.

It is very useful in overcoming aortic tortuosity and arteriosclerotic plaques that may otherwise arrest the advance of a less flexible guide wire.

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REFERENCES

1. Amplatz, K. Translumbar catheterization of abdominal aorta. *Radiology*, 1963, *81*, 927–931.
2. Cornell, S. H. Refinement in catheter angiography of abdominal aortic aneurysms: J-tip movable core guide wire. *Brit. J. Radiol.*, 1969, *42*, 227–229.
3. Judkins, M. P., Kidd, H. J., Frische, L. H., and Dotter, C. T. Lumen-following safety J-guide for catheterization of tortuous vessels. *Radiology*, 1967, *88*, 1127–1130.
4. Stocks, L. O., Halpern, M., and Turner, A. F. Complete translumbar aortography: teflon sleeve technique. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *107*, 835–839.

A DEFLECTOR CATHETER APPROACH TO THE ABDOMINAL AORTA

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PERCUTANEOUS catheterization of the abdominal aorta via the axillary-subclavian artery route is often difficult and time consuming.[2,4,5] The conventional curved catheter or tip deflector methods are effective, but some centers report a failure rate as high as 50 per cent among the experienced angiographer.[5]

The purpose of this communication is to present a simplified technique for negotiating the tortuous left subclavian (or innominate) artery-transverse arch junction with easy placement of the catheter into the descending thoracic aorta. This is a technique that is easily mastered by the trained angiographer, as well as the inexperienced.

MATERIAL AND METHOD

The material for this report is comprised of 20 patients. There are 15 males and 5 females. Their ages range between 52 and 75 years.

Indications for the percutaneous axillary approach were severe occlusive disease or tortuosity of the distal aorta and iliac arteries to the degree that the femoral artery approach was not tenable.

After the initial development of this approach, the majority of the manipulative aspects were performed by physicians in their early stage of angiographic training.

TECHNIQUE

The method described herein involves the use of a tip deflector set-up and any catheter with a hook-tail configuration.[1] This hook-tail catheter with its leading, blunt or curved configuration when introduced into the transverse arch prevents the catheter from entering the brachiocephalic arteries. In addition, there is less tendency for atherosclerotic plaques to become dislodged or impede the passage of the catheter.

The crucial maneuvers are as follows: (1) the catheter is allowed to pass freely from the subclavian artery into the ascending aorta, from either axillary artery approach (Fig. 1*a*); (2) a Cook 0.45, 0.38, or 0.25 deflector (depending on the size of catheter) is inserted to the tip of the catheter (Fig. 1*a*); (3) the deflector is maximally deflected, *stabilized*, and the catheter is payed out over it (Fig. 1*b*); this is achieved by holding the deflector handle in the deflected position with one hand and pushing the catheter forward over the deflector wire with the other hand—the relatively rigid deflector will result in the catheter proceeding at 180° to the original course (Fig. 1*b*); (4) approximately 10 cm. of the catheter is payed out (Fig. 1*b*); (5) the catheter and deflector are retracted as a unit until the deflector wire is in the immediate proximity to the origin of the left subclavian (or innominate) artery (Fig. 1*c*); (6) the catheter can then be further advanced over the deflector wire into the descending aorta (Fig. 1*d*); and (7) finally, the deflector is removed and the injection can be initiated.

If a *selective* catheter is required, a standard 145 cm. guide wire can be passed into the original catheter, this catheter removed, and a selective catheter advanced over the guide wire into the descending aorta.

COMMENT

The catheterization of the abdominal aorta and its main branches in patients with severe atherosclerosis is a difficult task.

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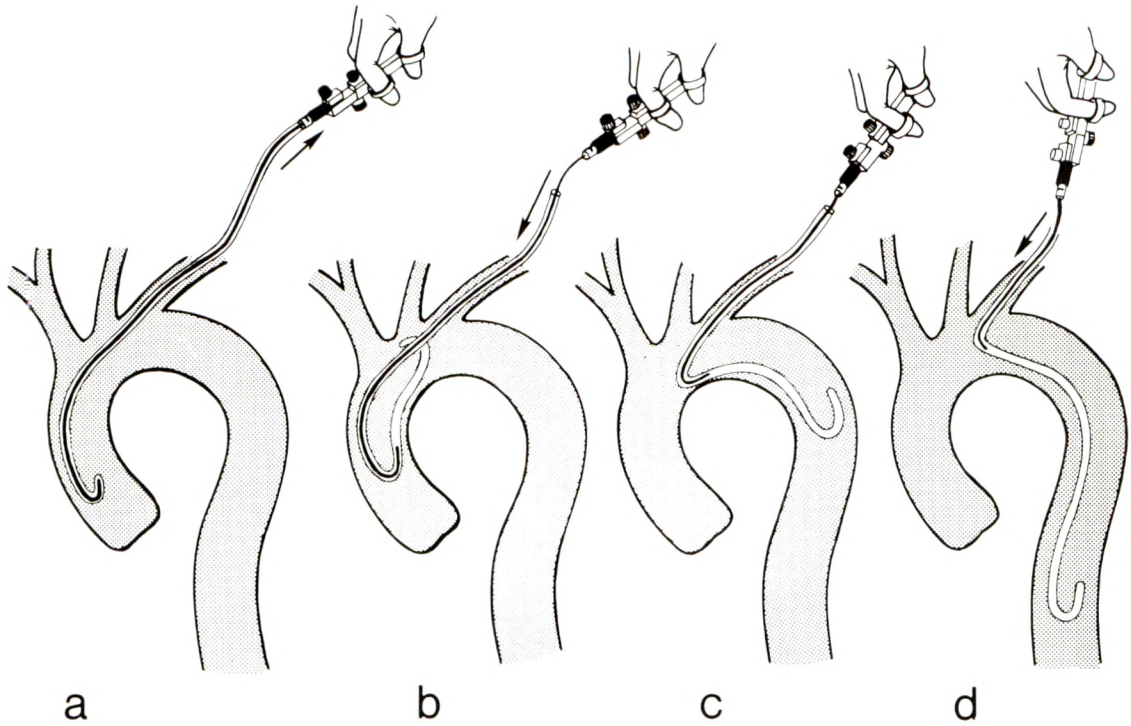


Fig. 1. Schematic drawing showing the steps in the deflector catheter approach to the descending aorta. (*a*) Catheter and guide wire have been advanced into the ascending aorta. Note that the tip of the guide wire is inserted to the tip of the catheter. (*b*) The deflector is maximally deflected, anchored, and the catheter payed out over the deflector. Note that the radius of the catheter tip prevents entry into the brachiocephalic arteries. (*c*) The catheter and deflector are pulled back. It is important to maintain the deflector tip in its deflected position. (*d*) The catheter can be advanced over the deflector into the descending aorta.

Even in the hands of the experienced, the failure rate is relatively high.[2,4,5] In fact, Thomas and Fletcher[5] in a recent communication advocated translumbar aortography as an alternate approach.

One of the major problems with the conventional axillary artery approach to abdominal aortography is that the guide wire or catheter cannot be negotiated into the descending thoracic aorta. This may occur because the wire or catheter becomes imbedded in or impeded by a plaque on the lesser curvature of the arch (Fig. 2). The relation of the subclavian artery to the arch results in repeated passage into the ascending aorta. Some angiographers take advantage of this phenomenon by deflecting the curved wire off the aortic valve.[3] This however, may result in a wire passing retrograde into the left ventricle or the catheter will not follow the wire into the descending aorta once the initial curve over the aortic valve is made.

Because of these difficulties and the necessity to retain selective abilities when performing abdominal aortography (which the translumbar approach militates against), the technique described herein was developed. It is a simple maneuver requiring little experience. We have found that this technique has readily been taught to and adapted by trainees. Since adopting this approach, we have not failed to catheterize the abdominal aorta either in a main stream or selective type examination.

A theoretic advantage should be reduction in the incidence of embolization, either cerebral or peripheral. This should occur because: (1) manipulation in and around the great vessels is reduced; and (2) the

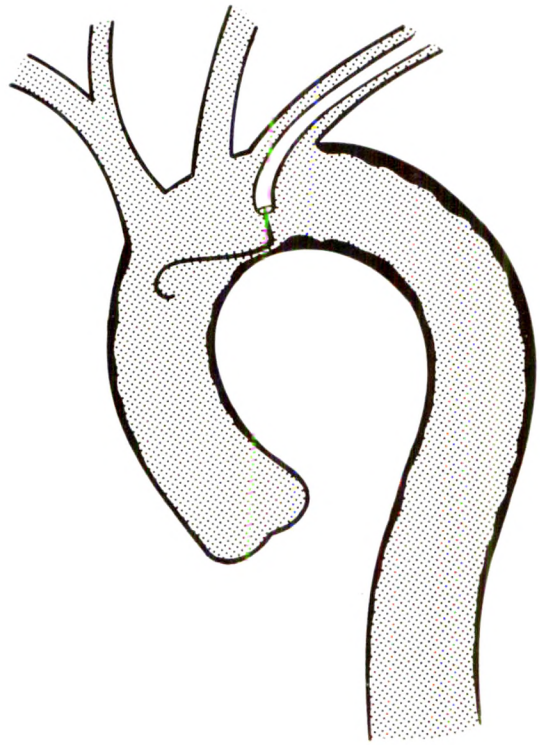


Fig. 2. Schematic representation of an atherosclerotic and elongated aorta with the typical course of the catheter via the left subclavian aorta. This illustrates the difficulty encountered by the operator when the conventional guide wire and catheter technique are used. This shows the guide wire being deflected by plaques into the ascending aorta rather than the descending aorta.

amount of time the catheter is *in situ* is decreased. The latter is manifest by less clot forming on the external surface of the catheter.

SUMMARY

This paper deals with a deflector catheter approach to the abdominal aorta.

The basic features are: (1) a Cook deflector: and (2) any hook-tail catheter.

The manipulative aspects which have allowed us to catheterize the abdominal aorta via the axillary arteries without failure in 20 successive patients are described.

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REFERENCES

1. Boijsen, E., and Judkins, M. P. Hook tail "closed-end" catheter for percutaneous selective cardioangiography. *Radiology*, 1966, *87*, 872–877.
2. Hanafee, W. Axillary artery approach to carotid, vertebral, abdominal aorta, and coronary angiography. *Radiology*, 1963, *81*, 559–567.
3. Miller, R. E., and Chau, G. T. Multiple curve guide wire for aortic catheterization. *Radiology*, 1969, *92*, 1569–1570.
4. Roy, P. Percutaneous catheterization via axillary artery: new approach to some technical roadblocks in selective arteriography. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1965, *94*, 1–18.
5. Thomas, M. L., and Fletcher, E. W. L. Large volume translumbar aortography in aortic occlusion. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *109*, 541–548.

"MINI-CATHETER" TECHNIQUE FOR FEMORAL RUN-OFF AND ABDOMINAL ARTERIOGRAPHY*

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THE high incidence of occlusive vascular disease together with an increasing number of specially trained radiologists and vascular surgeons have resulted in a greater demand for arteriography in both university and community hospitals. Angiography for abdominal and peripheral vascular disease comprises the majority of special procedures performed in most radiology departments.

Many methods for performing peripheral vascular arteriography have been advocated. The initial procedure for studying the abdominal aorta and peripheral arteries was translumbar aortography. Considerable clinical experience was being developed as early as 1942 by Nelson.[24] However, the original methods, together with the inherent toxicity of contrast media, resulted in a significant number of complications.[7,22] Recent technical modifications of the original methods have considerably reduced the morbidity.[4,15]

In the early fifties, Seldinger's technique[27] made injection into the distal aorta possible from the femoral artery. Traversing tortuous and athrosclerotic iliac arteries proved to be difficult and, at times, impossible. Moreover, the presence of a catheter in severely diseased iliac arteries resulted in a significant number of arterial thromboses. In the mid-sixties, unilateral common femoral needle injections with the release Valsalva maneuver appeared promising.[6] Although an excellent method, opacification of extremities is unequal, and the complete cooperation of the patient is required.

With subsequent development of the floppy[25] and "J" tipped guide wires,[18] the tortuous iliac artery has become more consistently and safely navigated. However, with these methods, there remains a 0.5 to 1.8 per cent incidence of arterial thrombosis[11,12,19,20,21] and a relatively high incidence of failure to negotiate the severely diseased iliac arteries. Even in 1970, forward venous angiography was still being advocated.[10] Although a safe method, results are inferior to selective techniques.

The majority of the aforementioned methods were practiced by us as late as 1969. Although uncommon, we were still concerned by the incidence of arterial thrombosis and the inability to enter the iliac arteries and aorta from the femoral artery. After becoming adept at using small catheters, etc., in infants by the femoral artery technique, it was decided to utilize the same approach in adults with occlusive vascular disease. Our experience with this technique began in April, 1970. This method allows us to pass the catheter through areas of stenosis and tortuosity previously non-navigable. Moreover, it is readily learned by the beginning angiographer, is less time consuming and no complications have occurred.

The purpose of this communication is to present what we term the "mini-catheter" approach to femoral arteriography.

MATERIAL AND METHOD

Eighty-five patients with severe occlusive vascular disease were examined from April 1970 through August 1971. With staff supervision, all examinations were performed by radiology residents during their special procedure rotation. The patients' ages ranged from 45 to 85 years, the majority being in the 60 to 75 year age group.

The puncture-catheter apparatus was

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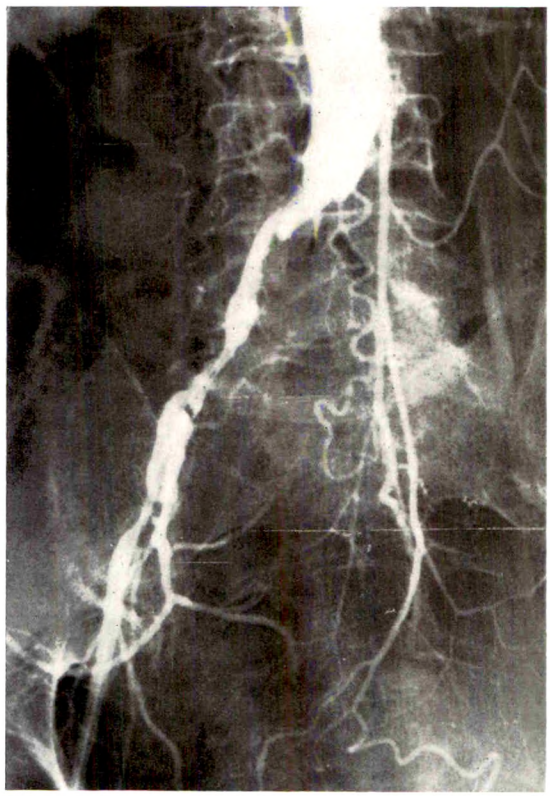


Fig. 1. Anteroposterior pelvic roentgenogram of femoral run-off series in a 75 year old male. This illustrates the passage of a No. 5 French teflon mini-catheter through multiple severely stenotic areas. The catheter did not occlude the femoral or iliac artery lumen at time of study.

that utilized in the pediatric age group. This included a No. 19 thin-walled Cournand needle with a pencil point stylus, together with a 0.025 inch safety guide wire. If the artery was difficult to puncture, a standard No. 19 gauge disposable needle proved effective as a substitute. When the iliac vessels were difficult to navigate, a 0.021 inch guide wire was used.

In the first half of this study, we employed straight guide wires. Recently, a 0.021 inch and a 0.025 inch guide wire with 3 mm. radius "J" tip became available. This was easier to manipulate along tortuous and plaqued arteries.

The "mini-catheter"* is a No. 5 French teflon catheter (I.D. 0.39, O.D. 0.65) pulled over a 0.025 inch guide wire. Five spirally arranged side-holes are made over a 1 cm. spread, 0.5 cm. from the tip. This catheter is now used for all femoral run-off studies, regardless of whether the injection is performed in the abdominal aorta, iliac or femoral arteries.

The smaller catheter-guide wire aspects have allowed us to negotiate through areas of stenosis previously unpassable with larger catheters (Fig. 1). One such maneuver for negotiating extremely tortuous or narrowed arteries is as follows: following percutaneous puncture of the femoral artery, the guide wire is passed up the artery until it meets resistance. A No. 5 teflon catheter is passed over the guide to the point of obstruction. The guide is removed and a test injection is performed to determine the status of the proximal portion of the artery. If the pathway is patent, the guide wire is re-inserted into the catheter to its tip. This latter maneuver yields stability to this system and, to date, has allowed us to pass the small catheter through extremely narrowed areas (Fig. 1). If unexpected difficulties are encountered in inserting the catheter over the standard 0.025 inch guide wire, a No. 4 French teflon catheter is used as a dilator.

For *abdominal* arteriography, an injection rate of 15 cc./sec. for 2 to 3 seconds was used without any evidence of catheter rupture. In 10 cases, 18 to 20 cc. per second were injected. Catheter rupture occurred at the hub in 2 instances. For optimal renal artery visualization, the catheter was positioned approximately 2 to 3 cm. below the origin of the renal arteries (usually at the L2–L3 interspace). For *femoral* run-off studies, the catheter was positioned at the aortic bifurcation. An injection rate of 8 to 12 cc./sec. for 4 to 8 seconds of renografin 76 was utilized.

The moving table-top method[1] was used in 55 of the patients and a long leg filming and manual table-top transit was used in 10 patients. The latter method was used at the Alachua County Hospital where only standard film changers were available. The

* Cook, Inc., Bloomington, Ind.

mini-catheter obviously permits any conceivable filming method. The catheter used in the early portion of this study was radiolucent. The present catheter is radiopaque.

RESULTS

Eighty-five femoral run-off studies were performed with good to excellent quality. Twenty-five aortograms of good quality were obtained as part of the 85 examinations (Fig. 2). The femoral artery approach was used if the pulse was 1 to 2+. The axillary approach was required in only 1 instance in a patient with complete block of both iliac arteries with reconstitution of the common femoral artery. The axillary approach was used in conjunction with a simple deflector method for catheterizing the descending thoracic and abdominal aorta.[13]

Owing to its small size and low coefficient of friction, the mini-catheter was easily and atraumatically inserted in all instances. No complications occurred in any case. There was no instance of a further decrease in pulse, arterial thrombosis, hematoma, or extravasation of contrast material. The arterial flow during the arteriography was not occluded by the catheter, even with extremely narrowed iliac arteries of the magnitude of 2 mm. or less (Fig. 1). Owing to the small size of the catheter, time of compression following removal of the catheter was minimal (usually less than 5 minutes), and has drastically reduced the compression time in hypertensive patients. This reduced time element was maintained in 2 patients who were on anticoagulants.

DISCUSSION

To date, the "mini-catheter" technique has been a tremendous success. The primary modification from other techniques is the use of a small No. 5 French teflon catheter. This, in turn, results in the use of small puncture needles and guide wires. These principles and techniques have enabled us to maintain an excellence in vascular diagnosis with optimal ease and safety.

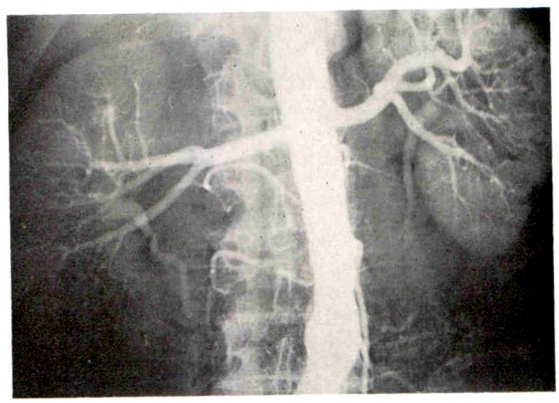


Fig. 2. Anteroposterior abdominal aortogram in a 67 year old male. This degree of opacification of both the aorta and renal arteries was accomplished using a No. 5 French teflon mini-catheter.

The most profound advantage has been the ability to pass the "mini-catheter" through areas of stenosis previously non-navigable with larger catheters (Fig. 1). From a psychologic point of view regarding patient apprehension, etc., the more remote femoral artery approach is better tolerated than the translumbar or axillary approaches. Furthermore, this technique is more easily translated and adapted by the beginning angiographer than our previous techniques. This has noticeably decreased the morbidity inherently associated with training of the neophyte angiographer.

As previously mentioned, we have had no thrombosis at the site of puncture. Admittedly, however, we are dealing with a relatively small patient population regarding statistical relevance. It is well established that clot is deposited on the exterior of the catheter. The clot itself and the amount stripped off during removal is determined by 3 factors:[3,14,16,17,23,28,29] (1) length of time the catheter remains in the artery; (2) diameter; and (3) length of the catheter. Therefore, the small diameter of the mini-catheter as well as the increased expediency in performing the procedure plays a potentially favorable role in maintaining complications to a near zero level.

Teflon as the material for the catheter was chosen for 2 reasons: (1) its ability to withstand high pressure; and (2) its ten-



Fig. 3. Anteroposterior pelvic roentgenogram of a femoral run-off study demonstrating complete occlusion of the mid left external iliac artery by a No. 7 French red Kifa catheter.

dency to result in less external clot formation than polyethylene.[26]

Another advantage of the mini-catheter has been the fact that it has not resulted in total occlusion of the punctured femoral artery (Fig. 3).

The addition of non-thrombogenicity[2,5,8,9] to the mini-catheter itself should enhance the safety of this technique. A method to bind heparin to teflon has not yet been achieved. Since a small heparin bound polyethylene catheter cannot withstand the high pressure needed for superior abdominal aortography (15 cc./sec.), the heparin bound polyethylene catheter can only be used for femoral run-off studies where a lower rate of injection would suffice.

SUMMARY

This communication describes a mini-catheter approach to abdominal and peripheral arterial run-off studies.

Our experience with 85 patients utilizing a No. 5 French teflon catheter has resulted in the following preliminary conclusions: (1) an ability to pass a catheter through severely stenotic areas of the femoral and iliac arteries previously found non-navigable with larger catheters; (2) no obstruction to the flow of contrast medium at the site of puncture or areas of severe stenosis at time of study; (3) it is speedily and atraumatically inserted; (4) no thromboses occurred in our series; and (5) it is a procedure easily transmitted to the beginning angiographer.

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REFERENCES

1. Agee, O. F., and Kaude, J. Arteriography of pelvis and lower extremities with moving table technique. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *107*, 860–865.
2. Amplatz, K. Simple non-thrombogenic coating. *Invest. Radiol.*, 1971, *6*, 280–289.
3. Amplatz, K. Editorial. Catheter embolization. *Radiology*, 1968, *91*, 392–393.
4. Amplatz, K. Translumbar catheterization of abdominal aorta. *Radiology*, 1963, *80*, 927–931.
5. Eriksson, J. C., Gillberg, G., and Lagergren, H. New method for preparing nonthrombogenic plastic surfaces. *J. Biomed. Mater. Res.*, 1967, *1*, 301–312.
6. Friedenberg, M. J., and Carlsson, E. Bilateral aorto-ilio-femoral arteriography via unilateral common femoral artery needle. *Acta radiol.* (Diag.), 1965, *3*, 1–8.
7. Gaylis, H., and Laws, J. W. Dissection of aorta as complication of translumbar aortography. *Brit. M. J.*, 1956, *2*, 1141–1146.
8. Glancy, J. F., Fishbone, G., and Heinz, E. R. Nonthrombogenic arterial catheters. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *108*, 716–723.
9. Gramer, R., Frech, R. S., and Amplatz, K. Preliminary human study with simple non-thrombogenic catheter. *Radiology*, 1971, *100*, 421–422.

10. GREENSPAN, R. H., BERNSTEIN, E. F., and LOKIN, M. K. Intravenous aortography. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *83*, 1034–1041.
11. HALPERN, M. Percutaneous transfemoral arteriography: analysis of complications in 1,000 consecutive cases. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1964, *92*, 918–934.
12. HAUT, G., and AMPLATZ, K. Complication rates of transfemoral and transaortic catheterization. *Surgery*, 1968, *63*, 594–596.
13. HAWKINS, I. F. Deflector axillary approach to abdominal aorta. To be published.
14. HOSHAL, V. L., AUSE, R. G., and HOSKINS, P. A. Fibrin sleeve formation on indwelling central venous catheters. *Arch. Surg.*, 1971, *102*, 353–358.
15. HUSNI, E. A. New needle and safer method of arteriography. *Surg., Gynec. & Obst.*, 1958, *106*, 627–630.
16. JACOBSSON, B., and SCHLOSSMAN, D. Angiographic investigation of formation of thrombi on vascular catheters. *Radiology*, 1969, *93*, 355–359.
17. JACOBSSON, B., and SCHLOSSMAN, D. Thromboembolism of leg following percutaneous catheterization of femoral artery for angiography: predisposing factors. *Acta radiol.* (Diag.), 1969, *8*, 109–118.
18. JUDKINS, M. P., KIDD, H. J., FRISCHE, L. H., and DOTTER, C. T. Lumen following safety J guide for catheterization of tortuous vessels. *Radiology*, 1967, *88*, 1127–1130.
19. KLASTER, F., BRISTOW, J. D., and GRISWOLD, H. Femoral artery occlusion following percutaneous catheterization. *Am. Heart J.*, 1970, *79*, 175–180.
20. KOTTKE, B., FAIRBAIRN, J., and DAVIS, G. Complications of aortography. *Circulation*, 1964, *30*, 843–847.
21. LANG, E. K. Survey of complications of percutaneous retrograde arteriography. *Radiology*, 1963, *81*, 257–263.
22. MCAFEE, J. C., and WILSON, J. K. Review of complications of translumbar aortography. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1956, *75*, 956–970.
23. NEJAD, M. S., KLAPER, M. A., STEGGERDA, F. R., and GIANTURCO, C. Clotting on outer surfaces of vascular catheters. *Radiology*, 1968, *91*, 248–250.
24. NELSON, O. A. Arteriography of abdominal organs by aortic injection. *Surg., Gynec. & Obst.*, 1942, *74*, 655–662.
25. ROSSIE, P., and VERDU, C. Floppy wire as aid in arterial catheterization. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *97*, 511–518.
26. SCHLOSSMAN, D. Personal communication.
27. SELDINGER, S. I. Catheter replacement of needle in percutaneous arteriography: new technique. *Acta radiol.*, 1953, *39*, 368–376.
28. SIEGELMAN, S. S., CAPLAN, L. H., and ANNES, G. P. Complications of catheter angiography: study with oscillometry and "pullout" angiograms. *Radiology*, 1968, *91*, 251–253.
29. TORMANEK, G., FRECH, R. S., and AMPLATZ, K. Arterial thrombus formation during clinical percutaneous catheterization. *Circulation*, 1970, *41*, 833–839.

SYSTEMIC HEPARINIZATION FOR ANGIOGRAPHY*

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ARTERIAL occlusions near the site of entry of angiographic catheters occur with a frequency variously reported from 0.4 per cent[6] to 2.3 per cent.[13] In the past such occlusions have been attributed to the displacement of arteriosclerotic plaques or to thrombi formed at the site of intimal trauma. More recently Nejad *et al.*[10] demonstrated in dogs that these arterial obstructions are most probably due to the stripping of thrombotic material from the surface of the angiographic catheters as these catheters are withdrawn through the wall of the vessels at the end of the procedures. This mechanism was also shown in humans by Siegelman *et al.*;[13] and was later confirmed by Formanek *et al.*,[3] and Jacobsson.[9]

The formation of thrombotic coating on the outer walls of vascular catheters may be lessened by the flushing injections of heparinized saline employed to prevent the formation of thrombus inside the catheters. This injected heparin does reach the general circulation, but its amount is probably inadequate to prevent the occasional thrombotic arterial occlusion.

To prevent thrombus formation on the outer walls of the intravascular catheters more effectively, these catheters have been soaked in heparin, treated with graphite-benzalkonium-heparin, and coated with benzalkonium-heparin wax or with silicone grease.[1,4,5,9,10] It has also been noted that certain plastics (polyurethane) are less thrombogenic than others (polyethylene, vinyl chloride, silastic), probably because of inherent or acquired electrical charges.[10] Further studies in this field are obviously needed.

Several investigators have suggested that the thrombotic deposition on the outer walls of catheters could be avoided by decreasing the coagulability of blood. Aspirin, dextran and heparin can be employed.

METHOD

Since 1969 we have utilized systemic heparinization. Our mode of administration of heparin is as follows:

As soon as the catheter is in the arterial system, usually in the external iliac artery or in the lower abdominal aorta, a single injection of 1,000 to 5,000 units of aqueous heparin is made through the catheter. The dosage is varied according to the size and weight of the patient, approximately 45 units/kg., taking into consideration the baseline clotting capacity of the patient's blood. The average initial injection is 3,000 units of aqueous heparin. The catheter is then flushed every 2 to 3 minutes with 5 to 10 ml. of a solution of 1,000 units of heparin in 500 ml. of normal saline.

The rationale for this procedure is to obtain an adequate decrease in the coagulability of blood as soon as the catheter is exposed to it. This is done with the initial injection of heparin. Flushing the catheter with a dilute heparin solution every few minutes keeps the lumen of the catheter patent and replaces, at least in part, the original heparin as it is metabolized.

MATERIAL AND RESULTS

In the past 2 years systemic heparinization has been utilized at M. D. Anderson Hospital and Tumor Institute during angiography in 525 patients to detect the presence and the extent of neoplastic disease. The majority of these procedures were

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done through a femoral approach using polyethylene catheters. Most examinations involved multiple selective studies and had an average duration of 65 minutes.

With the use of these larger amounts of heparin the possibility of hemorrhage during the procedure as well as after withdrawal of the catheter raises some concern. Fifteen of the 525 patients had bleeding at the puncture site which persisted after 15 minutes of arterial compression. These patients were given intravenously 10 mg. protamine sulfate for each 1,000 units of heparin injected intra-arterially at the start of the procedure—an average of 30 mg. protamine sulfate. This resulted in complete control of bleeding within 10 minutes. The bleeding incidence was similar to that which was observed in previous years when only intermittent flushing injections of a solution containing 5,000 units of heparin in 500 ml. of normal saline were employed. In patients examined by angiography to determine the specific site of bleeding, only intermittent flushing injections of heparinized saline are made.

None of the 525 patients manifested clinical signs or symptoms attributable to thrombotic arterial occlusion at the puncture site. Pull-out angiograms, arteriograms at the puncture site just prior to almost complete withdrawal of the catheter, were obtained in 78 patients. In 2 of 78 pull-out angiograms (2.5 per cent) there was a fine lucent line about the catheter which was thought to be a thrombus. This compares favorably with the 50 per cent incidence of thrombi visible on pull-out angiograms reported by Formanek *et al.*[3] In view of these results, systemic heparinization can help prevent the formation of thrombi.

Of the 525 patients, 1 was noted to have a decrease in the intensity of the arteria dorsalis pedis pulse following the procedure. He had no clinically significant sequelae. Another patient experienced hemiplegia during a 4 vessel cerebral arteriography. Both patients suffered embolization probably due to liberation of intraluminal thrombi or displaced plaques.

EXPERIMENTAL INVESTIGATION

In order to compare the value of systemic heparinization with that of flushing intermittent injections of dilute heparin, the following study was undertaken:

In 12 adult patients, Group I, the catheters were flushed every 2 or 3 minutes with 5 to 10 ml. of a solution of 5,000 units of aqueous heparin in 500 ml. of normal saline. This delivered an estimated average dosage of 2,000 units of heparin in 65 minutes.

Eleven other patients, Group II, were given systemic heparinization initiated by an injection of 45 units/kg. of aqueous heparin and followed by flushing intermittent injections of a solution of 1,000 units of heparin in 500 ml. of normal saline. This delivered an estimated 3,400 units of heparin in 65 minutes.

Venous blood samples were drawn at 0, 5, 10, 15, 30, 45, 60, 75 and 90 minutes after the beginning of the angiographic procedure and each sample was studied for activated clotting time (ACT),[7] activated partial thromboplastin time (APTT),[2,14] and prothrombin time (PT). The findings were averaged and incorporated in graphs shown by Figures 1, 2, and 3.

Group I shows that enough heparin was delivered by the intermittent flushing injections to affect ACT, APTT, and PT. The decrease in coagulability, however, was gradual and small, reaching its peak at 75 minutes or 10 minutes after the end of the procedure (Fig. 4).

Group II illustrates that the maximum effect of the initial injection of heparin occurs in 10 to 15 minutes with a gradual decrease during the procedure (Fig. 5). When the catheter is withdrawn, at an average of 65 minutes, there is no significant difference in the level of coagulability of the blood when the 2 groups are compared.

Systemic heparinization maintains effective anticoagulation during the procedure rather than reaching a peak just after the end of the procedure as is produced by intermittent injections of a flushing solution of heparinized saline. Prior to utilizing systemic heparinization a coagulation battery

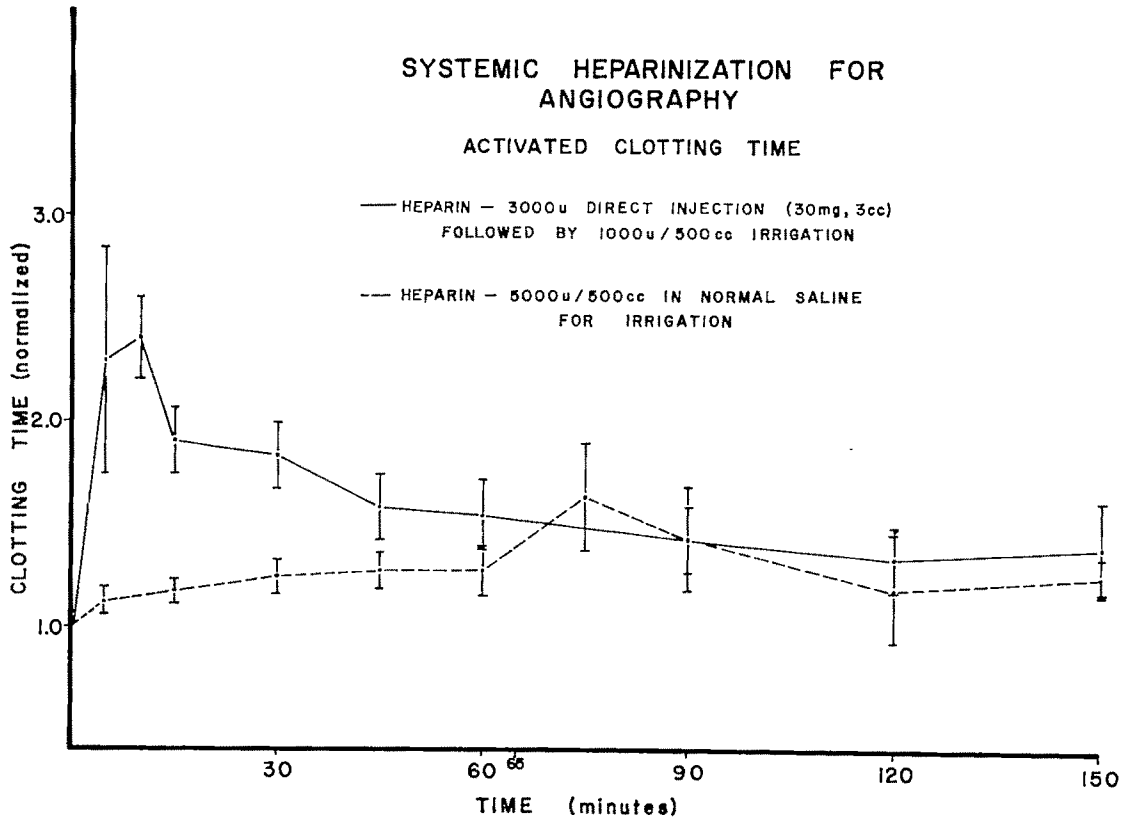


FIG. 1. Activated clotting time.

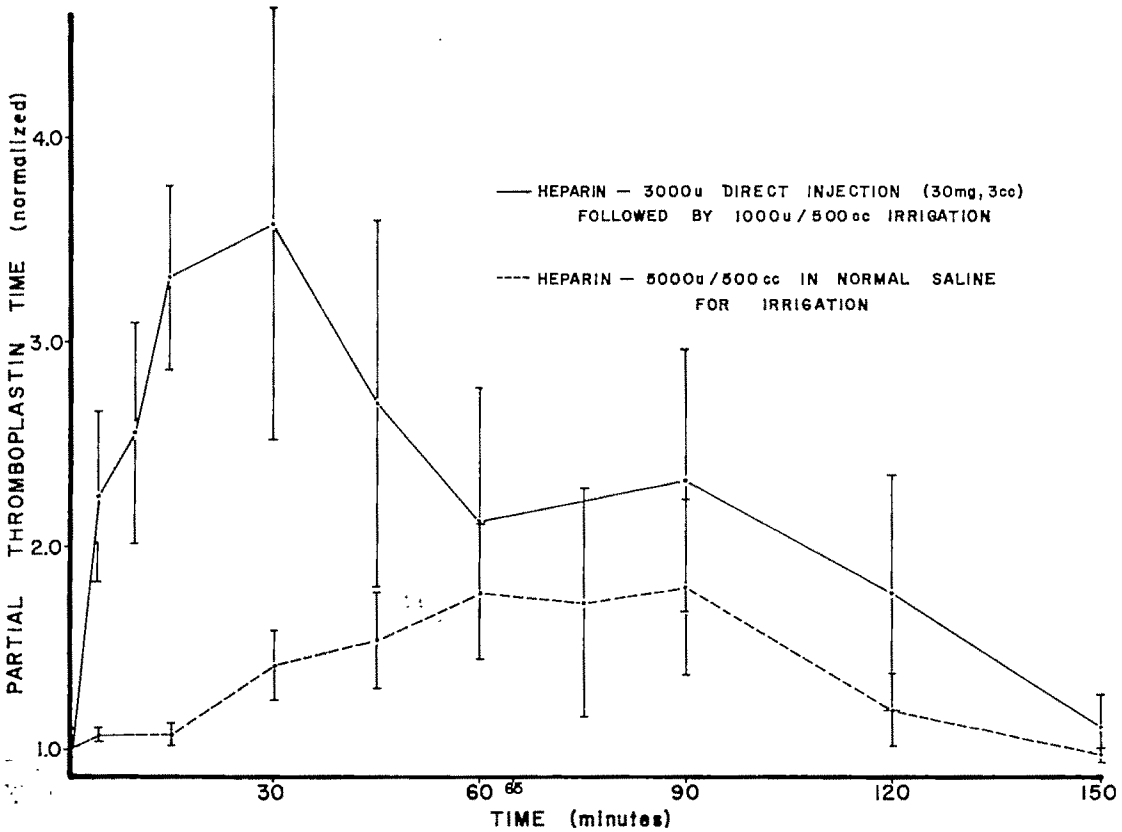


FIG. 2. Activated partial thromboplastin time.

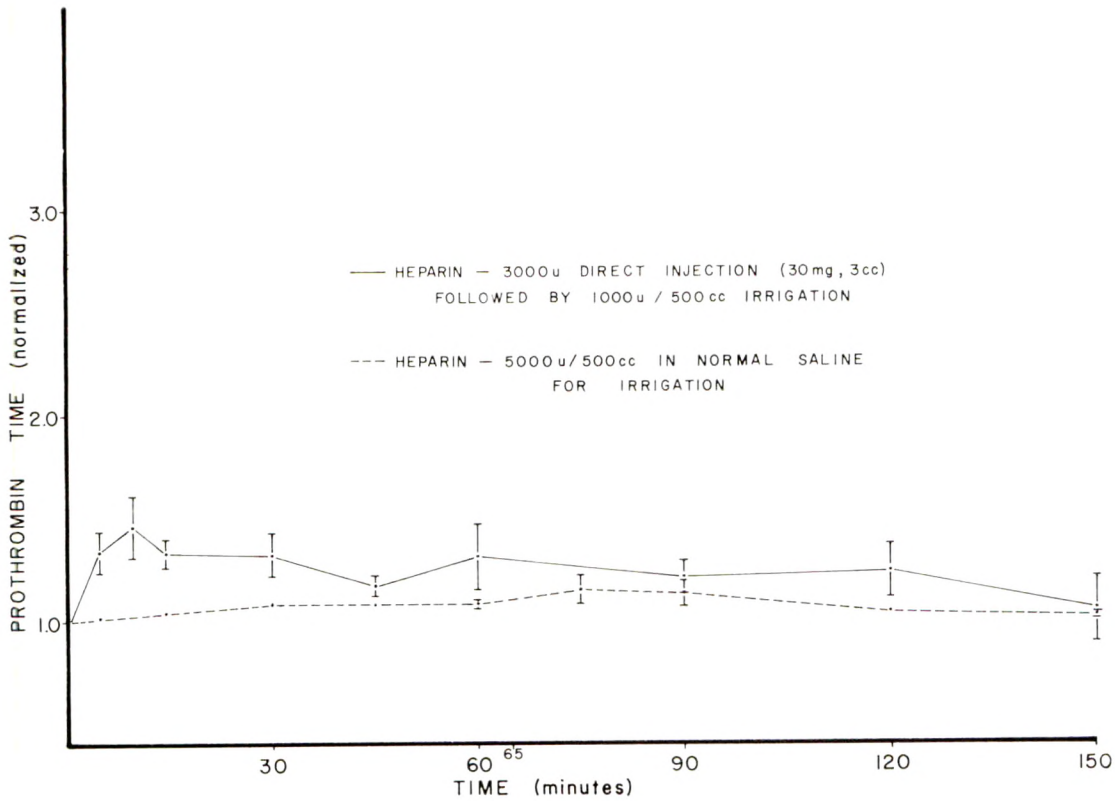


FIG. 3. Prothrombin time.

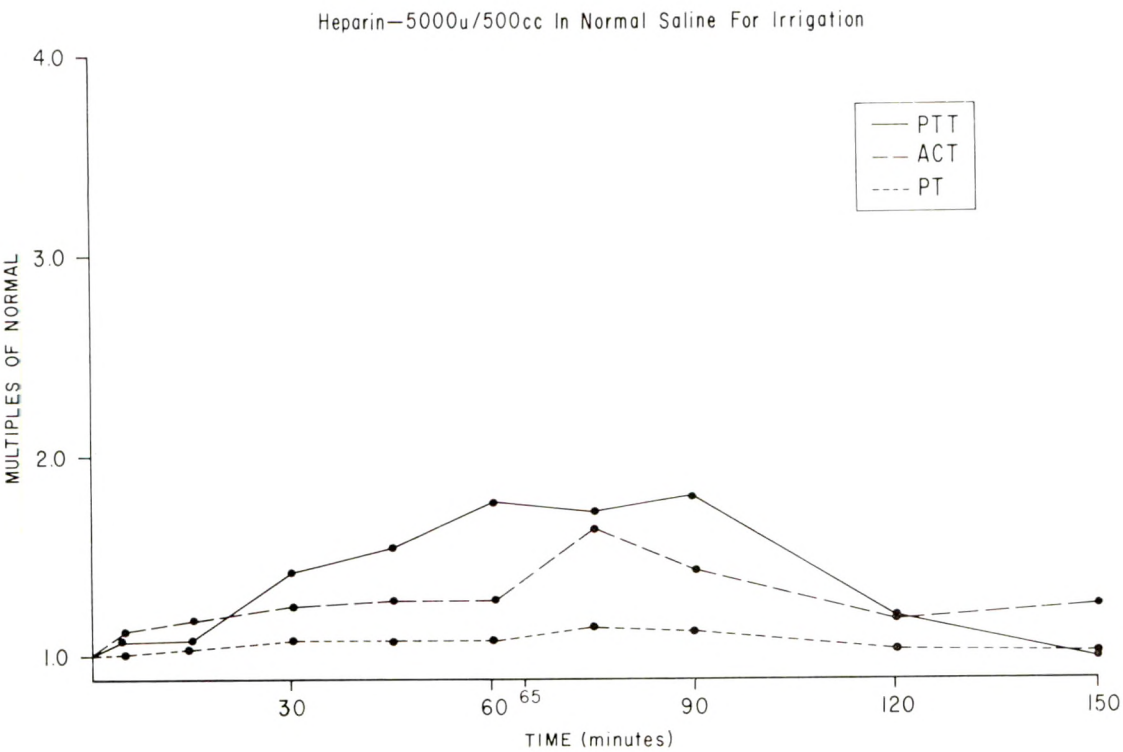


FIG. 4. Composite diagram. PTT = partial thomboplastin time; ACT = activated clotting time; PT = prothrombin time.

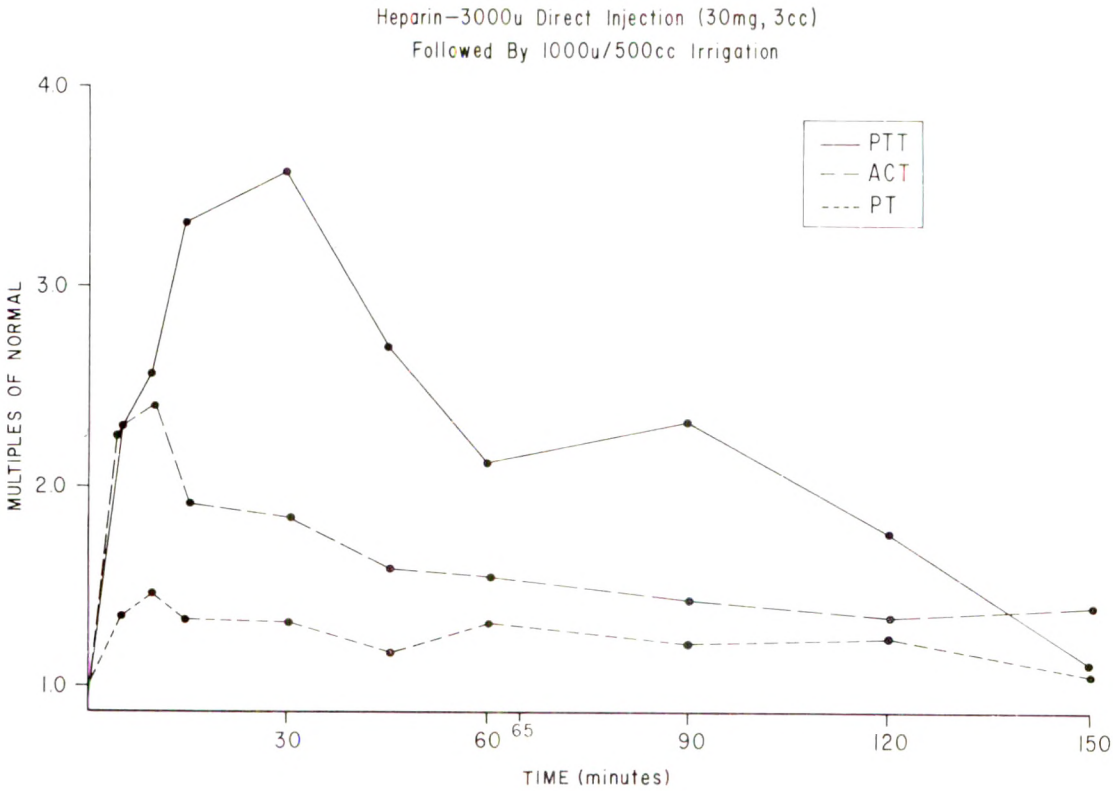


FIG. 5. Composite diagram. Key same as in Figure 4.

must be done which should include ACT, APTT, PT, as well as a platelet count.

SUMMARY

Systemic heparinization significantly decreases the complications of angiography due to thrombosis.

Effective systemic heparinization is achieved by a combination of the direct intra-arterial injection of heparin through the catheter at the onset of the procedure and flushing intermittently with a dilute solution of heparinized saline.

No increase in hemorrhage at the puncture site is noted due to this method of anticoagulation.

Certain precautions must be observed because of the larger doses of heparin employed.

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REFERENCES

1. AMPLATZ, K. Simple non-thrombogenic coating. *Invest. Radiol.*, 1971, *6*, 280–289.
2. BURDICK, C. O. Partial thromboplastin time in heparin therapy. Documentation of data. *Am. J. Clin. Path.*, 1971, *55*, 384.
3. FORMANEK, G., FRECH, R. S., and AMPLATZ, K. Arterial thrombus formation during clinical percutaneous catheterization. *Circulation*, 1970, *41*, 833–839.

4. Glancy, J. J., Fishbone, G., and Heinz, E. R. Nonthrombogenic arterial catheters. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *108*, 716–723.
5. Gott, V. L., Whiffen, J. D., Kiepke, D. E., Daggett, R. L., Boake, W. C., and Young, W. P. Techniques of applying graphite-benzalkonium-heparin coating to various plastics and metals. *Tr. Am. Soc. Artif. Int. Organs*, 1964, *10*, 213–217.
6. Halpern, M. Percutaneous transfemoral arteriography: analysis of complications in 1,000 consecutive cases. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1964, *92*, 918–934.
7. Hattersley, P. G. Activated coagulation time of whole blood. *J.A.M.A.*, 1966, *196*, 436–440.
8 Haut, G., and Amplatz, K. Complication rates of transfemoral and transaortic catheterization. *Surgery*, 1968, *63*, 594–596.
9. Jacobsson, B. Effect of pretreatment with dextran 70 on platelet adhesiveness and thromboembolic complications following percutaneous arterial catheterization. *Acta radiol.* (Diag.), 1969, *8*, 289–295.
10. Nejad, M. S., Klaper, M. A., Steggerda, F. R., and Gianturco, C. Clotting on outer surfaces of vascular catheters. *Radiology*, 1968, *91*, 248–250.
11. Nye, S. W., Graham, J. B., and Brinkhous, K. M. Partial thromboplastin time as screening test for detection of latent bleeders. *Am. J. M. Sc.*, 1962, *243*, 279.
12. Ross, R. S. Arterial complications. *Circulation*, 1968, Suppl. III 37, 38–39.
13. Siegelman, S. S., Caplan, L. H., and Annes, G. P. Complications of catheter angiography: study with oscillometry and "pullout" angiograms. *Radiology*, 1968, *91*, 251–253.
14. Spector, I., and Corn, M. Control of heparin therapy with activated partial thromboplastin times. *J.A.M.A.*, 1967, *201*, 75–77.

THE AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

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Fifty-fifth Annual Meeting: Broadmoor Hotel, Colorado Springs, Colorado, April 22–26, 1973.

EDITORIAL

CONGENITAL DEXTROCARDIA AND THE GENERAL RADIOLOGIST

THE CONCEPTS and classification of all types of congenital heart disease have been extensively revised, modified or extended during the past 30 years. The large advances in surgical treatment combined with selective angiography and associated physiologic and hemodynamic studies have provided the necessary anatomic and functional detail permitting refinements and extension of classification and nomenclature. This undoubtedly has resulted in an improvement in the management of patients with congenital heart disease.

The nomenclature and classification of congenital dextrocardia, however, have remained a problem. In the first place, congenital dextrocardia is not a pathologic entity *per se*. It is a positional anomaly that may or may not be associated with heart disease. The management of the patient, if such is required, is directed to diagnosis and treatment of the *other* concomitant cardiac anomalies. Secondly, the older literature is a thicket of imprecise terms, often quite variable in meaning as one goes from one paper to another. Much of the difficulty lay in a lack of anatomic detail since selective angiocardiography was not widely available in the earlier years. A much larger impediment was a lack of conceptual appreciation of the development and differentiation of the heart and how aberrations could produce the observed appearance.

Van Praagh and his associates[14] in 1964 introduced a new terminology and new approach to congenital dextrocardia. Others[4] had recognized that anomalous and discordant (in relation to the position of the viscera) bending of the primitive cardiac tube was responsible for "corrected transposition," a condition associated with a high incidence of other congenital anomalies and also found in a considerable proportion of cases of dextrocardia.[5,8] Complete transposition of the great vessels is found in a lesser but significant proportion of cases of dextrocardia (12 per cent in one series).[11] Selective angiography can demonstrate the relative position of the great arteries (transposed, normal or inverted) and usually shows the valves or valve rings and the internal morphology (trabeculation, conus structure, crista supraventricularis) that permits identification of the morphologic right and left ventricles. The position of the atria can be determined from the position of the stomach and liver and confirmed by angiography. With this kind of knowledge, why not designate each variant by more precise terms? Van Praagh and his colleagues[14,15] have proposed a terminology and classification of dextrocardia that first separates the cases into those with situs solitus (normal position of the abdominal viscera) and situs inversus (complete reversal of the abdominal viscera). These 2 groups can then be divided into those with (d) dextrocardiac loop and (l) levocardiac loop. These 4 groups can then be divided into those with nontransposed great arteries and transposed great arteries. The nontransposed groups are further divided into the normal and inverted arrangements; the transposed groups are divided into right and left-sided transposition with reference to the aorta. The development of this system followed a detailed study of a group of pathologic specimens and was confirmed by application in practical preoperative diagnosis. Its development and use have been a solid advance, and those radiologists who have mastered its use find that it works well.[6] However, there are certain attributes

of this system which although well known may not have been completely appreciated. It is *not* a classification of dextrocardia *per se*, but can be applied to the heart and viscera in any situs. When applied to dextrocardia, it must be preceded by the specific term "dextrocardia." The system is complicated and difficult to master and requires almost daily practice. Of the 12 possible combinations, 6 are common, and to these are added the syndromes of asplenia or polysplenia in which the system is less definitive since visceral and atrial situs may be undetermined.[7] It cannot be applied unless it is preceded by technically adequate selective angiography. It is most useful in the selection and preparation of patients for surgical treatment. Its application is thus very much in the realm of the cardiovascular radiologist and pediatric cardiologist, but may be relatively meaningless to the general radiologist because of infrequent use.

In order not to lose our perspective, we should remember that congenital intrinsic dextrocardia (as distinguished from extrinsic displacement) is not a common condition. It is thought to occur in 1 in 7,000 to 1 in 10,000 persons in the general population.[3,12] Dextrocardia without situs inversus is even rarer,[9] and another malposition, levocardia with situs inversus, is so uncommon that the general radiologist may see only a few cases in his lifetime exclusive of his training period. It is the author's opinion that frequency of significant and symptomatic heart disease in association with dextrocardia as reported in recent series[5,6,8] has been considerably exaggerated. The material usually was collected at large cardiac clinics and thus biased toward patients with severe associated anomalies. The experience of the general radiologist is likely to be considerably different in that a majority of his patients may be without significant heart disease. Selective angiocardiography may not be necessary or indicated in these cases, or if so, it is not immediately available. The generalist thus needs a system or approach based on findings obtained from conventional roentgenograms. Elliott *et al.*[5] have proposed such a system, although others have used it at least in part for years.

Methodical study of conventional and overexposed frontal roentgenograms of the chest supplemented in some instances by a barium swallow examination permits a determination of visceral and atrial situs with the exception of the rare instances of asplenia or polysplenia. The fundus of the stomach and the aortic arch are normally on the same side and opposite to the main mass of the usually asymmetric liver. The right atrium almost invariably lies on the side opposite the stomach and the aortic arch. Where the stomach and aortic arch are on opposite sides (discordant), a close study of the tracheobronchial tree as seen on an overexposed frontal roentgenogram[13] permits an identification of the right and left lung. The right main bronchus is short, wide and gives off the eparterial bronchus. The left main bronchus is much longer, narrower and runs a more horizontal course. (The reader is referred to the excellent article by Van Mierop *et al.*[13] for more details.) The atria lie on the same side as their respective lungs. In asplenia, there are bilateral right lungs, and in polysplenia, there *may be* bilateral left lungs, and the location of the atria and other viscera may then be uncertain (situs ambiguus).[13] The presence of either of these conditions may be further confirmed by the appearance of a symmetric bi-lobed liver and paramedian location of the stomach as seen on the chest roentgenogram.[2,7,10] Further close scrutiny of the overexposed frontal roentgenogram may identify an enlarged azygos vein or another anomalous venous structure on the left which suggests the presence of an interruption of the inferior vena cava with azygos return of the blood from the lower part of the body.

Further specific anatomic detail is usually not provided by conventional roentgenograms, but probabilities of abnormalities can be proposed. The referring physician can usually ascertain whether the pa-

tient is asymptomatic or has a high probability of significant associated cardiac disease. In an asymptomatic patient with situs inversus and a normal but reversed cardiac contour, the probability of an otherwise normal heart is very high.[1] On the other hand, in a patient with substantial clinical evidence of heart disease and dextrocardia and situs solitus, the probability of one of the transposition complexes, most likely corrected transposition, is very high.[5,8] This could be further supported if the left upper mediastinum is widened by what appears to be the ascending aorta.[13]

The terminology of the report could be quite simple using the terms dextrocardia with or without situs inversus. The presence or absence of significant heart disease could be designated as uncomplicated or complicated and would be in considerable part determined by the referring physician. Other suggested anomalies, such as tetralogy of Fallot, tricuspid atresia, single ventricle, etc., would be designated by their usual terms as applied to the heart in its normal position. "Dextroversion" seems to be a term that we should eliminate unless we can define it more precisely. "Version" implies turning, and the addition of "dextro" would indicate turning toward the right, whereas the explanation of dextrocardia is that of failure of the heart to turn normally to the left.[5] The definition of dextroversion given in Dorland's Illustrated Medical Dictionary, 24th edition, is "location of the heart in the right hemithorax, the left ventricle remaining on the left as in the normal position, but lying anterior to the right ventricle." Such a definition could not be applied to dextrocardia with corrected transposition since the morphologic left ventricle lies anteriorly and to the left. Thus, the uncomplicated dextroversion as described by Welch and Felson[16] would seem to be the correct usage of this term. With this much uncertainty, the replacement of the term "dextroversion" by "dextrocardia with situs solitus" is recommended. This could be further qualified by complicated or uncomplicated and the usual terms applied to associated cardiac anomalies.

My message to the general radiologist is that, although he is unable through lack of practice and opportunity to master an all inclusive and logical but esoteric and elaborate classification of dextrocardia, he still has prerogatives and responsibilities that he should continue to exercise. His methods are the conventional roentgenogram of the chest and a systematic logical approach. His report can be given in terms understandable to both himself and his colleagues. His limitations are the same as for those of any other type of congenital abnormality of the heart.

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REFERENCES

1. Burchell, H. B., and Pugh, D. G. Uncomplicated isolated dextrocardia ("dextroversio cordis" type). *Am. Heart J.*, 1952, *44*, 196–206.
2. Campbell, M., and Deuchar, D. C. Absent inferior vena cava, symmetrical liver, splenic agenesis, and situs inversus and their embryology. *Brit. Heart J.*, 1967, *29*, 268–275.
3. Cockayne, E. A. The genetics of transposition of the viscera. *Quart. J. Med.*, 1938, *7*, 479–493.
4. de la Cruz, M. V., Anselmi, G., Cisneros, F., Reinhold, M., Portillo, B., and Espino-Vela, J. An embryologic explanation for the corrected transposition of the great vessels: additional description of the main anatomic features of this malformation and its varieties. *Am. Heart J.*, 1959, *57*, 104–117.
5. Elliott, L. P., Jue, K. L., and Amplatz, K. A roentgen classification of cardiac malpositions. *Invest. Radiol.*, 1966, *1*, 17–28.
6. Ellis, K., Fleming, R. J., Griffiths, S. P., and Jameson, A. G. New concepts in dextrocardia; angiographic considerations. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1966, *97*, 295–313.
7. Ivemark, B. J. Implications of agenesis of spleen in pathogenesis of conotruncus anomalies in childhood. *Acta paediat.*, 1955, *44*, Supp. 104, 590.
8. Keith, J. D., Rowe, R. D., and Vlad, P. Heart Disease in Infancy and Childhood. The Macmillan Company, New York, 1958, p. 535.

9. Lowe, C. R., and McKeown, T. An investigation of dextrocardia with and without transposition of abdominal viscera, with a report of a case in one monozygotic twin. *Ann. Eugenics*, 1954, *18*, 267–277.
10. Ongley, P. A., Titus, J. L., Khoury, G. H., Rahimtoola, S. H., Marshall, H. J., and Edwards, J. E. Anomalous connection of pulmonary veins to right atrium associated with anomalous inferior vena cava, situs inversus and multiple spleens—a developmental complex. *Mayo Clin. Proc.*, 1965, *40*, 609–624.
11. Paul, M. H. Heart Disease in Infants, Children and Adolescents. Williams & Wilkins Company, Baltimore, 1968, pp. 527–552.
12. Torgersen, J. Genetic factors in visceral asymmetry and in the development and pathologic changes of lungs, heart and abdominal organs. *Arch. Path.*, 1949, *47*, 566–593.
13. Van Mierop, L. H. S., Eisen, S., and Schiebler, G. L. The radiographic appearance of the tracheobronchial tree as an indicator of visceral situs. *Am. J. Cardiol.*, 1970, *26*, 432–435.
14. Van Praagh, R., Van Praagh, S., Vlad, P., and Keith, J. D. Anatomic types of congenital dextrocardia; diagnostic and embryologic implications. *Am. J. Cardiol.*, 1964, *13*, 510–531.
15. Van Praagh, R., Van Praagh, S., Vlad, P., and Keith, J. D. Diagnosis of the anatomic types of congenital dextrocardia. *Am. J. Cardiol.*, 1965, *15*, 234–247.
16. Welch, R. A., and Felson, B. Uncomplicated dextroversion of the heart. *Radiology*, 1956, *66*, 24–32.

NEWS ITEMS

ANGIOGRAPHY

The Department of Radiology of Harvard Medical School will sponsor a 3 day course on "Angiography: 1972," at the Sheraton-Boston Hotel, Boston, Massachusetts, October 12–14, 1972.

The Chairman of the course is Herbert L. Abrams, M.D., Professor and Chairman, Department of Radiology, Harvard Medical School.

The course is designed for radiologists who participate in or intend to perform angiographic studies. As such, it will define the present state of the art with a critical look at certain technical aspects. It will detail the application of the angiographic method to visceral diseases in a series of authoritative lectures and demonstrations with particular emphasis on the intestinal tract, kidneys, liver, spleen, and pancreas. The subject of gastrointestinal bleeding will be dealt with in detail.

Although the course will cover established areas of angiographic examination and interpretation, it will also embody and emphasize recent advances, their potential and their limitations.

An outstanding faculty will present the course. Dr. Erik Boijsen, Professor of Radiology at the University of Lund and Head of the Department at the Malmö Hospital, and Dr. Kurt Amplatz, Professor of Radiology at the University of Minnesota, will be guest speakers.

The faculty will also include Herbert L. Abrams, M.D., Douglass F. Adams, M.D., A. Clifford Barger, M.D., Stanley Baum, M.D., Harvey Eisenberg, M.D., Kenneth Fellows, M.D., Ernest Ferris, M.D., Florencio A. Hipona, M.D., Norman K. Hollenberg, M.D., Paul C. Kahn, M.D., Harry Z. Mellins, M.D., Edward B. D. Neuhauser, M.D., Sven Paulin, M.D., and others.

For further information and applications, please write to the Department of Continuing Education, Harvard Medical School, 25 Shattuck Street, Boston, Massachusetts 02115.

NEURORADIOLOGY

The Thirty-fifth Annual Continuation Course in Radiology of the University of Minnesota is devoted this year to Neuroradiology and will be held in the Mayo Memorial Auditorium, October 16–20, 1972.

A practical and modern approach to this important segment of Diagnostic Radiology will be presented by a large faculty of acknowledged leaders and teachers in this field. Nuclear medicine will be included. Both the general radiologist and the neuroradiologist will benefit from this course. Neurologists and neurosurgeons are welcome.

In keeping with previous tradition an outstanding guest faculty has been assembled for the course under the direction of Dr. Eugene Gedgaudas, Professor and Head, Dr. Harold O. Peterson, Professor, Dr. Stephen A. Kieffer, Professor, and Dr. Lawrence H. A. Gold, Associate Professor, Department of Radiology.

The guest faculty will include: Drs. R. Thomas Bergeron, Los Angeles, California; David O. Davis, Salt Lake City, Utah; George duBoulay, London, England; Herbert I. Goldberg, Philadelphia, Pennsylvania; Derek Harwood-Nash, Toronto, Ontario; E. Ralph Heinz, Pittsburgh, Pennsylvania; Sadek K. Hilal, New York, New York; O. Wayne Houser, Rochester, Minnesota; Irvin I. Kricheff, New York, New York; Thomas H. Newton, San Francisco, California; D. Gordon Potts, New York, New York; David F. Reese, Rochester, Minnesota; Mannie M. Schechter, Bronx, New York; and Ernest H. Wood, New York, New York.

The remainder of the faculty will consist of members of the teaching staff of the University of Minnesota Medical School.

For further information please write to: Director, Postgraduate Medical Education, Nolte Center for Continuing Education, University of Minnesota, Minneapolis, Minnesota 55455.

RADIOLOGY AND NUCLEAR MEDICINE POSTGRADUATE COURSE

Duke University Medical Center

The Fourth Annual Radiology and Nuclear Medicine Postgraduate Course to be presented by the Department of Radiology at the Duke University Medical Center, Durham, North Carolina, will be held October 25–27, 1972.

The distinguished faculty includes: David H. Baker, M.D., New York, New York; William H. Briner, B.S., Durham, North Carolina; John A. Evans, M.D., New York, New York; Robert G. Fraser, M.D., Montreal, Canada; Jack K. Goodrich, M.D., Durham, North Carolina; John A. Goree, M.D., Durham, North Carolina; Herman Grossman, M.D., Durham, North Carolina; C. Craig Harris, M.S., Durham, North Carolina; E. Robert Heitzman, M.D., Syracuse, New York; Merle K. Loken, M.D., Minneapolis, Minnesota; Richard G. Lester, M.D., Durham, North Carolina; George M. McCord, M.D., Durham, North Carolina; Robert McLelland, M.D., Durham, North Carolina; William B. Seaman, M.D., New York, New York; Elias G. Theros, M.D., Washington, D. C.; and Joseph B. Workman, M.D., Durham, North Carolina.

For further information please contact: Robert McLelland, M.D., Program Director, Department of Radiology, Box 3808, Duke University Medical Center, Durham, North Carolina 27710.

POSTGRADUATE COURSE ON BONE AND JOINT RADIOLOGY

A Postgraduate Course on Bone and Joint Radiology is being sponsored by the Department of Radiology, Cornell University Medical College, October 30, 31 and November 1, 1972, in the Memorial Hospital Auditorium, New York, New York.

The topics are: anatomy and physiology of bone; pediatric disease; joint disease; hematologic disease; and bone tumors, followed by an arthrography workshop in the Hospital for Special Surgery Auditorium and Radiology Department.

The guest faculty includes: John A. Caffey, M.D., University of Pittsburgh School of Medicine; Jack Edeiken, M.D., Jefferson Medical College, Thomas Jefferson University; Frieda Feldman, M.D., Columbia University; Harold G. Jacobson, M.D., Montefiore Hospital and Medical Center; Lennart Krook, D.V.M., N. Y. State Veterinary College, Cornell University; John Moseley, M.D., Mt. Sinai School of Medicine; and Alex Norman, M.D., Mt. Sinai School of Medicine.

The Cornell University Medical College and the New York Hospital, Cornell Medical Center Faculty will comprise the local faculty.

Apply for enrollment to: John A. Evans, M.D., Department of Radiology, Cornell University Medical College, 1300 York Avenue, New York, New York 10021.

COURSE IN ARTHROGRAPHY, LYMPHOGRAPHY AND TEMPORAL BONE RADIOGRAPHY

The Departments of Radiology of the University of Utah, Salt Lake City, Utah, and of George Washington University, Washington, D. C., are sponsoring a radiology course titled "Arthrography, Lymphography, and Temporal Bone Radiography," to be held in seminar fashion, at the Sahara Hotel, Las Vegas, Nevada, January 30 through noon February 2, 1973.

The course will be given under a format considerably different from most previous radiology courses. Few formal lectures will be delivered. Two to three two-hour long seminar sessions will be held daily and the group size in each seminar will be no larger than 12 persons. All seminars will run simultaneously with groups shifting from room to room for their allotted sessions.

The seminar directors will be radiologists expert in their fields. Each will supervise de-

tailed case analysis by the participants. Thus, course registrants will have an opportunity to work closely with each instructor.

For further information, please contact: P. Ruben Koehler, M.D., University of Utah Medical Center, Salt Lake City, Utah 84112.

SYMPOSIUM RADIOLOGICUM PANCREATIS

Under the patronage of the International Society of Radiology and the German Roentgen Association, a symposium titled "Symposium Radiologicum Pancreatis" will be held in Munich, West Germany, May 31–June 6, 1973.

The subjects include: radiographic anatomy; pathologic anatomy; physiopathology; clinical aspects; upper gastrointestinal series; extended upper gastrointestinal series; hypotonic duodenography; intravenous cholangiography; percutaneous transhepatic cholangiography; pneumotomography; arteriography; splenoportography; duodenoscopic retrograde pancreaticography; and isotopic scanning.

There will be panel discussions; practical demonstrations; and an attractive social program.

The official languages are English, German, and French.

For registration and further information please contact, Prof. Dr. H. Anacker, Institut für Röntgendiagnostik der Technischen Universität München, Klinikum rechts der Isar, Ismaningerstrasse 22, 8 München 80, West Germany.

COLLEGIUM INTERNACIONAL DE RADIOLOGIA EN OTORRINOLARINGOLOGIA

The Collegium Internacional de Radiologia en Otorrinolaringologia will be holding its Sixth International Congress of Radiology in Madrid, October 22–24, 1973.

The Organizing Committee, following the indications of the Collegium Directory Committee, now proposes the major subjects for discussion in Round Tables.

The major subjects proposed are: normal vascularization in the internal auditory meatus, and its pathologic changes; role of radiology in the ethiopathogenesis of vertigo; role of external carotid angiography in otorhinolaringology; radiologic examinations of sphenoidal region; diagnosis and treatment of glomus jugulare tumors; and treatment of piriform sinus tumors.

For further details please write to Dr. M. Trujillo, President, or Dr. V. G. Queimadelos, Secretary, Fundacíon Jiménez Diaz. Avda. Reyes Católicos, 2, Ciudad Universitaria, Madrid 8, España.

BRITISH NUCLEAR MEDICINE SOCIETY

The First Annual Meeting of the British Nuclear Medicine Society will be held, April 12 and 13, 1973, in the Middlesex Hospital Medical School, London.

The program will include review and original papers with meetings of general interest, as well as specialized sessions for organ scanning, physics and laboratory investigations.

Please address inquiries to: The Honorary Secretary, Dr. E. R. Davies, Radiodiagnostic Department, Bristol Royal Infirmary, Bristol, BS2 8HW, England.

NOBLE GASES SYMPOSIUM

A Symposium on Noble Gases is being sponsored by the U. S. Environmental Protection Agency and the University of Nevada, Las Vegas, at the Flamingo Hotel in Las Vegas, Nevada, September 24–28, 1973.

The Symposium should provide comprehensive coverage of subjects related to noble gases. The areas to be covered include: natural elemental and isotopic abundance; physical and chemical properties; techniques for detection and measurement; production and environmental levels, including prediction for future developments; environmental releases and processes for reducing environmental releases of radioactive noble gases; biokinetics, bioeffects and dosimetry of radioactive noble gases; environmental standards; radon in uranium mines; health physics; extraterrestrial

noble gases; and application of noble gases.

Deadline for submission of abstracts is April 1, 1973. Manuscripts of papers accepted for presentation must be received by August 15, 1973.

For further information please write to: Dr. A. Alan Moghissi, Chairman of the Program Committee, Noble Gases Symposium, U. S. Environmental Protection Agency, P.O. Box 15027, Las Vegas, Nevada 89114.

SECOND INTERNATIONAL SYMPOSIUM ON CANCER DETECTION AND PREVENTION

The Second International Symposium on Cancer Detection and Prevention, promoted by the International Study Group on Detection and Prevention of Cancer (De P Ca), under the auspices of the International Union against Cancer (UICC) and the International Agency for Research on Cancer (IARC) of the World Health Organization, and organized by the Istituto di Oncologia "F. Addarii" and Bologna Cancer Centre, will be held in Bologna from April 9 to 12, 1973.

The program includes:

1. Lectures on: cancer prevention by environmental control; occupational carcinogenesis; host factors in oncogenesis; recent advances and perspectives of automation in cytology; aspects of experimental carcinogenesis related to cancer prevention; evaluation of cancer control measures (report from the Sheffield UICC meeting); carcinogenic hazards from drugs; and the possible importance of nitrosamines as carcinogens in humans.

2. Panels on: (a) scientific bases and possibilities of detection and prevention of cancer of the breast, lung, gastrointestinal tract, urinary tract and uterine cervix; (b) public health aspects in cancer control; and (c) recent advances on tumors with particular racial and geographic distribution (oral cancer, Burkitt's lymphoma, nasopharyngeal cancer, esophageal cancer, liver cancer).

3. Proferred paper sessions on the same topics as those of panels and on other fields of cancer detection and prevention.

Workshops for registered participants are planned the 2 days after the Symposium (April 13 and 14) on: epidemiologic techniques, cell pathology, thermography, testing of environmental carcinogens, cytogenetics, needle aspiration cytology, gastric cytology.

The official languages are English and French.

For further information please write to: Secretariat, Istituto di Oncologia "F. Addarii," Viale Ercolani 4/2-40138 Bologna, Italy.

It is with deep regret that we announce the death, on July 30, 1972, at St. Mary's Hospital in Rhinelander, Wisconsin, the town of his birth, of Dr. Edward L. Jenkinson, distinguished member and Past President of the American Roentgen Ray Society.

BOOK REVIEW

Illustrated Guide to X-Ray Technics. By John E. Cullinan, R.T., F.A.S.R.T., Administrative Assistant for Technical Affairs, Division of Radiology, Albert Einstein Medical Center, Philadelphia, Pa. Cloth. Pp. 204, with 150 illustrations. Price, $18.00. J. B. Lippincott Company, East Washington Square, Philadelphia, Pa. 19105, 1972.

This text is intended to make available to the radiologic technologist information which would enable the understanding of basic knowledge and the comprehension of the technical advances, as they occur in the field of diagnostic radiology. Although the physical principles are adequately surveyed, this is primarily not in the province of this text. Emphasis is placed upon the reason for a specific piece of equipment or accessory in combination with a specific technique, along with the advantages and limitations of each. Every day basic accessories are explained. The more complex procedures and equipment, including image intensifiers, cineroentgenography, serial film changers and video tape recorders are described.

The book consists of 12 sections including basic image detection principles, control of secondary radiation, roentgenographic tubes, direct roentgen enlargement techniques and exposure timing devices in the first 5 chapters. A sixth chapter is devoted to a general review of technical factors. The last 4 sections include discussions of mobile or bedside roentgenography, body section roentgenography, rapid serial film and cassette changers and basic electronic image detector principles.

The book is extremely well written and all of the material is explained in a lucid and easily read manner. The illustrations are superb and the accompanying teaching diagrams are outstanding. The book contains a wealth of useful information presented in simple terminology, particularly in areas that are quite complex. Newer advances are detailed and the chapters on direct enlargement techniques and body section roentgenography are extremely well presented.

In view of the ever increasing sophistication of instrumentation and techniques, the objective of the author is to assist both the technologist and the practicing radiologist to understand better the complexities of roentgenographic equipment and accessories, resulting in their more effective use. The author easily achieves this goal and covers a vast amount of material with an authoritative over-view of all areas.

This book is highly recommended not only as a valuable addition to the library of both technologists and radiologists, but also for all physicians using diagnostic roentgenographic equipment.

E. Nicholas Sargent, M.D.

BOOKS RECEIVED

Mental Retardation: An Atlas of Diseases with Associated Physical Abnormalities. By Lewis B. Holmes, M.D., Assistant Professor of Pediatrics, Harvard Medical School; Genetics Unit, Children's Service, Massachusetts General Hospital; Clinical Geneticist, Eunice Kennedy Shriver Center, Walter E. Fernald State School, Waverley, Mass.; Hugo W. Moser, M.D., Associate Professor of Neurology, Harvard Medical School; Neurologist, Massachusetts General Hospital; Assistant Superintendent, Walter E. Fernald State School; Co-Director, Eunice Kennedy Shriver Center, Walter E. Fernald State School, Waverley, Mass.; Saevar Halldórsson, M.D., Consultant in Pediatrics, Department of Pediatrics, University Hospital, Reykjavik, Iceland; Consultant Kópavogshaeli, Central Institution for the Mentally Retarded in Iceland; Pediatrician, Department of Pediatrics, St. Joseph's Hospital, Reykjavik, Iceland; Formerly, Clinical and Research Fellow in Pediatrics, Children's Service, Massachusetts General Hospital, and Department of Pediatrics, Harvard Medical School; Cornelia Mack, M.S., Program Specialist, Division of Family Health Service, Department of Public Health, Commonwealth of Massachusetts; Formerly, Research Assistant, Genetics Unit, Children's Service, Massachusetts General Hospital; Shyam S. Pany, M.D., Neurologist, Holy Family Hospital and Tirathram Shah Hospital, New Delhi, India; Formerly, Clinical and Research Fellow in Neurology and Neuropathology, Neurology Service, Massachusetts General Hospital and Department of Neurology, Harvard Medical School; and Benjamin Matzilevich, M.D., Instructor in Neurology, Harvard Medical School; Clinical Director, Walter E. Fernald State School, Waverley, Mass. Cloth. Pp. 430, with many illustrations. Price, $28.00. The Macmillan Company, 866 Third Avenue, New York, N. Y. 10022, 1972.

Practical Procedures in Diagnostic Radiology. By H. M. Saxton, M.B., M.R.C.P., F.F.R., D.M.R.D., Consultant Radiologist, Guy's Hospital, London; and Basil Strickland, F.R.C.P., F.F.R., F.A.C.R. (Hon.), Consultant Radiologist, Westminster Hospital, London; Consultant Radiologist, Brompton Hospital, London. Second edition. Cloth. Pp. 305, with 48 illustrations. Price, $15.00. Grune & Stratton, Inc., 111 Fifth Avenue, New York, N. Y. 10003, 1972.

Atlas of Nuclear Medicine. Volume 3: Reticuloendothelial System, Liver, Spleen and Thyroid. By Frank H. DeLand, M.D., Professor of Radiology, Department of Radiology, University of Florida College of Medicine, Gainesville, Fla.; Formerly, Associate Professor of Radiology and Radiological Sciences, Johns Hopkins Medical Institutions; and Henry N. Wagner, Jr., M.D., Professor of Medicine, Radiology and Radiological Sciences, Johns Hopkins Medical Institutions; with the assistance of Wendy A. North, M.I.R., Editorial Assistant, Johns Hopkins Medical Institutions. Cloth. Pp. 292, with 196 illustrations. Price, $21.00. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1972.

Fundamentals of the Tracer Method. By Teresa J. C. Welch, M.S., Assistant Professor, Department of Chemistry, Lindenwood College, St. Charles, Mo.; E. James Potchen, M.D., Professor of Radiology; Director of Nuclear Medicine, Mallinckrodt Institute of Radiology, Washington University School of Medicine, St. Louis, Mo.; and Michael J. Welch, Ph.D., Associate Professor of Radiation Chemistry in Radiology, Mallinckrodt Institute of Radiology, Washington University School of Medicine, St. Louis, Mo. Cloth. Pp. 187, with many illustrations. Price, $9.75. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1972.

Metabolic, Degenerative, and Inflammatory Diseases of Bones and Joints. By Henry L. Jaffe, M.D., Consultant Pathologist and Emeritus Director of Laboratories, Hospital for Joint Diseases, New York City; Consultant Pathologist, United Hospitals of Newark, N. J.; Honorary Consultant, Armed Forces Institute of Pathology, Washington, D. C. Cloth. Pp. 1,101, with 1,066 illustrations. Price, $35.00. Lea & Febiger, Washington Square, Philadelphia, Pa. 19106, 1972.

Cardiac Clinicopathological Conferences of the Massachusetts General Hospital. Edited by Benjamin Castleman, M.D., Chief, James Homer Wright Pathology Laboratories, Massachusetts General Hospital; Shattuck Professor of Pathological Anatomy, Harvard Medical School; and Roman W. DeSanctis, M.D., Physician, Massachusetts General Hospital; Clinical Professor of Medicine, Harvard Medical School; with a foreword by Paul Dudley White, M.D., Honorary Physician, Massachusetts General Hospital; Clinical Professor of Medicine, Emeritus, Harvard Medical School. Cloth. Pp. 464, with 54 illustrations. Price, $28.50. Little, Brown & Company, 34 Beacon Street, Boston, Mass. 02106, 1972.

Diagnosis of Peripheral Vascular Disease. By A. Kappert, M.D., Professor of Clinical Angiology, University of Bern, Switzerland; and T. Winsor, M.D., Associate Professor of Medicine, University of Southern California, Los Angeles, Calif. Cloth. Pp. 314, with 368 illustrations. Price, $32.00. F. A. Davis Company, 1915 Arch Street, Philadelphia, Pa. 19103, 1972.

Traité de Radiodiagnostic. Tome XIII: Neuroradiologie. Volume I. By H. Fischgold; Radioanatomie by G. Salamon, G. Guérinel and R. Louis; Méthodes d'Exploration by J. Metzger, J. Legré, G. Salamon, A. Wackenheim and D. Doyon. Cloth. Pp. 510, with 500 figures. Price, 375 F. Masson & Cie, Paris, France, 1972.

Effect of Dimethyl Sulfoxide (DMSO) on Cytoplasmic Barrier of Malignant Epidermal Cells: an investigation in skin-tumor-resistant mice. By Perttu Metsälä, Department of Pathology, and Radiologic Laboratory, University of Helsinki, Finland. Acta Radiologica Supplementum 307. Paper. Pp. 122, with many illustrations. Price, Sw. Kr. 35:-. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1971.

Mechanism of Blood Clearance of Colloidal Gold in Mice: an atoxic clinical investigation using activation analysis. By Jørgen Rygård, Radium Centre, Copenhagen, Denmark. Acta Radiologica Supplementum 308. Paper. Pp. 240, with some figures. Price, Sw. Kr. 35:-. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1971.

A Mathematical Model for Radiation Fields of Telecobalt Treatment Units: with special reference to the isodoses of Rocus. By Lauri Patomäki, Radiotherapy Clinic, University Central Hospital, Helsinki, Finland. Acta Radiologica Supplementum 309. Paper. Pp. 82, with some figures. Price, Sw. Kr. 35:-. Acta Radiologica, Box 2052, Stockholm 2, Sweden, 1971.

Die Ultraschalldiagnostik in der Schwangerschaft. By Priv.-Doz. Dr. med. Hans-Jürgen Holländer, Oberarzt der Univ.-Frauenklinik, Münster (Westf.); with a foreword by Prof. Dr. med. Günther Reiffenstuhl, Direktor der Univ.-Frauenklinik, Münster (Westf.). Cloth. Pp. 181, with 112 figs. Urban & Schwarzenberg, München, 1972.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga. 30322. Annual Meeting: Washington-Hilton Hotel, Washington, D. C., October 3–6, 1972.

AMERICAN RADIUM SOCIETY
Secretary, Dr. Felix N. Rutledge, M. D. Anderson Hospital and Tumor Institute, Houston, Texas 77025. Annual Meeting: Broadmoor Hotel, Colorado Springs, Colorado, April 22–26, 1973.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. Hillier L. Baker, Jr., Mayo Clinic, Rochester, Minn. 55901. Annual Meeting: Palmer House, Chicago, Ill., November 26–December 1, 1972.

AMERICAN COLLEGE OF RADIOLOGY
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago, Ill. 60606. Annual meeting: San Francisco, Calif., April 2–7, 1973.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. Antolin Raventos, Davis, Calif. Annual Meeting: New York, N. Y., June 24–28, 1973.

AMERICAN BOARD OF RADIOLOGY
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.

Oral examinations will be held in the following cities during the next 2 years: Atlanta, Ga., Dec. 4–8, 1972, Sheraton-Biltmore Hotel; Chicago, Ill., June 4–8, 1973, Marriott Motor Hotel; and Dallas, Tex., Dec. 3–7, 1973, Statler Hilton Hotel.

Written examinations are scheduled in 13 large centers.

Applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.

Deadline for filing applications for any examination in 1973 is September 30, 1972.

AMERICAN BOARD OF NUCLEAR MEDICINE, INC. A Conjoint Board of the American Boards of Internal Medicine, Pathology and Radiology and sponsored by the Society of Nuclear Medicine.
Chairman, Dr. Merrill A. Bender, Suite 2402, 211 E. 43rd St., New York, N. Y. 10017.

AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE
Secretary, D. John Wright, Ph.D., Department of Radiology, Temple Univ. Health Sciences Center, Philadelphia, Pa. 19140. Annual Meeting in Chicago, Ill., Nov. 30 and Dec. 1, 1972.

AMERICAN SOCIETY OF THERAPEUTIC RADIOLOGISTS
Secretary, Dr. Herman D. Suit, Radiation Medicine, Massachusetts General Hospital, Boston, Mass. 02114. Annual Meeting: Biltmore Hotel, Phoenix, Ariz., November 1–4, 1972.

AMERICAN INSTITUTE OF ULTRASOUND IN MEDICINE
Secretary, John M. Reid, Ph.D., 556 18th Ave., Seattle, Wash. 98122. Annual Meeting, Philadelphia, Pa., Oct. 29–Nov. 1, 1972.

AMERICAN SOCIETY OF NEURORADIOLOGY
Secretary, Dr. David O. Davis, Department of Radiology, Univ. of Utah Medical Center, Salt Lake City, Utah 84112. Annual Meeting, Boston, Mass., April 26–28, 1973.

THIRTEENTH INTERNATIONAL CONGRESS OF RADIOLOGY
President-Elect, Dr. Juan Gómez López. *Secretary General*, Dr. José Bonmatí, Lagasca, 27 Madrid 1, España. Meeting: Madrid, Spain, Oct. 15–20, 1973.

INTERNATIONAL SOCIETY OF RADIOLOGY
Hon. Secretary-Treasurer, Prof. Eric Samuel, Radiologist-in-Charge, The Royal Infirmary, Edinburgh EH3 9Y-W, Scotland.

ELEVENTH INTER-AMERICAN CONGRESS OF RADIOLOGY
Counselor for the United States, Dr. Juan A. del Regato, Penrose Cancer Hospital, 2215 North Cascade Ave., Colorado Springs, Colorado 80907. Meeting: Bogotá, Colombia in 1975.

INTER-AMERICAN COLLEGE OF RADIOLOGY
President, Dr. Miguel Dao y Dao, Apartado Postal 14213, Candelaria, Caracas, Venezuela, S.A.
Secretary General, Dr. Rubén Merenfeld, Instituto Diagnóstico, Apartado Postal 14213, Candelaria, Caracas, Venezuela, S.A. Meeting: Bogotá, Colombia in 1975.

THIRD CONGRESS OF THE EUROPEAN ASSOCIATION OF RADIOLOGY
Meeting in June, 1975 in Edinburgh, Scotland.

SECOND ASIAN AND OCEANIAN CONGRESS OF RADIOLOGY
Honorary Secretary, Dr. J. J. Martin, Box 805 F., G.P.O., Melbourne, 3001, Australia.
Meeting in 1975.

ALABAMA CHAPTER OF ACR
Secretary-Treasurer, Dr. William V. Weldon, Medical Arts Building Birmingham, Ala. 35205. Meets time and place of Alabama State Medical Association.

ALASKA RADIOLOGICAL SOCIETY
Secretary, Dr. Bruce C. Wright, Providence Hospital, Anchorage, Alaska. Meets third Wednesday each month.

AMERICAN THERMOGRAPHIC SOCIETY
Secretary-Treasurer, Dr. Marc. S. Lapayowker, Department of Radiology, Temple Univ. Hosp., 3401 N. St., Philadelphia, Pa. 19140.

ARIZONA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Wesley S. Fee, 2421 E. 6th St., Tucson, Ariz. 85719. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

ARK-LA-TEX RADIOLOGICAL SOCIETY
Secretary, Dr. Erich K. Lang, Confederate Memorial Medical Center, LSU School of Medicine, Shreveport, La. 71101.

ARKANSAS CHAPTER OF ACR
Secretary-Treasurer, Dr. David H. Newbern, 4301 W. Markham, Little Rock, Ark. 72205. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS
Secretary-Treasurer, Dr. Melvyn H. Schreiber, 2909 Dominique Dr., Galveston, Tex. Annual Meeting, Vancouver, B. C., Canada, May, 1973.

ATLANTA RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Ernst Ruder, Dept., of Radiol., Crawford Long Memorial Hosp., Atlanta, Ga. 30308. Meets on four Thursday evenings during the academic year at a time announced in early September of each year, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

BAVARIAN-AMERICAN RADIOLOGIC SOCIETY
Secretary, Lucien F. Burke, Maj. MC, Radiol. Service, US Army Gen. Hosp., Frankfurt, APO, New York 09757.

BLOCKLEY RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. R. John Gould, 41 Lombardy Rd., Drexel Hill, Pa. 19026.

BLUEGRASS RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Eugene Slusher, Lexington

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

Clinic, 1221 S. Broadway, Lexington, Ky. 40504. The Society meets once each month during the school year.

Bronx Radiological Society, New York State, Chapter ACR
Secretary-Treasurer, Dr. Leon J. Corbin, 1369 Rosedale Ave., Bronx, N. Y. 10472. Meets 4 times a year.

Brooklyn Radiological Society
Secretary-Treasurer, Dr. Sarah Lieberson, 1298 St. Marks Ave., Brooklyn, N. Y. Meets first Thursday of each month, October through June.

Buffalo Radiological Society
Secretary, Dr. Charles J. Riggio, 5301 Willow Lake Ct., Clarence, N. Y. Meets second Monday evening each month, October to May inclusive, at University Club.

California Radiation Therapy Association
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets semiannually in San Francisco and Los Angeles.

California Radiological Society, California Chapter of ACR
Secretary-Treasurer, Dr. John L. Gwinn, 4650 Sunset Blvd., Los Angeles, Calif. 90027.

Catawba Valley Radiological Society
Secretary, Dr. Emmett R. White, P.O. Box 10, Rutherford College, N. C. 28671. Meets every Thursday, Dept. of Radiology, Valdese General Hosp., Valdese, N. C., at 12:30 P.M.

Central New York Radiological Society
Secretary-Treasurer, Dr. John Sanborn, Radiology Department, Crouse-Irving Memorial Hosp., Irving Ave., Syracuse, N. Y. 13210. Meets first Monday each month October through May.

Central Ohio Radiological Society
Secretary-Treasurer, Dr. James V. Blazek, 2586 Lane Rd., Columbus, Ohio 43220. Meets second Thursday in October, November, January, and March 15, and May 19 at Fort Hayes Hotel, Columbus, Ohio.

Chicago Radiological Society, Division of the Illinois Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Raymond L. Del Fava, 355 Ridge Ave., Evanston, Ill. 60201. Meets third Thursday of each month, October to April, except December, at the Bismarck Hotel, Chicago, Ill.

Cleveland Radiological Society
Secretary-Treasurer, Dr. Daniel E. Wertman, 11311 Shaker Blvd., Cleveland, Ohio 44104. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and April.

Colorado Radiological Society, Chapter of ACR
Secretary, Dr. Jerome D. Sutherland, 3705 E. Colfax Ave., Denver, Colo. 80206. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society
Secretary, Dr. William W. Walthall, Jr., 130 Maple St., Springfield, Mass. Meets in April and October.

Dallas-Fort Worth Radiological Society
Secretary-Treasurer, Dr. Norman L. Presley, 222 Medical Tower, 1550 W. Rosedale, Fort Worth, Tex. 76104. Meets the 3rd Monday of every month at 6:30 P.M., at the Cibola Inn, Arlington, Tex.

Delaware Chapter of ACR
Secretary, Dr. James H. Taylor, Wilmington Medical Center, Wilmington, Del. 19899.

East Bay Radiological Society
Secretary-Treasurer, Dr. Peter E. Kane, Children's Hosp. Med. Ctr., Oakland, Calif. 94609. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

East Tennessee Radiological Society
Secretary-Treasurer, Dr. George H. Wood, 205 Medical Arts Bldg., Knoxville, Tenn. 37902. Meets in January and September.

Florida Radiological Society, Chapter of ACR
Secretary, Dr. Ralph C. Aye, Tampa General Hospital, Tampa, Fla. 33606. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. J. C. Hewitt, 1 Davis Blvd., Tampa, Fla. 33606. Meets in January, March, May, July, September and November.

Georgia Radiological Society, Chapter of ACR
Secretary, Dr. Richard S. Colvin, Emory University Clinic, Atlanta, Ga. 30322. Meets in spring and fall at Annual State Society Meeting.

Greater Cincinnati Radiological Society
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

Greater Louisville Radiological Society
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

Greater Miami Radiological Society
Secretary-Treasurer, Dr. Stanley I. Worton, 1400 N.W. 10th Avenue, Miami, Florida 33136. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

Greater St. Louis Society of Radiologists
Secretary-Treasurer, John J. Lang, 507 Fairways Circle, St. Louis, Mo. 63141.

Hawaii Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Robert DiMauro, Kauikeolani Children's Hosp., P.O. Box 3799, Honolulu, Hawaii 96812. Meets third Monday of each month at 7:30 P.M.

Health Physics Society
Secretary, John H. Pingel, Argonne National Laboratory, 9700 S. Cass Ave., Argonne, Ill. 60439. Annual Meeting: Deauville Hotel, Miami Beach, Fla., June 17–21, 1973.

Houston Radiological Society
Secretary, Dr. Neill B. Longley, 6436 Fannin, Suite 601, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025.

Idaho State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Hugh P. Smith, Jr., 130 E. Bannock, Boise, Id. 83702. Meets in the spring and fall.

Illinois Radiological Society, Inc., Chapter of ACR
Secretary, Dr. B. Jay Hill, Mercy Hospital, Stevenson at King Drive, Chicago, Ill. 60616. Meets in the spring and fall.

Indiana Roentgen Society, Inc., Chapter of ACR
Secretary, Dr. L. Ray Stewart, 852 S. Alvord Blvd., Evansville, Ind. 47714.

Iowa Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John Huston Jr., 1948 First Ave. N.E., Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Wm. R. Allen, 155 S. 18th St. Kansas City, Kan. 66102. Meets in spring with State Medical Society and in winter on call.

Kentucky Chapter of ACR
Secretary-Treasurer, Dr. Joseph G. Whelan, Jr., 1313 St. Antony Place, Louisville, Ky. 40204. Meets in April and September.

Kings County Radiological Society
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N.Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Knoxville Radiological Society
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Long Island Radiological Society
Secretary, Dr. Jerome Zwanger, 1200 Stewart Ave., Garden City, N. Y. 11533. Meets monthly.

Los Angeles Radiological Society
Secretary, Dr. Mathew E. O'Keefe, 13303 E. Hadley St. Whittier, Calif. 90601. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif. Midwinter Radiological Conference, Century Plaza Hotel, Los Angeles, Calif. 90067, date to be announced.

Louisiana Radiological Society
Secretary-Treasurer, Dr. Robert T. Cook, Southern Baptist Hosp., 2700 Napoleon Ave., New Orleans, La. 70115.

Louisiana-Texas Gulf Coast Radiological Society
Secretary-Treasurer, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Tex. 77701.

Maine Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Richard W. Taylor, Radiology Department, St. Mary's General Hospital, Lewiston, Maine 04240. Meets in June, September, December and April.

Maryland Radiological Society, Chapter of ACR
Secretary, Dr. George Burke, 4000 North Charles St., Baltimore, Md. 21218.

Massachusetts Radiological Society, Chapter of ACR
Secretary, Dr. Norman L. Sadowsky, The Faulkner Hosp., 1153 Centre St., Jamaica Plain, Mass. 02130.

Memphis Roentgen Society
Secretary-Treasurer, Dr. Jerry C. Phillips, The University of Tennessee College of Medicine, Department of Radiology, Walter C. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38103. Meets first Monday Oct. through May at the University Club.

Miami Valley Radiological Society
Secretary, Dr. Darwood B. Hance, Reid Memorial Hospital, Richmond, Ind. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

Michigan Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. George A. Kling, Harper Hospital, 3825 Brush St., Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

Mid-Hudson Radiological Society
Secretary-Treasurer, Dr. Herbert S. Berlin, Hopewell Junction, N. Y. Meets 7:00 P.M., first Wednesday of each month, September to May.

Milwaukee Roentgen Ray Society
Secretary-Treasurer, Dr. Thomas C. Lipscomb, 1004 North Tenth St., Milwaukee, Wis. 53233. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Harlan W. Hawkinson, 6 Buffalo Rd., St. Paul, Minn. 55110. Meets twice annually, fall and winter.

Mississippi State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Nancy W. Burrow, 1327 Peachtree St., Jackson, Miss. 39202. Meets third Thursday of each month at the Heidelberg Hotel, Jackson, at 6:00 P.M.

Missouri Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ronald G. Evens, Mallinckrodt Institute of Radiology, St. Louis, Mo. 63110.

Montana Radiological Society, Chapter of ACR
Secretary, Dr. Jon A. Anderson, Doctor's Building, 1231 N. 29th Street, Billings, Mont. 59101. Biennial Symposium at the Holiday Inn, Bozeman, Mont., July 28–30, 1971.

Nebraska Chapter of ACR
Secretary-Treasurer, Dr. N. Patrick Kenney, 3618 S. 114th Ave., Omaha, Nebr. 68144. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

Nevada Radiological Society, Chapter of ACR
Secretary, Dr. William G. Arbonies, 887 Marsh, Reno, Nev. 89520.

New England Roentgen Ray Society
Secretary, Dr. Stefan C. Schatzki, 1180 Beacon St. Brookline Mass. 02146. Meets the third Friday of each month, October through April, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 4:30 P.M.

New Hampshire Roentgen Ray Society, Chapter of ACR
Secretary-Treasurer, Dr. Edward P. Kane, Claremont Gen. Hosp., Claremont, N. H. 03743. Meets four to six times yearly.

New Mexico Society of Radiologists Chapter of ACR
Secretary, Dr. Carole R. Simmons, Dept. of Radiol., Presbyterian Hospital Center, Albuquerque, N. M. 87106. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

New Orleans Radiological Society, Inc.
Secretary-Treasurer, Dr. Jimmy L. Mains, P.O. Box 446, Gretna, La. 70053. Meets bimonthly at local restaurants selected by the President.

New York Roentgen Society
Secretary-Treasurer, Dr. Howard R. Gould, 153 West 11th Street, New York, N. Y. 10011. Meets monthly on the 3rd Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Conference, Waldorf-Astoria Hotel, New York, N. Y., Apr. 26–28, 1973. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

New York State Chapter of ACR
Secretary-Treasurer, Dr. John J. Magovern, 74 N. Village Ave., Rockville Centre, N. Y. 11570.

North Carolina Chapter of ACR
Secretary-Treasurer, Dr. James F. Martin, 300 S. Hawthorne Road, Winston-Salem, N. C. 27103.

North Dakota Radiological Society, Chapter of ACR
Secretary, Dr. Marshall Landa, 1702 13th St., So., Fargo, N. D. 58102. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. David F. Bew, University Hospital of Jacksonville, 655 W. Eighth Street, P.O. Box 2751, Jacksonville, Florida 32203. Meets quarterly in March, June, September and December.

Northeastern Oklahoma Radiological Society
Secretary-Treasurer, Dr. Theodore J. Brickner, Jr., 5919 S. Gary Place, Tulsa, Okla. 74110.

Northeastern New York Radiological Society
Secretary, Dr. Robert C. Friedman, 325 Presidential Way, Guilderland, N. Y. Meets in Albany area on third Wednesday of October, November, March, April, and May.

Northern California Radiation Therapy Association
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets quarterly.

Northern California Radiological Society
Secretary, Dr. Julian B. Holt, 3704 Mission Ave., Suite 26, Carmichael, Calif. 95608. Meets fourth Monday of Sept., Nov., Jan., March and May at Aldo's Restaurant in Sacramento.

Northwestern New York Radiological Society
Secretary, Dr. Barbara E. Chick, Glens Falls Hospital, Glens Falls, N. Y. 12801.

Northwestern Ohio Radiological Society
Secretary, Dr. Vito J. Zupa, Mercy Hospital, Department of Radiology, Toledo, Ohio.

Ohio State Radiological Society, Chapter of ACR
Secretary, Dr. Thomas F. Ulrich, Barberton Citizens Hospital, Barberton, Ohio 44203.

Oklahoma State Radiological Society, Chapter of ACR
Secretary, Dr. Richard B. Price, 204 Medical Tower Bldg., Oklahoma City, Okla. 73112. Meets in January, May and October.

ORANGE COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Norman Arnett, 400 North Pamona, Fullerton, Calif. 92632. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at the Orange County Medical Association Bldg., Orange, Calif.

OREGON STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Ronald W. Philips, 2305 Washington, Milwaukie, Ore. 97222. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

ORLEANS PARISH RADIOLOGICAL SOCIETY
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

PACIFIC NORTHWEST RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Robert S. Miller, 13753 S.W. Farmington Rd., Beaverton, Oregon 97005. Annual meeting.

PENNSYLVANIA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Theodore A. Tristan, Harrisburg Polyclinic Hosp., Harrisburg, Pa. 17105.

PHILADELPHIA ROENTGEN RAY SOCIETY
Secretary, Dr. Howard M. Pollack, Department of Radiology, Episcopal Hospital, Philadelphia, Pa. 19125. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

PITTSBURGH ROENTGEN SOCIETY
Secretary, Dr. Stephen C. Bruno, Shadyside Hospital, 5230 Centre Ave., Pittsburgh, Pa. 15232. Meets second Wednesday of month, October through June, at Park Schenley Restaurant.

RADIATION RESEARCH SOCIETY
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016. Annual Meeting: St. Louis, Mo., April 29–May 2, 1973.

RADIOLOGICAL SOCIETY OF CONNECTICUT, INC., CHAPTER OF ACR
Secretary-Treasurer, Dr. Carl W. Scheer, 335 Cook Ave., Meriden, Conn. 06450. Meetings are held quarterly.

RADIOLOGICAL SOCIETY OF GREATER CINCINNATI
Secretary-Treasurer, Dr. Alvin Nathan, c/o Academy of Medicine, 320 Broadway, Cincinnati, Ohio 45202. Meets first Monday of each month at Cincinnati Academy of Medicine.

RADIOLOGICAL SOCIETY OF GREATER KANSAS CITY
President-Secretary, Dr. Arch W. Templeton, Univ. of Kansas Med. Ctr., Kansas City, Kansas 66103. Meets 5 times a year on given dates.

RADIOLOGICAL SOCIETY OF LOUISIANA, CHAPTER OF ACR
Secretary-Treasurer, Dr. Robert T. Cook, 1820 Octavia St., New Orleans, La. 70115. Meets semiannually during Louisiana State Medical Society meeting and 6 months later.

RADIOLOGICAL SOCIETY OF NEW JERSEY, CHAPTER OF ACR
Secretary, Dr. Sidney Ketyer, St. Elizabeth Hosp., 225 Williamson St., Elizabeth, N. J. 07207. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

RADIOLOGICAL SOCIETY OF RHODE ISLAND, CHAPTER OF ACR
Secretary-Treasurer, Dr. John J. O'Brien, 593 Eddy St., Providence, R. I. 02903.

RADIOLOGICAL SOCIETY OF SOUTHERN CALIFORNIA
Secretary-Treasurer, Dr. E. Nicholas Sargent, 1200 North State St., Box 677 Los Angeles, Calif. 90033. Meets three times a year, usually October, February and May.

RADIOLOGICAL SOCIETY OF THE STATE OF NEW YORK
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

REDWOOD EMPIRE RADIOLOGICAL SOCIETY
Secretary, Dr. Lee F. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

RICHMOND COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Donald P. King, 218 Gun Club Rd., Richmond, Va. 23221. Meets first Thursday of each month at various hospitals.

ROCHESTER ROENTGEN RAY SOCIETY, ROCHESTER, N. Y.
Secretary-Treasurer, Dr. Kenneth E. Robinson, 1441 East Avenue, Rochester, New York 14610. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual meeting: Brown Palace Hotel, Denver, Colo., Aug. 17–19, 1972.

SAN ANTONIO-CIVILIAN MILITARY RADIOLOGICAL SOCIETY
Secretary, Dr. Robert M. Maurer, Santa Rosa Medical Center, San Antonio, Tex. 78207. Meets third Wednesday of each month at Fort Sam Houston Officers' Club at 6:30 P.M.

SAN DIEGO RADIOLOGICAL SOCIETY
President-Secretary, Dr. James M. Lee, Scripps Clinic and Research Found., 476 Prospect St., La Jolla, Calif. 92037. Meets first Wednesday of each month at the Town & Country Hotel.

SAN FRANCISCO RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. James G. Moore, Mary's Help Hospital, 1900 Sullivan Ave., Daly City, Calif. 94015.

SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

SECTION ON RADIOLOGY, MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA
Secretary-Treasurer, Dr. Louis Wener, Cafritz Memorial Hosp., 1310 Southern Ave., S.E., Washington, D. C. 20032. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

SECTION ON RADIOLOGY, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Mario A. Calonje, 1514 Jefferson Highway, New Orleans, La. 70121. Annual Meeting, New Orleans, La., Nov. 13–16, 1972.

SECTION ON RADIOLOGY, TEXAS MEDICAL ASSOCIATION
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

SHREVEPORT RADIOLOGICAL CLUB
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

SOCIETY FOR PEDIATRIC RADIOLOGY
Secretary, Dr. John L. Gwinn, Children's Hospital, 4614 Sunset Blvd., Los Angeles, Calif. 90027. Annual meeting: Washington-Hilton Hotel, Washington, D. C., October 1–2, 1972.

SOCIETY OF GASTROINTESTINAL RADIOLOGISTS
Secretary-Treasurer, Dr. Wm. B. Seaman, The Presbyterian Hospital, Columbia-Presbyterian Medical Center, 622 W. 168th St., New York, N. Y. 10032.

SOCIETY OF NUCLEAR MEDICINE
Secretary, Dr. E. James Potchen, Edward Mallinckrodt Institute of Radiology, 660 S. Euclid Ave., St. Louis, Mo. 63110. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. Annual meeting.

SOUTH BAY RADIOLOGICAL SOCIETY
Secretary, Dr. A. D. Benjamin, 596 Carroll St., Sunnyvale, Calif. 94086. Meets quarterly at various hospitals (contact Secretary).

SOUTH CAROLINA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

SOUTH DAKOTA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. M. F. Petereit, 2416 S. Phillips, Sioux Falls, S. D. 57105. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Society
Secretary-Treasurer, Dr. John Byfield, 1000 West Carson St., Torrance, Calif. 90502. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 7544, Mobile, Ala. 36607. Seventeenth Annual Meeting: Grand Hotel, Point Clear, Ala., Jan. 26–28, 1973.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Carl T. Stubblefield, Bedford County Hospital, Columbia, Tenn. 38401. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas State Radiological Society, Chapter of ACR
Secretary, Dr. Herman C. Sehested, 815 Medical Tower, Room 100, 1550 W. Rosedale St., Fort Worth, Tex. 76104. Annual meeting.

The Fleischner Society
President, Dr. Robert G. Fraser, 1555 Summerhill Ave., Montreal 109, P.Q., Canada. Annual meeting.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John F. Stucki, Department of Radiology, McKay-Dee Hosp., 3939 Harrison Blvd., Ogden, Utah 84403. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society, Chapter of ACR
Secretary, Dr. J. Lorimer Holm, R.R.#3, Barre, Vt. 05641.

Virginia Chapter of ACR
Secretary-Treasurer, Dr. James S. Redmond, Suite 7, Medical Center, Lynchburg, Va. 24501. Next Meeting: Nov. 6, 1972 at the Conference Ctr., Williamsburg, Va.

Washington, D. C., Chapter of ACR
Secretary-Treasurer, Dr. Albert M. Zelna, 9110 Seven Locks Rd., Bethesda, Md. 20014.

Washington State Radiological Society, Chapter of ACR
Secretary-Treasurer, to be announced.

West Virginia Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Andrew W. Goodwin, II, 200 Med. Arts Bldg., Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society; other meetings arranged by program committee.

Westchester County Radiological Society
Secretary, Dr. Max C. King, 1276 Fulton Ave., New York, N. Y. 10007. Meets on second Tuesday of October, December, February and May.

Wisconsin Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Marvin L. Hinke, 630 S. Central Ave., Marshfield, Wisc. 54449. Meets twice a year, May and September.

Wisconsin Society of Therapeutic Radiologists
Secretary, Dr. Robert W. Edland, Gundersen Clinic, LaCrosse, Wisc. 54601. Meets quarterly.

Wyoming Radiological Society, Chapter of ACR
Secretary, Dr. Ross J. Collie, Box 96, Lander, Wyo. 82520. Meets in fall with State Medical Society and in spring on call of President.

Mexico, Puerto Rico and Central America

Asociación Costarricense de Radiología
Secretary, Dr. Jorge Vargas Segura, Apartado 5367, San José, Costa Rica.

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá
Secretary-General, Dr. Francisco Mirambell, Apartado 3352, San José, Costa Rica, Central America.
Meets annually in a rotating manner in the six countries.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad de Radiología de El Salvador
Secretary, Dr. Carlos Mejia, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

Sociedad Mexicana de Radiología, A.C.
Coahuila No. 35, México 7, D.F., México.
Secretary-General, Dr. Jesús Rodríguez Carbajal. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. Heriberto Pagán Sáez, Box 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
Moyne, 121 Boul. Taschereau, Greenfield Park, P.Q., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretary, Dr. G. H. du Boulay, 32 Welbeck St., London, W. 1, England. Meets monthly from October until May.

Canadian Association of Physicists, Division of Medical and Biological Physics
Honorary Secretary-Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada.

Canadian Association of Radiologists
Honorary Secretary, Dr. Champlain Charest, 1555 Summerhill Ave., Montreal 109, Que., Canada. Annual meeting: Montreal, Que., Canada, March 11–17, 1973.

Edmonton and District Radiological Society
Secretary, Dr. L. A. Koller, Suite 360, Professional Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada. Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

Faculty of Radiologists
Honorary Secretary, Robert Morrison, 47 Lincoln's Inn Fields, London, W.C.2, England. The scientific meetings held in January, February and March 1973 will be combined sessions with the British Institute of Radiology and the Section of Radiology of The Royal Society of Medicine.

Faculty of Radiologists, Royal College of Surgeons in Ireland
Hon. Secretary, Dr. D. F. Canwell, 123 St. Stephens Green, Dublin 2, Ireland.

Montreal Radiological Study Club
Secretary, Dr. W. Paul Butt, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1, England.

Société Canadienne-Française de Radiologie
Secretary General, Dr. Hubert Sasseville, Verdun Hospital, Montréal, P.Q., Canada. Meets every third Tuesday from October to April.

The Hospital Physicists' Association
Honorary Secretary, J. B. Massey, B.Sc., Christie Hosp. and Holt Radium Institute, Physics Department, Withington, Manchester M20 9BX, England.

Toronto Radiological Society
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September through May.

Royal College of Radiologists of Australasia
Honorary Secretary, Dr. T. P. Loneragan, 147 Macquarie St., Sydney, N.S.W. 2000, Australia.

South America

Colégio Brasileiro de Radiologia
Secretary-General, Dr. Miguel Mario Céntola, Caixa Postal 5984, São Paulo, Brazil.

Federación Argentina de Sociedades de Radiología
Branches of the Federation are: Sociedad de Radiólogos de la Provincia de Córdoba; Sociedad Argentina de Radiología; Asociación Argentina de Radiología; Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario; Sociedad de Radiología y Medicina Nuclear del Centro y Sudeste de la Provincia de Buenos Aires (Mar del Plata); Sociedad Salteña de Radiología y Medicina Nuclear; Sociedad de Radiología de Tucumán; Sociedad de Radiologia del Nordeste Argentino; Sociedad de Radiología de La Plata; and Sociedad de Radiología de San Juan.
Secretary-General-Treasurer, Dr. Roberto Habichayn, Av. Colón 637, Córdoba, Argentina.

Sociedad Argentina de Radiología
Secretary-General, Dr. Armando B. de Onaindia, Santa Fé 1171, Buenos Aires, Argentina. Meets first Wednesday evening, April through December.

Sociedad Boliviana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Armando Rocha Amoédo, Caixa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 p.m. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. Tomás Strauszer, Casilla, 13426 Santiago, Chile. Meets fourth Friday of each month at Av. Santa María at 7:00 p.m.

Sociedad Colombiana de Radiologia
Secretary-General, Dr. Rodrigo Caro Molina, Cali, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología
Secretary, Dr. Carlos Palau Jimenez, Casilla 5469, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Hugo Volpe Rios, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary-General, Dr. Miguel Chiappori Cambana, Colegio Médico del Perú, Casilla Correo, 10307, Lima, Perú. Meets monthly except during January, February, and March.

Sociedad de Radiología del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

Sociedad de Radiología del Nordeste Argentino
Secretary-General, Dr. Francisco J. Velar, Catamarca 561, Corrientes, Argentina.

Sociedad de Radiología de La Plata
Secretary, Dr. Hiram D. Haurigot, Calle 50 No. 374, La Plata, Argentina.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Manuel Gonzáles Maseda, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiología de Pernambuco
Secretary, Dr. Manoel Medeiros Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenología y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Córdoba, Argentina.

Sociedad de Radiología, Radioterapéutica y Medicina Nuclear, de Rosario
Secretary-General, Dr. Federico Frágola, Santa Fe 1798, Rosario, Argentina.

Sociedad Salteña de Radiología y Medicina Nuclear
Secretary, Dr. Julio Luis Baldi, Av. Sarmiento 536, Salta, Argentina.

Sociedad Venezolana de Radiología y Medicina Nuclear
Secretary-General, Dr. Salvador Itriago Borgas, Apartado Postal 9213, Candelaria, Caracas, Venezuela. Meets monthly, third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Röntgen-Gesellschaft
Secretary, Dr. Ernst Kotcher, Wiener Allgemeine Poliklinik Mariannengasse 10, A-1090 Wien, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik.

Société Royale Belge de Radiologie
General Secretary, Dr. Joseph Baeyens, Alost, Belgium. Meets in February, March, May, June, September, October, November and December.

Société Européenne de Radiologie Pédiatrique
Secretary, Dr. P. Hicken, The Children's Hosp., Ladywood Middleway, Birmingham B16 8ET, Great Britain. Tenth Annual Meeting, Birmingham, April 11–13, 1973.
Permanent Secretary, Clément Fauré, Hôpital des Enfants Malades, 149, rue de Sèvres, 75 Paris 15e, France.

Société Française de Radiologie Médicale, Médecine Nucléaire et Electrologie, and its branches: Société du Sud-Ouest, du Littoral méditerranéen, du Centre et Lyonnais, du Nord, de l'Ouest, de l'Est, et d'Alger et du Massif Central. Parent Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru Paris 8e, France.

Société Française de Neuroradiologie
Secretary-General, Professeur agr. R. Djindjian 16, rue de l'Université 75, Paris 7e, France. Annual meeting.

Československá Radiologická Společnost
Secretary, Associate Professor Jaromír Kolář, M.D., Sc.D., Radiological Clinic, Praha 2, U nemocnice 2, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

Deutsche Röntgengesellschaft
President, Professor Dr. med. Walter Frommhold, Director of Medizinisches Strahleninstitut der Universität Tübingen, Röntgenweg 11, 7400 Tübingen, Germany.

Società Italiana di Radiologia Medica e Medicina Nucleare
Secretary, Prof. Giorgio Cittadini, Ospedale S. Martino, 16132 Genova, Italy. Meets annually.

Societas Radiologica Danica
Secretary, Dr. Bent Langefeldt, Tretommervej 20a, DK-8240 Risskov, Denmark.

Nederlandse Vereniging voor Radiologie
Secretary, Dr. J. D. Mulder, Dept. of Rad., Akademisch Ziekenhuis, Leiden, Netherlands.

Scandinavian Radiological Society
Secretary-General, Professor C. E. Unnérus, M.D., Hagalung-Tapiola, Havsvindsvägen 5 C., Finland.

Sociedad Española de Radiología y Electrología Médicas y de Medicina Nuclear
Secretary, Dr. José Bonmatí, Villanueva, 11, Madrid 1, España. Meets every second Friday of each month, Oct. to June, inclusive, in Madrid.

Sociedade Portuguesa de Radiologia e Medicina Nuclear
Secretary-General, Dr. Luís Aires de Sousa, Av. Elias Garcia, 123- 1, Esg.-Lisboa 1-Portugal.

Schweizerische Vereiningung für Radiologie, Nuklearmedizin und Strahlenbiologie (Association Suisse de Radiologie, Médecine Nucléaire et de Radiobiologie)
Secretary, Dr. med. Gustav Schoch, Bethesdaspital, Gellerstrasse 144, 4000 Basel, Switzerland.

Asia

Indian Radiological Association
Secretary, Dr. L. R. Parthasarathy, 44 Constable Road, Madras—23, India.

Indonesian Radiological Society
Secretary, Dr. Gani Iljas Sasmitaatmadja, Radiology Dep., Faculty of Medicine, University of Indonesia, Salemba 6, Djakarta, Indonesia.

Iranian Radiological Society
Secretary, Dr. Issa Yaghmai, P.O. Box No. 14-1151, Teheran, Iran. The Society meets on the second Saturday of each month.

Israel Radiological Society
President, Dr. I. Izkovitch, 2 Wingate Ave., Beit Harofe, Haifa, Israel.

Japanese American Radiological Society
Secretary, Thomas J. Imray, Maj. MC, Chief, Radiol. Dep., US Army Hosp., Camp Zana, Japan, APO San Francisco, Calif. 96343. Meets 3rd Thursday, monthly at various locations in Tokyo.

Philippine College of Radiology
Secretary-Treasurer, Dr. Edmundo V. Villacorta, Box 1284, Commercial Center, Makati, Rizal D-708, Philippines. Meets every 3 weeks on Fridays. Annual convention: Jan. 1973, Manila.

Radiological Society of Thailand
Secretary, Prof. Rojana Suvansuthi, M.D., Ph.D., Depart. of Rad., Sirizaj Hospital, Faculty of Medicine, Mahidol University, Bangkok 7, Thailand.

Africa

Association of Radiologists of West Africa
Honorary Secretary, Dr. T. M. Kolawole, University College Hospital, University of Ibadan, Ibadan, Nigeria.

Radiological Society of South Africa
Secretary, Dr. B. Rosenberg, P.O. Box 3200, Johannesburg, South Africa.

South African International Radiological Congress
Director, Dr. Paul Sneider, P.O. Box 4878, Johannesburg, South Africa.

ABSTRACTS OF RADIOLOGIC LITERATURE

E. FREDERICK LANG, M.D., Editor

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ROENTGEN DIAGNOSIS

Head

Jansen van Wigmont, J. W. Growing skull fractures in children. *Radiol. clin. et biol.*, 1972, *41*, 76–81. (From: Department of Radiology, Leiden University Hospital, Leiden, The Netherlands.)

The author reports a case and reviews the literature on growing skull fractures of childhood.

Growing skull fractures of childhood indicate a post-traumatic syndrome occurring only in young children and consisting of an enlarging defect in the bony skull at the site of the fracture.

The roentgenographic appearance is characterized by the widened fracture and sharply defined somewhat scalloped margins of bone, these occurring within a very short period (few weeks) with damage to the underlying dura and arachnoid.

A review of the different names given to this condition, *e.g.*, leptomeningeal cysts, is followed by an emphasis on the name "growing skull fractures."

Analysis of experimental studies indicates that the fundamental factor in these cases is always a lesion of the dura and arachnoid in the form of a tear or a rent.

The case reported is a 2 year and 4 month old child, who sustained a linear parietal fracture following a fall from a height of 5 m. and who in a period of 2 years apparently insidiously developed the growing fracture and had it discovered following minor trauma and roentgenography for that purpose.

Cranioplasty and closure of the dura defect was done on an elective basis.

The author suggests that it may well be desirable in the future to have skull fractures in children under the age of 8 years followed by roentgenography after 12 weeks in order to avoid missing growing fractures. —*Lionel W. Young, M.D.*

Ice, R. D., Updegrave, W. J., and Bogucki, E. I. Influence of dental radiographic cones on radiation exposure. *J. Am. Dent. A.*, Dec., 1971, *83*, 1297–1302. (Address: R. D. Ice, Ph.D., Nuclear Medicine Section, University Hospital, University of Michigan, Ann Arbor, Mich. 48104.)

Skin exposures from 65 kvp. and 90 kvp. dental radiographic units, using long cones, long lead-lined cones, short cones, short lead-lined cones, and sharp pointed cones were evaluated.

Comparisons were made in 100 patients who each had had a full mouth series of roentgenograms (20 films).

Radiation measurements were made with thermoluminescent dosimeters positioned on the patient's body.

The anatomic regions measured were: parotic gland; thyroid gland; gonadal, premolar, occipital, subcorneal, and central forehead areas. The exposure to the operator was also measured.

Analysis of variance indicated a significant difference in radiation exposure at both 65 kvp. and 90 kvp., as well as significant differences between cones.

Comparisons were made between 65 kvp. and 90 kvp. with respect to the total radiation exposure.

The results indicate that the lead-lined cones are the most effective in reducing skin exposure from scatter radiation in clinical dental radiography.—*Eugene C. Klatte, M.D.*

Lamarque, J. L., Ginestié, J. F., Serrou, B., Fabre, J. M., and Sénac, J. P. Apport de l'artériographie carotidienne externe dans l'exploration radiologique des tumeurs du sinus maxillaire. (Contribution of external carotid arteriography to the radiologic study of tumors of the maxillary sinus.) *J. de radiol., d'électrol. et de méd. nucléaire*, June-July, 1971, *52*, 357–366. (From: Service Central de Radiodiagnostic, Clinique Saint-Eloi, 34-Montpellier, France.)

The external carotid angiogram, by visualizing the internal maxillary artery, enables a clear demonstration of the posterior extension of pathology of the maxillary sinus. It will also become a channel for chemotherapy if such a treatment is judged to be useful.

The external carotid artery starts between the levels of C-3 and C-5, has 7 lateral branches (superior thyroid, lingual, facial, ascending pharyngeal, occipital, posterior auricular and parotid arteries) and 2 terminals; *i.e.*, the superficial temporal and the internal maxillary artery.

Under general anesthesia, the superficial temporal artery is catheterized proximally to 3 cm. down the bifurcation of the external carotid artery, and arteriograms are taken at the rate of 2 per second during the first 3 seconds, and 1 per second thereafter, for 10 more seconds, in each of the lateral, occipital and forehead-nose positions. Routine roentgenograms in the same positions are made for subtraction purposes.

The lateral arteriograms were most useful in the experience of the authors, as they could demonstrate clearly the so-called surgical arterial circle in the pterygo-maxillary fossa, made of infraorbital, alveolar and superior palatine branches of the internal maxillary artery.

Out of 12 cases, they could demonstrate convincingly posterior extension of maxillary sinus pathology in 10 patients with malignancy.

The diagnostic arteriographic findings were topographic changes such as pushing away of the internal maxillary artery and its branches at the pterygo-maxillary fossa, with—at times—stenosis and

rigidity, and even pressure defect. There was also persistence of the radiopaque material within the tumoral mass demonstrating the irregular contour of the tumor.—*Jirair N. Sarian, M.D.*

Neck and Chest

Salvia, Carlos, Dreyer, Mario P., Laplume, Sergio O., and Caubet, Juan Carlos. Laringocele ventricular: comunicación de un caso, estudio clínico y radiológico. (Ventricular laryngocele: report of a case, clinical and radiologic study.) *Prensa méd. argent.*, 1972, *59*, 108–111. (From: Facultad de Medicina, VII[a] Cátedra de Medicina, Universidad Nacional de Buenos Aires, Argentina.)

A ventricular laryngocele is a dilated Morgagni's ventricle (a horizontally situated elliptic space, lateral to the glottis, between the adjacent aspects of the false and true vocal cords). In humans this space is very small, representing a vestigial residue of air sacs present in anthropoids. The mucosal lining of this space contains mucous glands.

These laryngoceles can be anatomically classified as internal (medial to the thyrohyoid membrane), external (project through the thyrohyoid membrane into the superficial layers of the lateral neck) or mixed (combined features).

Situations that increase the intralaryngeal pressure (chronic cough, wind instruments playing etc.) would predispose to their appearance.

They can be asymptomatic, or produce dysphonia, dyspnea, foreign body sensation, dysphagia, or palpable cervical tumor in the submandibular area.

The diagnosis can be made by laryngoscopy, or laryngography (contrast and laminagraphic studies).

One case of an external laryngocele is reported in detail.—*Julian B. Alvarez, M.D.*

Olefsky, Jerrold, Kempson, Richard, Jones, Henry, and Reaven, Gerald. "Tertiary" hyperparathyroidism and apparent "cure" of vitamin-D-resistant rickets after removal of an ossifying mesenchymal tumor of the pharynx. *New England J. Med.*, April, 1972, *286*, 740–745. (From: Departments of Medicine, Pathology and Radiology, Stanford University School of Medicine and Veterans Administration Hospital, Palo Alto, Calif.)

A 40 year old man with bone pain, hypophosphatemia, normocalcemia, elevated serum alkaline phosphatase and increased renal phosphate clearance with roentgenologic evidence of osteomalacia was diagnosed to have vitamin-D-resistant rickets which responded to vitamin D and oral phosphate until a pharyngeal tumor was removed. Serum phosphate then became elevated and hypercalcemia occurred, at which point therapy was stopped. Normal serum phosphate and alkaline phosphatase were noted with elevated levels of serum calcium and parathyroid hormone. It is postulated that the apparent cure of vitamin-D-resistant rickets was on the basis of tumor secretion of a vitamin D antagonist.

The authors indicate a histologic similarity of cell type of the pharyngeal tumor in the case reported and 3 similar cases in the literature and suggest that the cases be referred to as ossifying mesenchymal tumors associated with vitamin-D-resistant rickets.

The development of tertiary hyperparathyroidism following the pharyngeal tumor removal is a particularly important aspect of the report in reference to the postulated role of parathyroid glands in the pathogenesis of vitamin-D-resistant rickets. Thus increased levels of parathyroid hormone cannot be implicated in all cases of vitamin-D-resistant rickets and, in fact, cure of the clinical syndrome can occur in the presence of elevated levels of circulating parathyroid hormone.—*Lionel W. Young, M.D.*

Abdomen

Lust, Franz J. An early case of Linitis Plastica: roentgenographic studies of the mucosa. *Proc. Rudolf Virchow med. Soc.*, 1968, *27*, 236–243. (Address: 133 East 73rd Street, New York, N. Y. 10021.)

Linitis Plastica was first described in 1954. It is an infiltrative lesion of the stomach, and the roentgenographic signs are due to fibrous infiltration with development of excessive connective tissue in the gastric wall. Some of the causes are gastric syphilis, tuberculosis and carcinoma. It has also been described as due to an innate fibrous tissue diathesis and this possibility is suggested by the occasional occurrence of diffuse fibrosis in other portions of the intestinal tract and in the gallbladder. The fibrosis may be due to an irritant factor of low virulence acting over a long period of time.

By far the commonest cause is carcinoma, and apparently its growth is so slow that connective tissue proliferation keeps pace with the progress of the neoplasm.

Early diagnosis can be established by the study of gastric mucosa with contrast materials. The abnormality is usually not demonstrable by fluoroscopy, and early diagnosis is difficult.

The author presents a case of Linitis Plastica due to carcinoma in a 44 year old woman in whom 2 sets of roentgenograms, made 6 months apart, at separate institutions, were available to show the progress of the lesion.—*E. Frederick Lang, M.D.*

Kenefick, John S. Carcinoma of the duodenum. *Brit. J. Surg.*, Jan., 1972, *59*, 50–55. (From: Royal Free Hospital, London, England.)

Five patients with carcinoma of the second part of

the duodenum presenting with obstructive jaundice are reported.

Primary carcinoma of the duodenum is rare, accounting for 0.03–0.05 per cent of all carcinomas. Three per cent of gastrointestinal carcinomas occur in the small bowel, and of these, 45 per cent arise in the duodenum. Most malignancies of the duodenum are adenocarcinomas (87 per cent). The remainder are leiomyosarcomas, lymphosarcomas and reticulum cell sarcomas. Thirty-five per cent are situated in the first part of the duodenum, 45 per cent in the second, and 10 per cent in the third and fourth parts. Duodenal carcinoma can be ulcerative, polypoid, annular and/or infiltrative.

The average duration of symptoms before diagnosis is 10 months. Patients may present with 1 or a combination of 4 major abnormalities: duodenal obstruction; biliary obstruction; hemorrhage; and/or penetration.

In a review of 31 cases of duodenal carcinoma, Burgerman found the incidence of symptoms to be as follows: abdominal pain, 74 per cent; weight loss, 74 per cent; vomiting, 55 per cent; hematemesis, 23 per cent; melena, 29 per cent; jaundice, 23 per cent; abdominal mass, 19 per cent; and anemia, 55 per cent.

The diagnosis is established by barium studies of the upper gastrointestinal tract. Important signs are alteration of the mucosa, intraluminal filling defects, altered contour and peristalsis of the duodenum, and the effects of duodenal obstruction. Hypertonic duodenography may be of value in detecting earlier lesions. Preoperative cholangiography is advisable in all patients presenting with obstructive jaundice.

Approximately half the patients come to surgery, and of these, 54 per cent have palliative surgery and 38 per cent have radical surgery. The choice of treatment depends on the extent of the lesion and the general condition and age of the patient. Pancreaticoduodenectomy should be performed when feasible, as this gives the best results. The operative morbidity is 18.5 per cent and the 5 year survival is 40 per cent. In the present series radical surgery was not possible in any case and local excision of the tumor and adjacent duodenal wall was performed. This procedure resulted in the relief of symptoms in each case, but was not curative. Three patients survived for 8, 9, and 12 months, respectively, but then died with metastases. Two patients are still alive 16 and 18 months, respectively, after operation, but 1 of these had hepatic metastases at the time of operation.

Carcinoma of the duodenum should be considered in the differential diagnosis of all cases of obstructive jaundice. Careful roentgenologic investigation is the most important diagnostic approach and radical surgery holds the best prognosis.—*Frederick Stitik, M.D.*

Wang, T. K., Tolnai, G., Campbell, J. S., Sirois, J., and Liepa, E. Crohn's disease of the appendix. *J. Canad. M.A.*, Feb., 1972, *106*, 233–236. (Address: Dr. G. Tolnai, Department of Pathology, Faculty of Medicine, University of Ottawa, Ottawa, Ontario, Canada.)

Granulomatous inflammation typifying Crohn's disease was localized within, or confined to, appendices in 6 patients, 2 of whom developed lesions attributable to Crohn's disease elsewhere in the gut.

The remaining 4 patients have remained symptom free for periods varying from 2 to 6 years.

Histologic evidence of Crohn's disease was also present in 5 of 46 appendices available for reexamination in a survey of 63 cases of Crohn's enterocolitis.

It is suggested that appendiceal involvement in Crohn's disease is not uncommon.—*Eugene C. Klatte, M.D.*

Carpenter, W. S., Allaben, R. D., and Kambouris, A. A. Fistulas complicating diverticulitis of the colon. *Surg., Gynec. & Obst.*, April, 1972, *134*, 625–628. (From: Departments of Surgery, Harper Hospital and Mount Carmel Mercy Hospital, Detroit, Mich.)

The authors reviewed the records of 92 patients treated surgically for diverticular disease of the colon. There were 16, or 17.4 per cent, in whom some type of fistula occurred. Eight fistulas developed in males and 8 in females. Sigmoidovesical fistulas were the most common being present in 16 patients (56 per cent). Other fistulas noted were sigmoidovaginal (2 patients), sigmoidovesicovaginal (1 patient), sigmoidoileal (1 patient), and sigmoidocutaneous (3 patients).

The authors discuss the clinical signs and symptoms of the various fistulas.

Adequate resection of the diseased colon with anastomosis of the uninvolved intestine is essential to effect a cure in a fistula secondary to diverticulitis. Colostomy alone is inadequate. The lower margin of the resection should be at the pelvic floor. A one stage colonic resection is the procedure of choice. Marked inflammatory reaction or abscess formation, rather than presence of a fistula, contraindicates a one stage procedure.—*Eugene C. Klatte, M.D.*

Marenco, Enrique R., Rozier, Enrique G., and de Crescenzo, Salvador. Actinomicosis abdominal. (Abdominal actinomycosis.) *Prensa méd. argent.*, 1972, *59*, 27–31. (From: Policlinica Bancaria, Unidad Proctología, Buenos Aires, Argentina.)

Human actinomycosis is produced by the *Actinomyces bovis* (*israeli*). In 60 per cent of the cases the process involves the cervicofacial area, in 20 per cent the thoracic area is involved, and in 20 per cent the disease is localized in the abdominal-pelvic area, particularly in the cecal-appendicular region.

The local symptomatology varies according to the area of involvement.

The general symptoms (low grade fever, asthenia, anorexia etc.) are nonspecific. The abdominal involvement produces mucus-bloody diarrhea, or constipation due to stenosis, crampy pain, localized tenderness and palpable tumor.

The diagnosis is made by demonstrating the *Actinomyces*, either by gross inspection (yellowish granules), or by microscopic study, or by culture.

Medical treatment (penicillin, sulfas) will cure most of the cases; surgery is indicated for residual changes.

Two case histories are described in detail. One case presented as a partially obstructing tumor of the transverse colon, in which the diagnosis was made after pathologic study of the resected specimen; intensive penicillin therapy was instituted postoperatively. The other case presented as a rectal granuloma complicating colonic diverticulitis and peritoneal carcinomatosis.—*Julian B. Alvarez, M.D.*

MISCELLANEOUS

DIAZ, GLORIA E., and SALVIDEA, JULIO C. Supresión suprarenal por trombosis de la vena central mediante cateterismo selectivo: comunicación previa. (Suprarenal suppression by thrombosis of the central vein by means of selective catheterization: preliminary communication.) *Prensa méd. Argent.*, Nov., 1971, *58*, 1899–1903. (From: Clinica Modelo de Lanús, Buenos Aires, Argentina.)

In a patient with carcinoma of the breast with advanced metastases, bilateral adrenalectomy was considered. Instead of surgery the central vein of the adrenal gland was catheterized by means of a catheter introduced percutaneously through the femoral vein (Seldinger's technique). An injection of 5 ml. of 2 per cent Xylocaine was made through the catheter and a small amount of contrast material was given to establish the correct position of the catheter. Then 2 ml. of blood was withdrawn, allowed to clot, and reinjected through the catheter. Finally a small amount of sclerosing fluid was injected.

One week later it was possible to observe clinical signs of adrenal insufficiency (systemic hypotension, weakness, easy fatigability) associated with corresponding humoral changes (fall in plasma cortisol levels and urinary excretion of steroids).—*Charles M. Nice, Jr., M.D., Ph.D.*

MONNEROT-DUMAINE. Un animal de laboratoire peu sympathique mais instructif: le scorpion. (The scorpion—a laboratory animal, little liked, but useful.) *La nouvelle Presse méd.*, Feb., 1972, *26*, 571.

The biology of the scorpion is particularly striking. Man, one of the most fragile of the living beings, is greatly interested in studying the means of resistance of the scorpion to the environment, and the factors which have contributed to its survival through the ages.

The scorpion manifests great resistance to thermal changes, becomes rigid and apparently dead when placed in a Frigidaire, but will rapidly regain its activity when subjected to a draft of warm air and returns to normal when immersed in normal physiologic solution. It survives prolonged fasting, with a record of 36 months by a female of the species. Now a terrestrial animal, the scorpion will survive 48 hours of submersion in water, and will also survive when 7 of its 8 respiratory orifices are occluded. It is an animal difficult to anesthetize or kill.

The resistance of the scorpion to radiation is the highest among all living beings, as observed following the atomic explosions in the Pacific. The LD_{50} is 75,000 r as compared with 600–700 r in man, 1,800 r in fish, 3,000 r in the aquatic salamander, 12,000 r in the mollusk and 64,000 r in the Drosophila.

This extraordinary resistance may be explained, in part, on the basis of rare cellular division, low metabolic activity and low total nucleic acid level. Other contributing factors may be the high copper content of the hematocyanine, the role of taurine, and the luminescence of the cuticle indicating a high resistance to radiation.

The scorpion has survived eons of time and countless environmental changes. An aquatic animal over 400 million years ago, it exists at present as a terrestrial animal. It is a living fossil.—*William H. Shehadi, M.D.*

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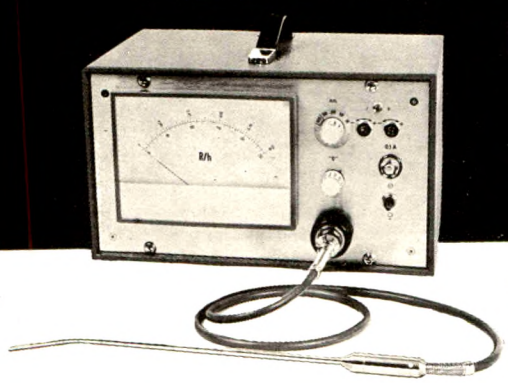
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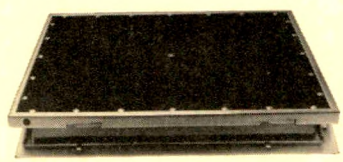
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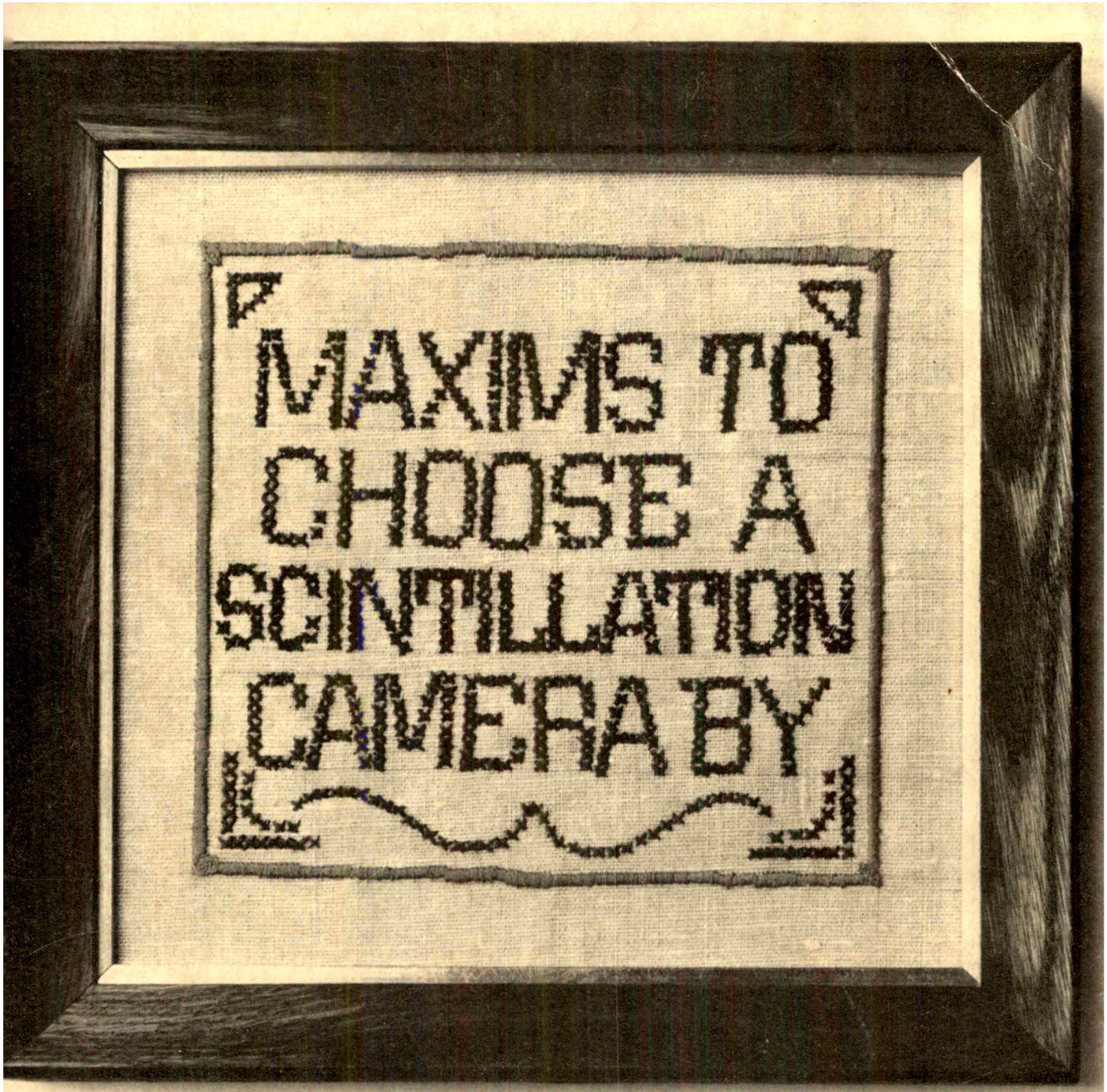
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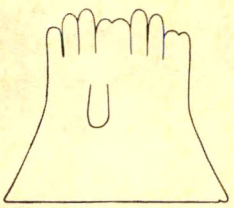
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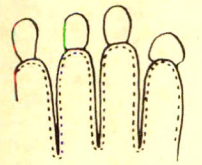


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tinuity and metastasize by lymphatics and blood. Slow growing tumors, which remain localized for a long period, have a better prognosis than rapidly diffuse neoplasms and treatment is more successful in younger patients.

Available therapeutic possibilities include surgery with local wide excision, amputation or possibly lymph node dissection, or radiotherapy, and/or a combination of both.

The author reviews the literature and finds that the best survival percentages appear to be obtained with a combination of surgery and radiotherapy, using either pre- or postoperative irradiation and regional lymph node irradiation. Doses in the 4,500 to 6,000 rad range in 28 to 35 days covered most of the treatments described in the literature. Some mention was also made of chemotherapeutic perfusion procedures which are helpful in some patients.

Twenty-seven of 28 cases were included in the author's study, which extended from January 1952 to June 1968. Of the 28 patients, 27 remained available for follow-up. The median survival was 17 months in men and 30 months in women. Treatment was divided into 3 groups: irradiation plus surgery; surgery alone; and irradiation alone.

The series was so small that it was difficult to draw any unequivocal conclusions. The longest survivals occurred among the patients treated with surgery alone. The younger patients tended to have a longer interval before recurrence. Above the age of 25 years recurrence appeared earlier.

Because of the inability to draw unequivocal conclusions from the small number of patients, the author advocates centralization of treatment of these rare tumors as the only means of achieving optimal therapy.

Optimal therapeutic regime according to the author seems to be: preoperative radiation of 800 rads; excision biopsy; completion of irradiation to the 5,000–6,000 rad level; and radical excision 3 to 6 weeks after the end of irradiation, including amputation where necessary.—*Robert P. Andrews, M.D.*

Fried, K., and Krause, J. Die metaphysäre Dysplasie—Morbus Pyle. (Metaphyseal dysplasia—Pyle's disease.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Feb., 1972, *116*, 224–228. (Address: Dr. K. Fried, csl. armády 1179, Kladno, Č.S.S.R.)

Metaphyseal dysplasia is a rare congenital skeletal abnormality; only a few cases have been reported since Pyle in 1931 described this dysplasia that bears his name.

Six additional cases of this condition are reported by the authors. The typical roentgenologic changes of metaphyseal dysplasia are described, and their differentiation from craniometaphyseal dysplasia is discussed.

The distal ends of the femur, humerus, and radius, as well as both ends of the tibia, exhibit a flask-like flare with a thin cortex and diminished over-all density. The spongiosa in the region of the widening is either fine or coarse. In 3 patients the affected metaphyses are surrounded by shallow exostoses. In all cases there is ulnar angulation of the widened radius; the ulna is relatively short with a lack of modeling only in its distal 3 cm. Five cases show broad, ill-defined, longitudinal bands and 1 case thin transverse lines in the affected bone segments. As the widening may extend for one-half the diaphysis, the authors suggest to call this condition metaphyseodiaphyseal dysplasia.

Craniometaphyseal dysplasia is a distinctly different condition and can easily be distinguished roentgenographically: The flaring of the metaphysis is far milder than that seen in Pyle's disease; there is frontal, paranasal and occipital hyperostosis with absent or limited pneumatization that is missing in Pyle's disease; marked thickening of the ribs, clavicles, pubic and ischial bones occur to a far lesser degree in Pyle's disease. Furthermore, Pyle's disease is asymptomatic, whereas affection of the 2nd, 3rd, 7th, and 8th cranial nerves, respiratory symptoms due to narrowed nasal lumen and occasional increased intracranial pressure are seen in craniometaphyseal dysplasia.—*Peter F. Winter, M.D.*

Spiegel, Phillip G., Kengla, Kenneth W., Isaacson, Alvin S., and Wilson, John C., Jr. Intervertebral disc-space inflammation in children. *J. Bone & Joint Surg.*, March, 1972, *54-A*, 284–296. (From: Department of Orthopedics, Children's Hospital of Los Angeles, Los Angeles, Calif.)

The authors report 45 cases of disk-space inflammation in children, the average age of onset being 6½ years, with an average duration of symptoms of 4½ weeks.

The most prominent symptoms in this condition are back and hip pain with refusal to walk. Ninety-one per cent of the cases show an elevated erythrocyte sedimentation rate. The first roentgenographic sign is disk-space narrowing, usually in the lumbar area. The interspace between the fourth and fifth lumbar vertebrae is most frequently narrowed (40 per cent in this series).

Special investigations included 15 biopsies with 4 patients yielding positive bacteriologic cultures. The biopsy specimens showed changes consistent with acute and chronic inflammation.

Additional roentgenographic changes include narrowing accompanied by irregularity or haziness of the vertebral end plates. Cavitation may occur in the vertebral end plate, can be persistent, and is best detected with laminagrams. In the process of healing the end plate may become sclerotic with partial restoration of the space. Interbody fusion may occur as a late finding with scoliosis and

kyphosis, wedging over this space and segmental or total vertebral body enlargement (vertebra magna).

The etiology of disk-space inflammation in children is unknown. The major etiology is presumed to be infectious. Additionally, trauma has been suggested as a cause as well as avascular necrosis.

The authors point out the difficulty in diagnosing this condition, which must be distinguished from acute pyogenic vertebral osteomyelitis, tuberculous osteomyelitis, *Brucella* and *Salmonella* osteomyelitis, Scheuermann's disease, and disk inter-space narrowing because of trauma. In long-term follow-up the patients are relatively asymptomatic with 42 per cent having residual mild scoliosis with minimum rotation. Apparently about 25 per cent had partial or almost complete interbody ankylosis.—*Lionel W. Young, M.D.*

Houston, C. Stuart, Awen, C. F., and Kent, H. P. Fatal neonatal dwarfism. *J. Canad. A. Radiologists*, March, 1972, *23*, 45–61. (From: University Hospital, Saskatoon, Saskatchewan, Canada.)

Fatal neonatal short-limbed dwarfism includes 6 apparently distinct entities. The mechanism of death is related to the small thoracic cage in all. Differentiation of the types of dwarfism is important from the standpoint of genetic counseling, and the radiologist plays a major role in the diagnosis.

Four types fall into the broad classification of chondrodystrophy, which may be divided according to the appearance of the vertebral bodies. Achondrogenesis, in which vertebral bodies are not ossified, and asphyxiating thoracic chondrodystrophy, with normal appearing vertebrae, are always autosomal recessive. Thanatophoric dwarfism, with flat vertebral bodies, has now been reported in 2 families with 2 instances each and, thus, requires further genetic studies to determine whether these familial occurrences are exceptions or whether it is also an autosomal recessive condition. Hyperchondrogenesis (metatropic dwarfism), which also has flat vertebral bodies with dumbbell-shaped bones, is nonfamilial.

Two severe forms of well known diseases may occasionally cause fatal dwarfism in the newborn: hypophosphatasia, which is always autosomal recessive; and osteogenesis imperfecta lethalis, which sometimes is.

Mention is made of true achondroplasia, which is autosomal dominant, and diastrophic and camptomelic dwarfism, but the authors had not encountered examples of these.

All of the other types are illustrated by case reports, and the clinical, pathologic and roentgenologic characteristics correlated and tabulated.—*Lois Cowan Collins, M.D.*

Farfan, H. F., Huberdeau, R. M., and Dubow, H. I. Lumbar intervertebral disc degeneration: the influence of geometrical features on the pattern of disc degeneration—a post mortem study. *J. Bone & Joint Surg.*, Apr., 1972, *54-A*, 492–510. (Address: H. F. Farfan, M.D., 5300 Cote des Neiges, Montreal 26, Quebec, Canada.)

One hundred and eighty-two specimens of whole lumbar spines (12th thoracic to 2nd sacral vertebrae) were obtained at autopsy; anteroposterior and lateral roentgenograms and diskograms were made at all levels. The inclination of the lumbar disks with reference to the long axis of the spine was determined as well as the degree of the lumbar curve.

For the purposes of this study an annular rupture was assumed to be present, if there was any abnormal penetration of contrast material into or through the annulus. No attempt was made to classify annular damage from the plain roentgenographic appearance. When the annulus appeared normal, but the disk accepted more than 1 ml. of contrast medium, the disk was assumed to have a Schmorl's node even if this did not show on the roentgenogram. Thus the incidence of annular rupture and of Schmorl's nodes at the different levels was correlated with the amount of lumbar curve. The cut surfaces of the disk were also examined for evidence of radial fissures in the annulus, and the presence and location of complete radial fissures were determined.

The incidence of annular damage (abnormal penetration of contrast medium into or through the annulus) increased with age and was the highest at the lumbosacral level, second highest between the fourth and fifth lumbar vertebrae and least common between the first and second lumbar vertebrae. This distribution also varied with the degree of lumbar curve. The joints between the fourth and fifth lumbar vertebrae in spines with an increased lordotic curve, showed an incidence of annular damage nearly twice that of the lumbosacral joint. The distribution of degenerative changes in the lumbar intervertebral joints varies with the lumbar lordosis, and the amount of lordosis is not related to sex and age. An increased inclination of the lumbosacral joints seems to protect it against annular damage.

Five basic patterns of radial tears or fissures of the annulus can be identified: posterolateral; posterior bilateral; posterocentral; lateral; and anterior. The pattern of the tear appears to be related to at least 2 geometric features of the joint: (1) shape of the disk, (in disks with a flattened posterior annular surface, posterolateral fissures develop, while in disks with rounded posterior surfaces, posterior mid-line ruptures are found); and (2) symmetry of the articular processes (asymmetric articular processes lead to asymmetric degeneration, while symmetric processes lead to symmetric degeneration).

The highest incidence of Schmorl's nodes was be-

tween the third and fourth lumbar vertebrae, and relatively more of these defects were found in "flat" spines than in "lordotic" spines. The rarity of Schmorl's nodes in the sacrum is noteworthy.— *E. Nicholas Sargent, M.D.*

Vermess, Michael, Pearson, Kent, Einstein, Albert B., and Fahey, John L. Osseous manifestations of Waldenström's macroglobulinemia. *Radiology*, March, 1972, *102*, 497–504. (Address: Michael Vermess, M.D., Department of Diagnostic Radiology, The Clinical Center, National Institutes of Health, Bethesda, Md. 20014.)

Macroglobulinemia, as initially described by Waldenström in 1944, is a chronic, neoplastic disease similar to multiple myeloma. The previous distinguishing feature between the 2—lack of bone involvement in Waldenström's macroglobulinemia—is refuted in this article.

The authors present 41 cases of the disease.

The 4 classes of bone lesions are: (a) generalized demineralization; (b) single osteolytic lesions; (c) multiple osteolytic lesions; and (d) cyst-like lesions.

The authors consider the cyst-like symmetric lesions of the acetabula as potentially aiding in the differential diagnosis between myeloma and macroglobulinemia. Three of the patients in the series demonstrated the bilateral symmetric bubbly iliac lesions. Unfortunately, the nature of these lesions was not proved by biopsy. Therefore, specificity for macroglobulinemia cannot be related. A total of 43.9 per cent of the patients (18 of 41) showed bone pathology.

The authors list a rather extensive bibliography for those interested in the historic aspects of this entity.

The differential diagnosis of the various lymphoproliferative diseases must be based upon serum protein studies.—*J. P. Eberts, M.D.*

Weston, W. J. Joint space widening with intracapsular fractures in joints of the fingers and toes of children. *Australasian Radiol.*, Nov., 1971, *15*, 367–371. (From: Hutt Hospital, Lower Hutt, New Zealand.)

The author reports 5 cases, showing widening of the joint space in intracapsular fractures of the digits presumably secondary to hemarthrosis.

The synovial cavity and joint capsule must be intact and the fracture must reach the periosteum.

After 2 to 4 weeks the joint width returns towards normal.—*Martha Mottram, M.D.*

Blood and Lymph System

Denck, H., and Olbert, F. Die Angiographie der weiblichen Brust. (Angiography of the female breast.) *Radiol. clin. et biol.*, 1972, *41*, 90–98. (Address: Prim. Doz. Dr. H. Denck, I. Chirurgische Abteilung, Krankenhaus der Stadt Wien-Lainz, Wien, Austria.)

Angiographic examinations of the breast were performed in 25 patients with breast tumors by selective catheterization of the lateral thoracic and/or internal mammary arteries via brachial artery puncture.

There was good correlation between the angiographic interpretation and the histologic diagnosis; the angiographic diagnosis was correct in 23 patients, and, in addition, in some cases nonpalpable axillary lymph node metatases could be demonstrated by a distinct tumor blush.

The method is felt to be too laborious to become part of a routine work-up, but it is considered to be a useful completion of other diagnostic procedures such as mammography and thermography.

This is particularly true in cases with questionable, ill-defined masses, with diffused suspicious swelling of one breast, or with fibrocystic disease, in which a sudden induration is noted. It is also considered to be helpful in detecting local recurrences.

Despite the highly significant results, the authors do not think that the angiographic examination can replace the exact histologic diagnosis.—*Peter F. Winter, M.D.*

Gros, Ch., Warter, P., Haehnel, P., and Tongio, J. L'artériographie mammaire. (Mammary arteriography.) *J. de radiol., d'électrol. et de méd. nucléaire*, June-July, 1971, *52*, 353–356. (From: Service Central de Radiologie, C.H.U., 67-Strasbourg, France.)

The authors' conclusion, based on 25 cases of mammary angiography, is that this investigative method is not just supplemental, but is of definite value in cases where mammography, thermography, echography and needle aspiration cytologic studies give doubtful or contradictory diagnostic results. This also would be true for post-therapeutic cases.

The authors prefer the femoral catheterization (Seldinger technique), with injection of the subclavian arteries, for technical reasons. No incidents or accidents are reported.

The following observations were made regarding malignant tumors: increase in the size of the blood vessels irrigating the tumor, *i.e.*, increase in the caliber of the external mammary and the perforating branch of the internal mammary arteries; visualizations of pathologic tumoral vessels with stenotic parts resembling a cork-screw; opacification of the tumor resulting in dense tumorograms, in spite of the presence of arteriovenous shunts within the tumor which are the cause of more precocious and more evident venous phase as compared to normal breast angiography; and, finally, visualization of tortuous, irregular pathologic veins.

These angiographic findings are disseminated in a case of diffuse, edematous, carcinomatous mastitis.

In noncircumscribed dysplasias of the breast, the vascular image is normal, or even diminished.

In circumscribed dysplasias (cysts or fibroadenomata), the arterioles surround the tumor, giving a basket image, as the tumor is not visualized.

In the abscess, the arterioles and venules are much more dense, but not tortuous and irregular.

After radiotherapy, angiography permitted the authors to differentiate a residual tumoral nodule from a fibrous nodule devoid of cancer.—*Jirair N. Sarian, M.D.*

Edwards, J. M., Peachey, R. D. G., and Kinmonth, J. B. Lymphangiography and surgery in lymphangioma of the skin. *Brit. J. Surg.*, Jan., 1972, *59*, 36–41. (From: St. Thomas's Hospital Medical School, London; St. Helier Hospital, Carshalton; and St. John's Hospital for Diseases of the Skin, London, England.)

Eight cases of lymphangioma circumscriptum are reported: 6 were investigated by lymphangiography; 3 by lymphography.

Lymphangioma circumscriptum is a variant of lymphangioma in which local eruption of persistent thin walled translucent vesicles appears in groups of patches on the skin. In many cases several patches of involvement are present in one area. They may be found in any area of the skin, but more commonly arise over the proximal parts of the limbs and the adjacent limb girdles. Lesions of this type often appear at birth or soon after, but may be seen in childhood or even in later life. Patients present complaining of recurrent drainage or infection of the areas, as well as for cosmetic reasons. Physical examination demonstrates thickening and swelling beneath the skin indicating a subcutaneous component.

Lymphangiography of the affected limb using the standard technique was carried out in 6 patients. In all cases the subcutaneous lymphatic vessels and regional lymph nodes were normal. No direct communication between the subcutaneous lymphatics or regional lymph nodes to the lymphangioma was demonstrated. Three patients underwent lymphography. Lipiodol was injected directly into the skin vesicles. Multiple deep cystic spaces were outlined, demonstrating the subcutaneous component. Delayed roentgenograms taken up to 5 days after lymphography showed no movement of the contrast material towards the regional lymph nodes.

Surgery often must be extensive, due to the marked subcutaneous involvement. Short term follow-up has indicated good patient acceptance.—*Frederick Stitik, M.D.*

Neviaser, Robert J., Butterfield, William C., and Wieche, Donald R. The puffy hand of drug addiction: a study of the pathogenesis. *J. Bone & Joint Surg.*, Apr., 1972, *54-A*, 629–633. (Address: R. J. Neviaser, M.D., Room 932, George Washington University Clinic, 2150 Pennsylvania Avenue, N. W., Washington, D. C. 20037.)

The "puffy hand sign of narcotic addiction" is described in detail. The swelling characteristically extends from the proximal segments of the fingers to the wrists, obliterating the usual surface anatomy of the dorsum of the hand. This results from long-standing use of the dorsal veins of the hands for intravenous injection and, when all the veins become thrombosed, the needle is inserted into the dorsal aspect of the web spaces.

The puffy hand must be differentiated from the palmar fascial space infections with dorsal edema; the importance of this bilateral finding as an aid in the detection of the drug addict is noted. The clinical findings appear to be the end result of lymphatic obstruction. There may be superimposed local scarring secondary to inflammatory reactions at the injection site.

Venous obstruction does not appear to be a causative factor, but is rather a coexisting lesion. Based on biopsies of the skin and subcutaneous tissue, venograms, and lymphangiograms, the pathogenesis has been shown to be destruction of the lymphatics leading to fibrotic replacement of the subcutaneous tissue.

In most patients, lymphangiography is unsuccessful due to the fact that the lymphatics are obliterated and the patent blue dye does not stain the lymph vessels, precluding their identification prior to lymphography. In the 1 successful lymphangiogram described, the normal deep lymphatics did not fill; a few superficial channels along the radial aspect of the forearm and wrist were outlined, and abnormal collateral vessels were found around the elbow.—*E. Nicholas Sargent, M.D.*

RADIATION THERAPY

Hill, D. R., and Margolis, L. W. Radiation therapy management of retinoblastoma. *Radiol. clin. et biol.*, 1972, *41*, 139–144. (From: Section of Therapeutic Radiology, Department of Radiology, University of California, School of Medicine, San Francisco, Calif.)

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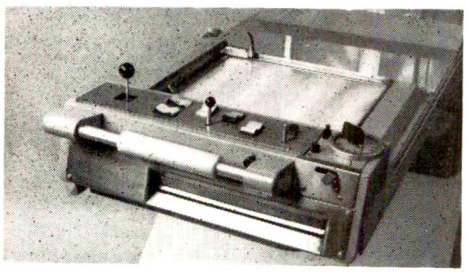
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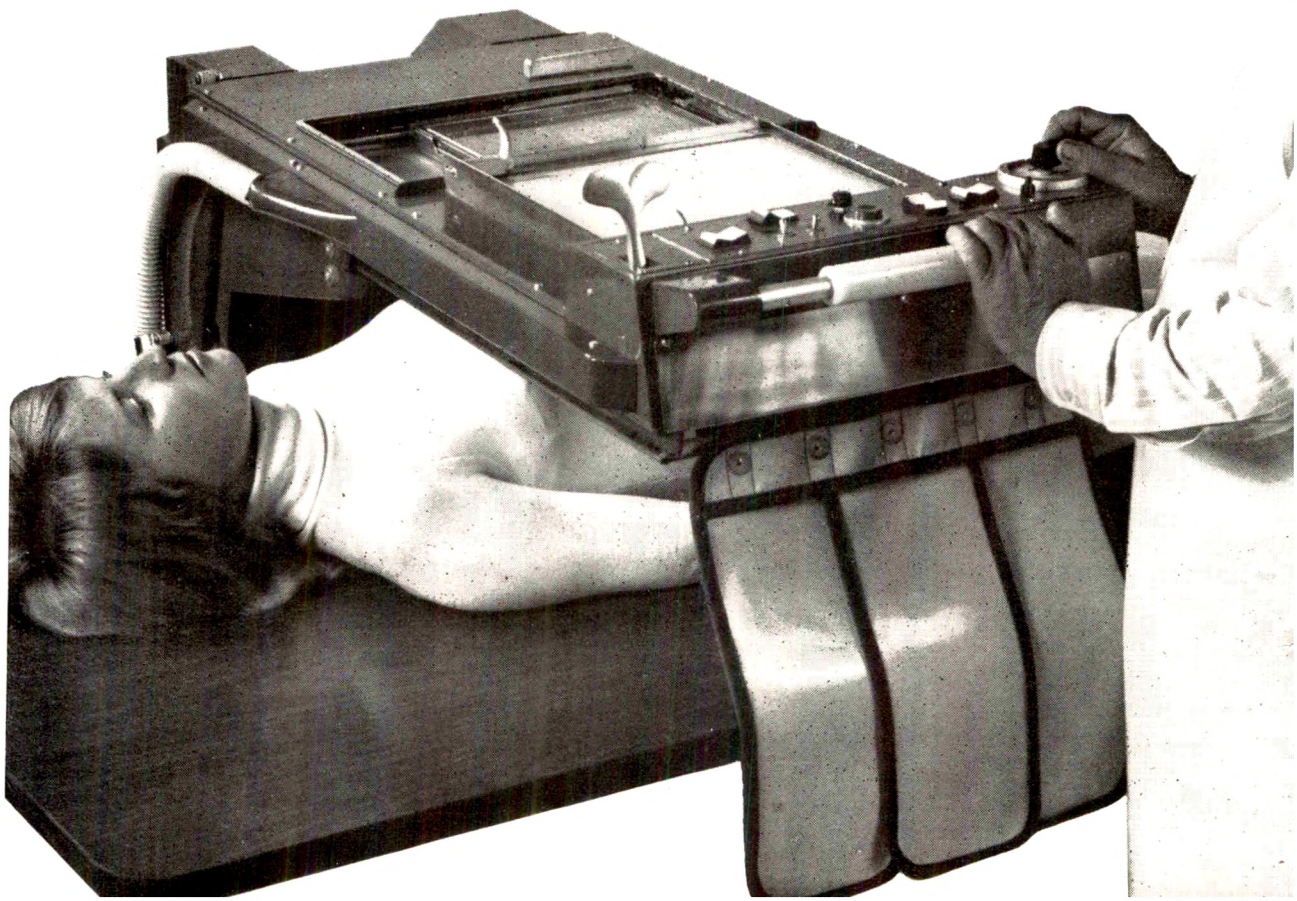
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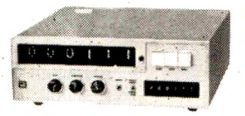
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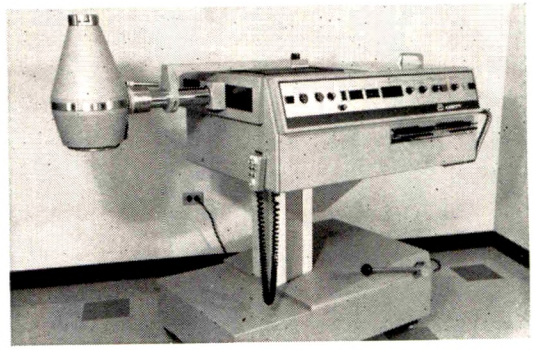
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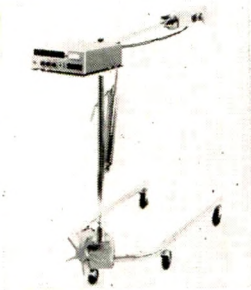
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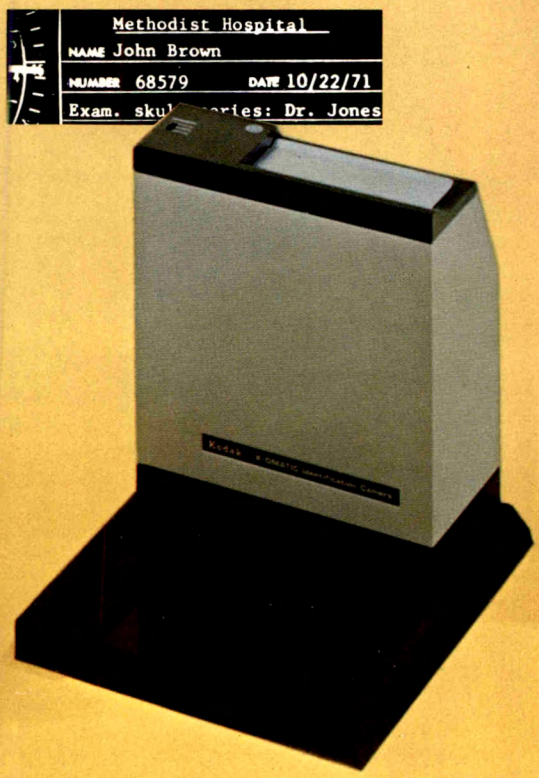
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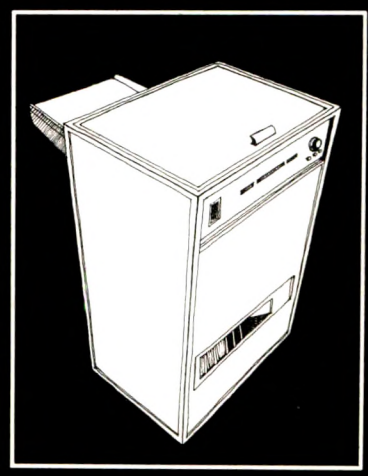




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In many cases, where x-ray units are operated centrally from a console, compromises in the positioning of both the console and the examination units is necessary.

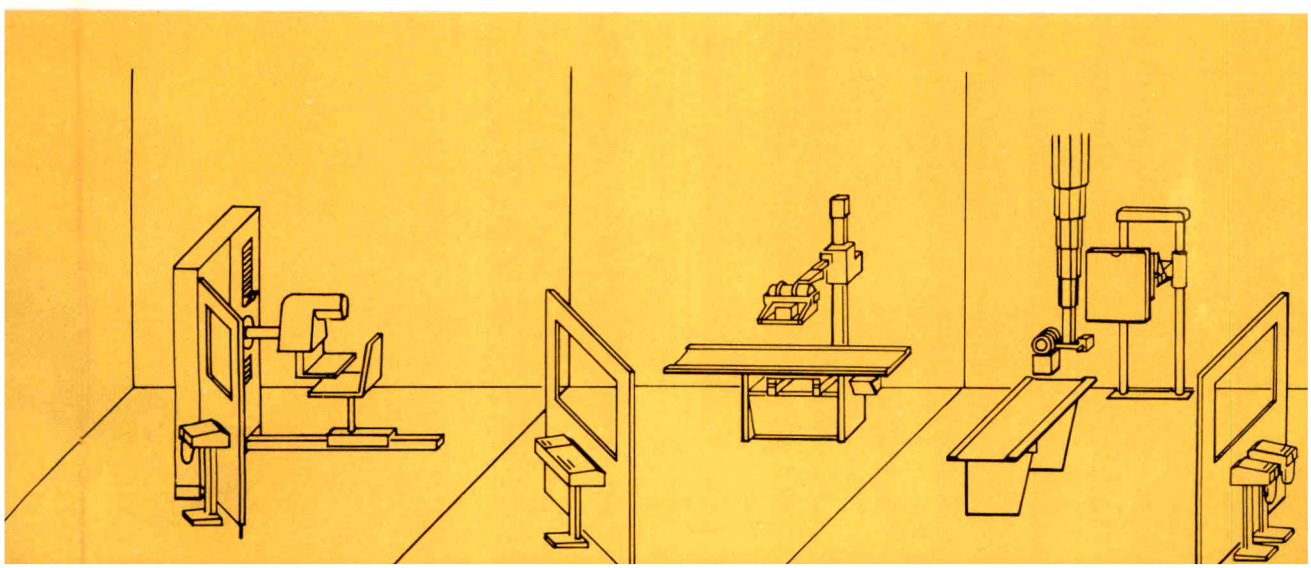
With the unit consoles of the Tridoros Optimatic system, the lay-out can be arranged in a more flexible manner.

In the setups shown below, technicians can preset pushbuttons either before or after positioning the patients. The actuated pushbuttons do not interfere with operation of other units. Only during the brief interval when an actual exposure is being made at one console is the use of the generator blocked for the other consoles. These exposure intervals are visually displayed by means of indicator lights on each console.

The new Siemens system permits the complete decentralization of any or all x-ray equipment. This is a significant advantage when an old installation is being modernized.

At left: Bucky wall stand and Bucky table with paired unit consoles.

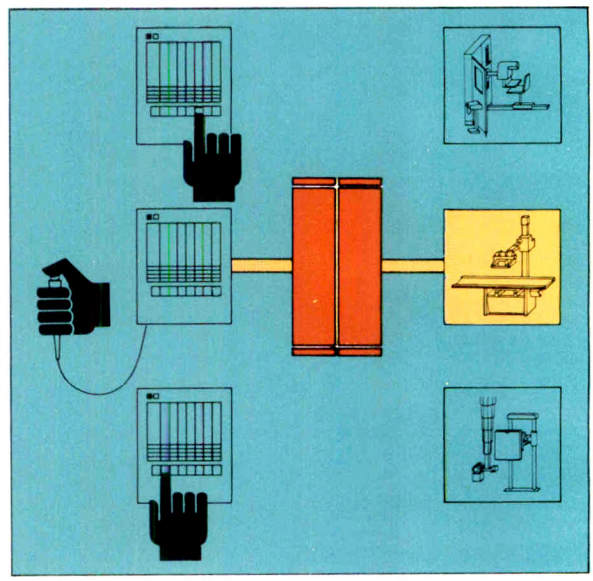
Below: Four consoles are set up near the units they control. With this kind of arrangement and the automated exposure of the Siemens system, technicians can concentrate on patient positioning.



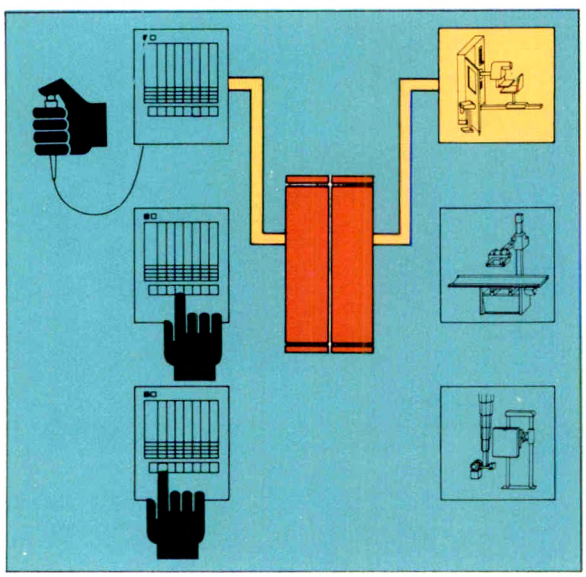
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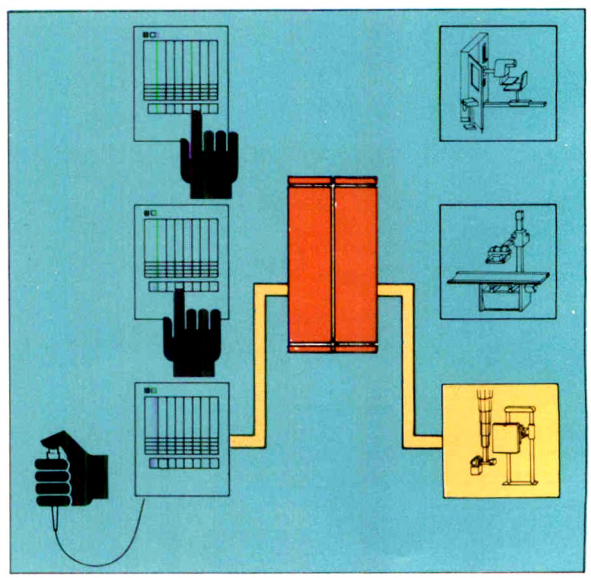
The preceding pages will give you an idea of how this new Siemens system can help you make the most efficient use of your radiological equipment and personnel. If you'd like to know more, please write Siemens Corporation, Medical Systems Division, 186 Wood Avenue South, Iselin, N.J. 08830.



(2) Programs for mammography and Bucky wall unit are selected while planigraphic exposure is made at unit console.



(1) Exposure is triggered for mammography while programs are selected at planigraphic table and Bucky wall stand.



(3) Third exposure is made at unit console controlling Bucky wall stand as programs are selected for the other two radiographic units.



WILLIAM B. SEAMAN, M.D.
HICKEY LECTURER, 1972

THE AMERICAN JOURNAL OF ROENTGENOLOGY
RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 116 OCTOBER, 1972 NO. 2

MOTOR DYSFUNCTION OF THE GASTRO-INTESTINAL TRACT*

HICKEY LECTURE, 1972

By WILLIAM B. SEAMAN, M.D.
NEW YORK, NEW YORK

IT IS a signal honor to have been invited to give this Lecture in memory of Dr. Preston M. Hickey, Founder and First Editor of the AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY & NUCLEAR MEDICINE, *Professor of Radiology at the University of Michigan, and one of the very great figures in American Radiology. I feel even more honored when I read the list of the distinguished Hickey Lecturers who have preceded me.*

Dr. Hickey was also trained in pathology and was especially known for his roentgen-pathologic correlations. Likewise, he was interested in the physiology of the gastrointestinal tract and one of his papers was titled, "Peristalsis of the Colon." In his Caldwell Lecture of 1928 Dr. Hickey commented, "Before I became interested in roentgen studies of deglutition my ideas upon the physiology of one of the most common phenomena of the human body were extremely hazy; since studying the act of swallowing with the roentgen ray, I have found in conversation with many physicians that the knowledge of factors in the swallowing of a morsel of food was very inaccurate." This statement has some truth today. He then went on to point out the advantages of studying swallowing by using serial roentgenograms.

In view of this, the subject I have chosen for tonight, "Motor Dysfunction of the Gastrointestinal Tract," seems relevant for a Lecture in honor of this great man.

The foundation of diagnostic radiology has been, and still is, the correlation of the roentgen image with the underlying gross, and to a lesser extent, microscopic pathology. Radiologic attention has been given to the motor activity of the gastrointestinal tract for many years, but roentgen diagnoses are still largely based on pathologic anatomy rather than abnormal physiology. In view of the progress that has been made in recent years, in part due to the advent of improved manometric techniques and cineroentgenography, a fundamental body of knowledge has been acquired that does enable some diagnoses to be made on the basis of abnormal motor function. Much remains

* Thirty-sixth Annual Hickey Memorial Lecture presented at the Joint Meeting of the Wayne County Medical Society, the Wayne State University College of Medicine, and the Michigan Radiological Society, Detroit, Michigan, March 2, 1972.

From the Department of Radiology, The Presbyterian Hospital in the city of New York at Columbia-Presbyterian Medical Center.

TABLE I

ROENTGENOGRAPHIC SIGNS OF MOTOR DYSFUNCTION OF THE GASTROINTESTINAL TRACT

Primary Signs
Peristalsis
Increased
Decreased
Absent
Spasm
Incoordination
Sphincter dysfunction
Secondary Signs
Diverticula
Zenker's
Epiphrenic
Jejunal
Colonic
Ileus
Incomplete emptying

to be done and more new problems have been born than old ones solved. The motor phenomena of the gastrointestinal tract are observed and recorded every day by thousands of radiologists who are in a unique position to be clinical physiologists.

It is significant that a physiologist, Walter B. Cannon, was the father of gastrointestinal radiology. Only a few months after Röntgen announced his great discovery, Cannon, then a student in Bowditch's physiology laboratory at Harvard University, began investigating the passage of bismuth down the esophagus of a goose. His first studies were published in 1897 by Bowditch. The motor physiology of the esophagus is still not completely understood.

In the gastrointestinal tract, attempts at pathologic correlation may fail even in the presence of organic disease because some of the factors responsible for the abnormal roentgen image are dynamic, and frequently not observable in the excised tissue or at autopsy. Theros, in his Hickey Lecture of 1969,[12] stressed the role of the radiologist as a gross pathologist. It has been said that the radiologist can not rightfully expect to do any better in the detection of disease, than the pathologist who can hold the organ in his hand. However, the radiologist has a great advantage over the pathologist: he is able to observe dynamic processes and make diagnoses based on abnormal function.

Motor dysfunction of the gastrointestinal tract can be rather simply classified on the basis of the dynamic roentgen changes that may be observed. Thus, muscular contractions may be increased, decreased, absent or constant (spasm) or lack the necessary coordination or synchronization to be functionally effective. Secondary effects, resulting from such abnormal function, include ileus, diverticula, dysfunction of the sphincters and incomplete emptying (Table I).

Spontaneous rhythmic contractility is an inherent property of the small intestine. It has been well established that this is due to periodic depolarization of the muscle cell membrane which is accompanied by changes in the electrical potential. This rhythmic slow wave electrical potential referred to as Basal Electrical Rhythm (B E R), originates in the duodenum; thus, the duodenum serves as a pacemaker from which the B E R is conducted down the intestine.[7]

Whether muscular activity occurs with each change in electrical potential is apparently determined by the physiologic stimulus of localized intraluminal pressure. The latter releases serotonin from the argentaffine cell which is considered an important hormonal activator of the afferent neurons of the myenteric plexus.

That there is another hormone involved in the mechanism of myenteric neuronal activation and consequent coupling of mechanical and electrical activity, has been demonstrated by Hiatt *et al.*[6] They have isolated a peptide from the bovine posterior pituitary that is distinct from oxytocin and vasopressin and have named it coherin. They have demonstrated that this substance has the capability of converting the incoherent, variable, rapidly changing potential of the B E R in the fast

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Siemens now offers a way to expand radiological facilities while at the same time optimizing the use of equipment and key personnel.

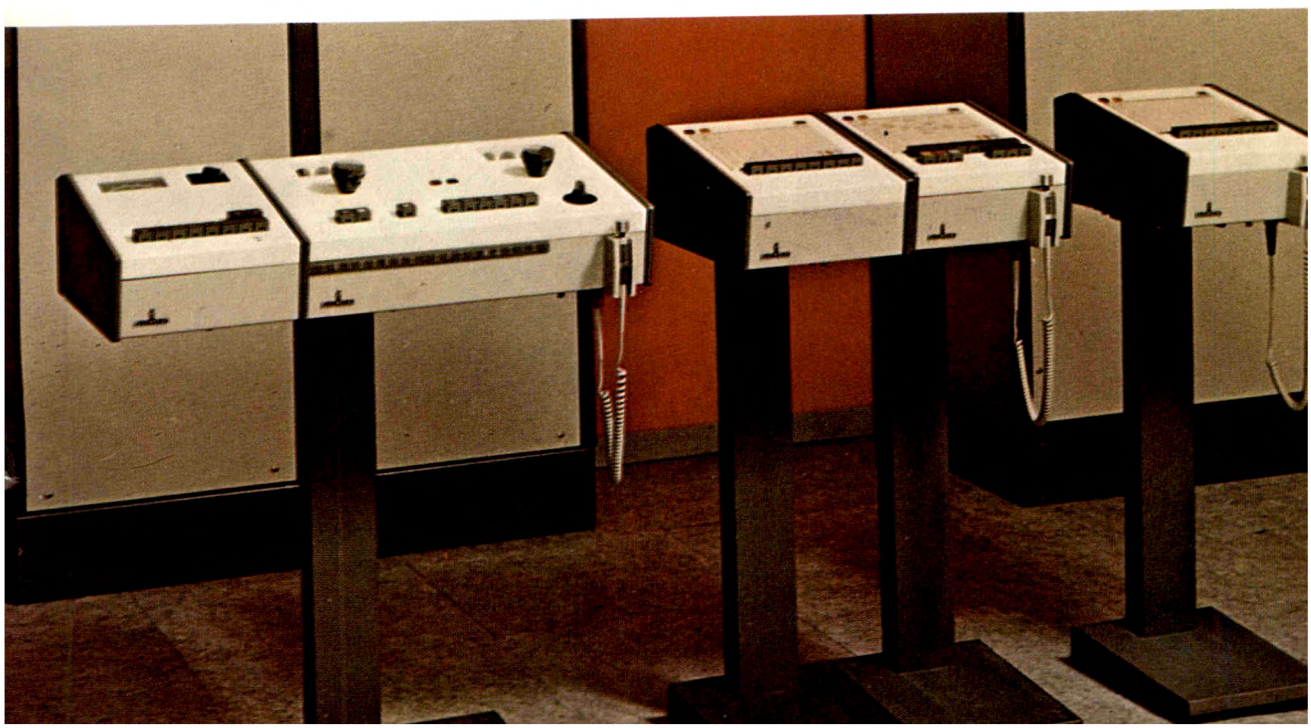
The Tridoros Optimatic system consists of a single centrally located, time-shared master generator with an analog computer and three or more pushbutton operated local consoles.

A new Tridoros Optimatic installation with four examination units usually costs considerably less than a similar setup using individual generators. But the major advantage of the new system is in the way it cuts to a minimum the number of decisions and adjustments the operator has to make to get optimum results.

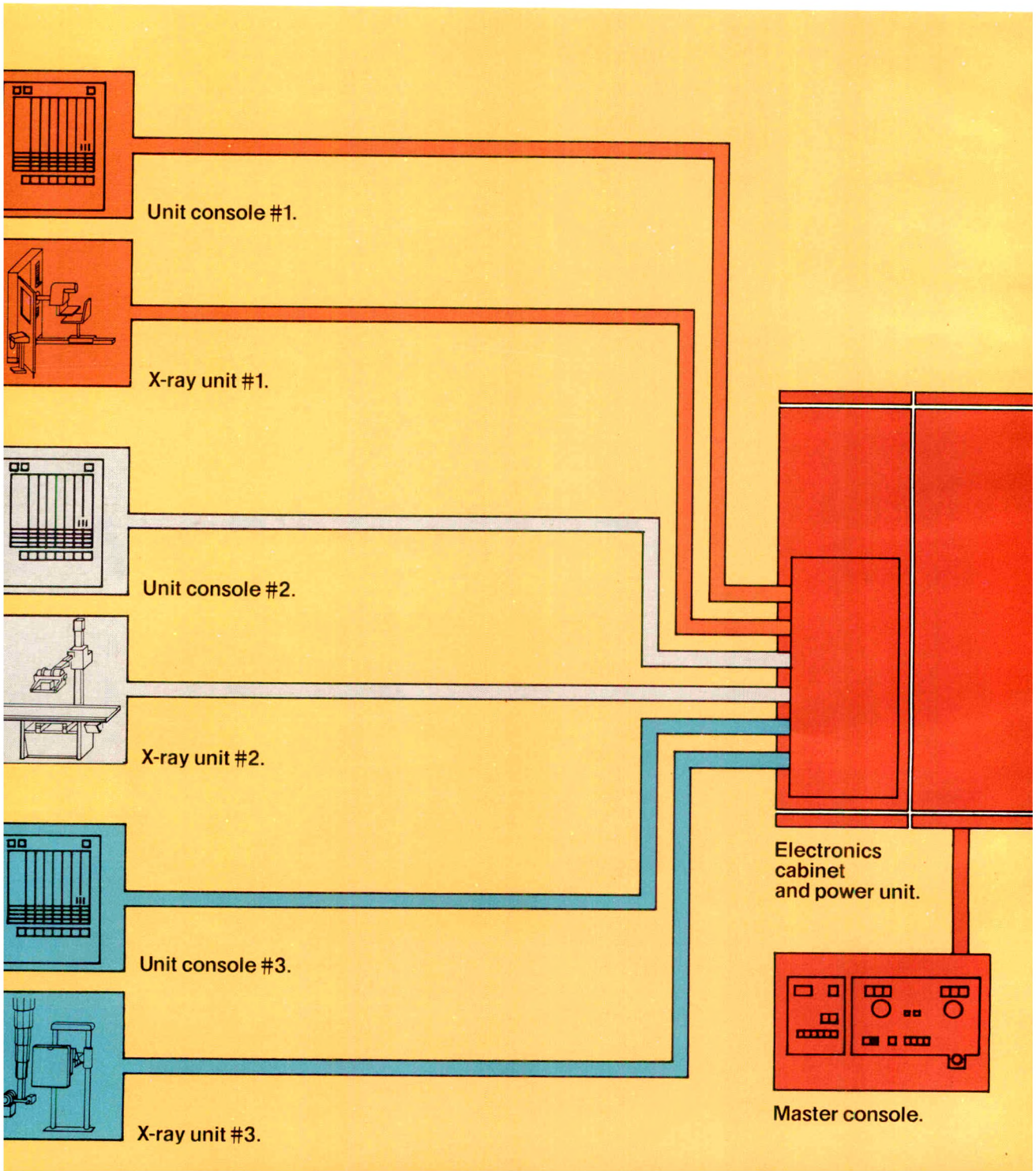
Technicians can now handle a much greater patient load while producing consistently superior radiographs.

At left: Technician starts automatically timed exposure at unit using overtable tube.

Below: Elements of the Tridoros Optimatic system including electronics cabinet (left rear), power unit (right rear) and left to right, master console and three unit consoles.



Consoles are "human-engineered" to provide organ-related pushbutton exposure control.



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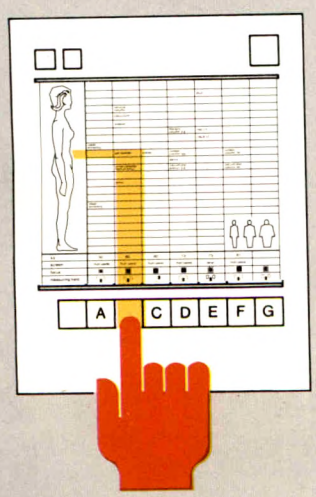
A Tridoros Optimatic unit console is set up in the immediate vicinity of each examination unit so that technicians are close to their patients.

To set the exposure at one of the consoles, the technician pushes one of the seven organ-related buttons. This automatically selects the kV, focal spot, screen combination and film blackening needed for the particular type of exposure to be made.

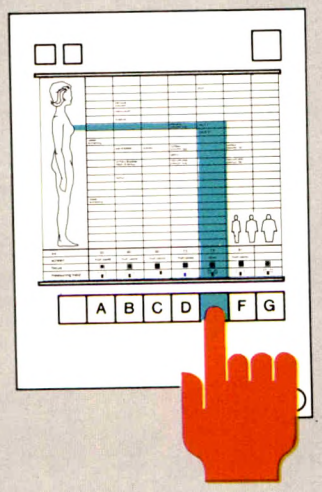
These unit console panels have a number of important advantages. They make data selection simpler than ever before. They greatly reduce the possibility of errors. And they assure consistently high quality radiographs.

At left: A Tridoros Optimatic setup consisting of three examination units with their individual consoles. Any unit can also be operated from the master console at lower right.

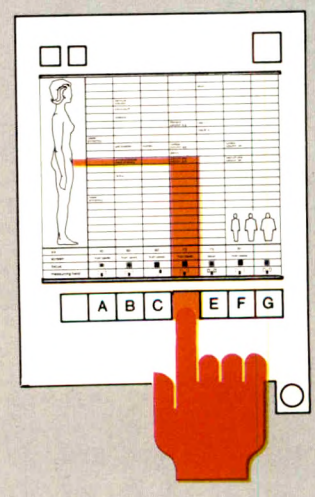
Below: Technician prepares for exposure by pushing desired organ-related button.



Pushbutton "B" sets typical Bucky table program for gall bladder, thin patient: 60kV, high-speed screen, small focal spot, Iontomat* center chamber.



Pushbutton "E" sets typical Bucky table program for ribs 1-7, medium patient: 73kV, detail screen, small focal spot, Iontomat* center chamber.



Pushbutton "D" sets typical Bucky table program for sacrum and coccyx a.p., heavy patient: 73kV, high-speed screen, large focal spot, Iontomat* center chamber.

Now x-ray department layouts can be planned for optimum use of available space.



ing dog to a highly organized cephalocaudal coherent pattern.

Coherin, in the blood stream of a dog, sensitizes the gut to intraluminally instilled serotonin (5-hydroxy tryptamine). Hiatt *et al.*[7] postulate that the site of action is the mucosal receptor, which is part of the afferent system of the myenteric plexus; this receptor requires the presence of both serotonin and coherin to be completely activated.

The potential clinical value of the above depends on the existence of disease states resulting from neuromuscular dysfunction of the small intestine. Hiatt *et al.*[7] claim that there are many such disease states including acute small intestinal obstruction, and postgastrectomy problems.

Following a partial gastrectomy, motor dysfunction of the efferent loop may be a cause of postoperative vomiting as pointed out by Golden.[5] This syndrome usually begins after 8 to 12 days of satisfactory convalescence and is characterized by: profuse vomiting; a large amount of retained secretions in the gastric remnant; and passage of barium through the stoma and into the efferent loop for several centimeters where it becomes arrested (Fig. 1). This apparent obstruction disappears spontaneously after a period of time, thus suggesting that the failure of the barium to progress is not due to a mechanical obstruction at the stoma but to a physiologic disturbance of the efferent jejunal loop. Four such patients were treated with coherin by Hiatt *et al.*[7] and within 48 hours after initiation of therapy were able to take fluids by mouth.

Another example of functional obstruction following gastric surgery is seen in the following patient:

R.G. Persistent vomiting and weight loss of 2 months' duration was the chief complaint of this 75 year old man, who had a gastroenterostomy for duodenal ulcer 20 years earlier. A diagnosis of partial intestinal obstruction at the region of the stoma was made because of the dilated duodenal loop demonstrated roentgenographically. No improvement either clinically or roentgenologically followed a takedown of his gastroenterostomy. Following 1 week of coherin therapy, marked clinical and roentgenologic improvement was noted (Fig. 2, *A–C*).

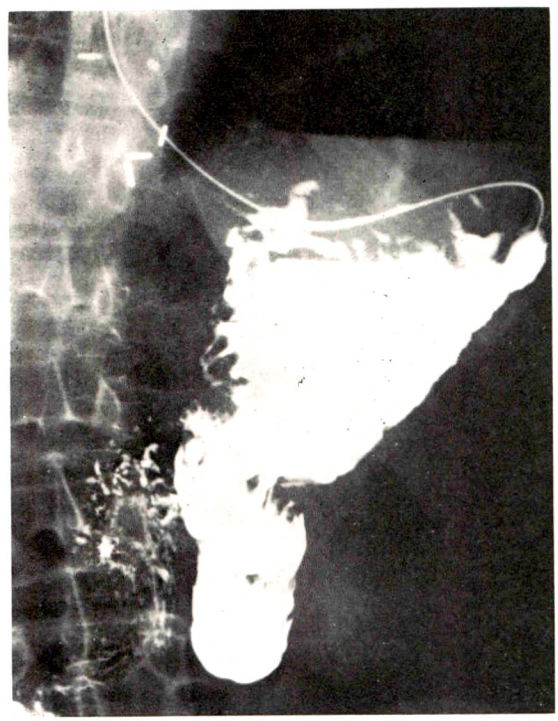


Fig. 1. Functional obstruction of the efferent loop following a Billroth II partial gastrectomy and vagectomy. Abdominal distention and vomiting began on the seventh day. This barium study done on the fifteenth day shows no obstruction at the stoma, but the barium fails to progress more than a few inches down the efferent loop. The patient was given supportive therapy and discharged well on the 45th postoperative day.

Disappearance of the motor activity in the small intestine, or ileus is associated with a wide variety of conditions and classically is associated with ileus of the colon. Air-fluid levels limited to the small intestine are usually regarded as a sign of mechanical obstruction.

At the Columbia-Presbyterian Medical Center, it is the rigid policy that all patients diagnosed as having small bowel obstruction are treated by intestinal intubation. Surgery is carried out only when the obstruction is identified, unless signs of intestinal ischemia develop. Hiatt *et al.*[8]

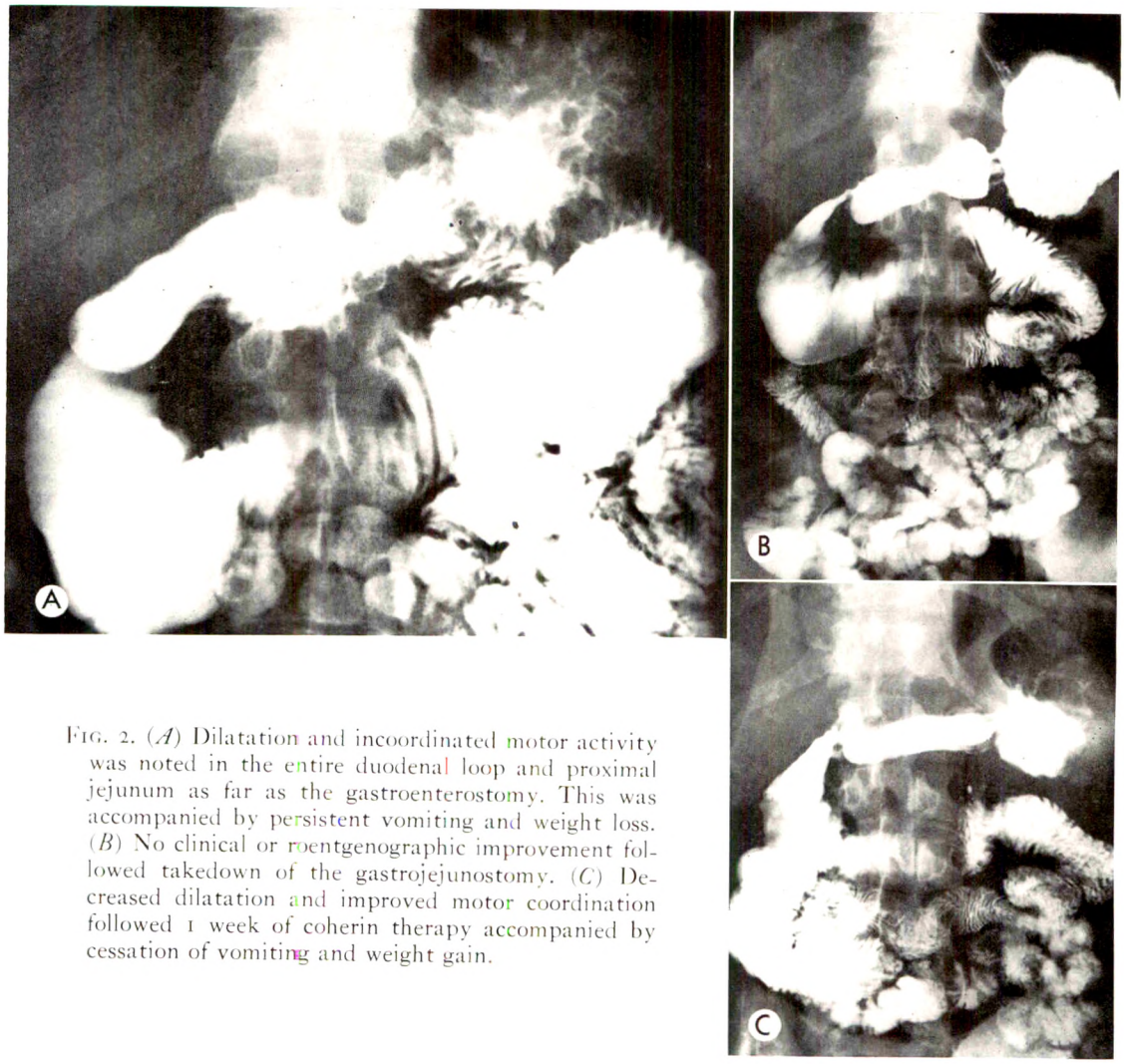


Fig. 2. (*A*) Dilatation and incoordinated motor activity was noted in the entire duodenal loop and proximal jejunum as far as the gastroenterostomy. This was accompanied by persistent vomiting and weight loss. (*B*) No clinical or roentgenographic improvement followed takedown of the gastrojejunostomy. (*C*) Decreased dilatation and improved motor coordination followed 1 week of coherin therapy accompanied by cessation of vomiting and weight gain.

analyzed 345 patients admitted with the clinical and radiologic diagnosis of mechanical small bowel obstruction and found that in only 55 per cent could mechanical occulsion of the intestinal lumen be demonstrated. In 38 per cent the intestinal lumen was patent as evidenced by the passage of an intestinal tube or barium into the colon, or by the spontaneous disappearance of the obstruction. This suggests a motor disturbance as the underlying etiology (Fig. 3, *A* and *B*).

Pseudo-obstruction of the small bowel is a term used to describe a clinical and roentgenologic picture simulating mechanical obstruction in which no organic occlusion of the bowel is present. This may be associated with a wide variety of diseases (Table II). There is a small group with no underlying cause that was first described by Naish *et al.*[10] in 1960. This disease which usually begins in young adults is characterized by episodes of obstruction, abdominal pain and distention, vomiting, diarrhea and steatorrhea.

Roentgenologically there is striking hypomotility of the entire gastrointestinal tract. Despite its similarity to scleroderma, the absence of skin changes, other visceral involvement and the characteristic micro-

scopic findings in the bowel wall of scleroderma, all strongly suggest that this is a separate entity.

Dyer *et al.*[3] found a lesion of the myenteric plexus in a similar patient. The usual histologic techniques revealed no significant abnormalities. When silver preparations were made of sections cut parallel to the bowel, the neurones were found to be reduced in numbers with thick, irregular and clubbed processes. There was an increase in the number of Schwann cells in the nerve trunks and a reduction in the number of axones.

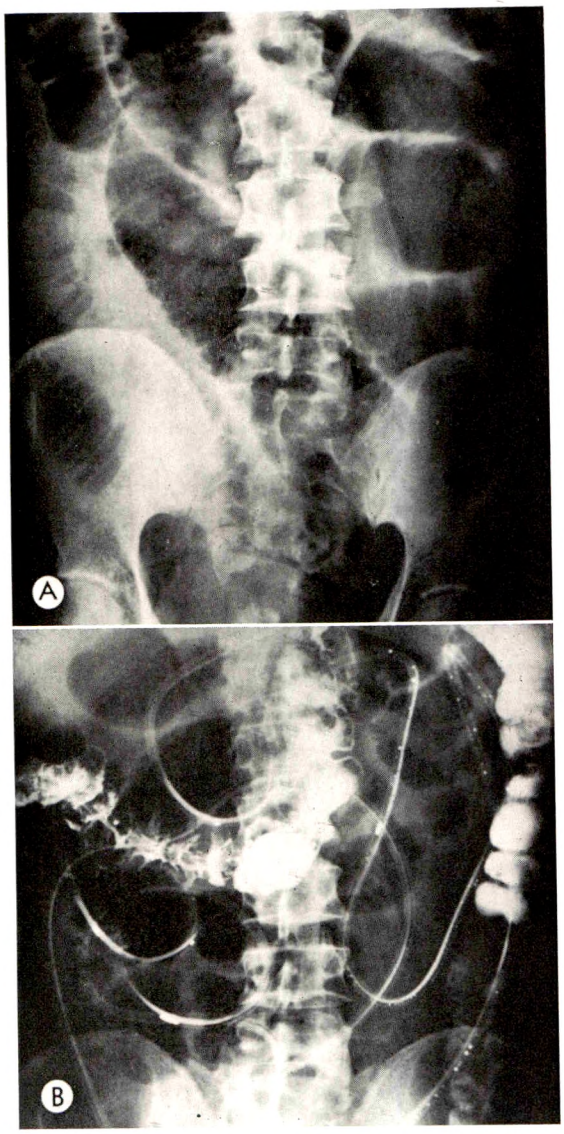


Fig. 3. (*A*) Striking small bowel dilatation. Note absence of colonic gas, thus simulating mechanical small bowel obstruction. (*B*) In 5 days the tube had progressed to the terminal ileum and injected barium passed rapidly into the colon, indicating the absence of mechanical obstruction.

Table II

DISEASES ASSOCIATED WITH PSEUDO-OBSTRUCTION OF THE SMALL BOWEL

1. Renal failure
2. Pneumonia
3. Pancreatitis
4. Congestive heart failure
5. Myxedema
6. Scleroderma
7. Chronic mesenteritis
8. Trauma
9. Nontropical sprue
10. Idiopathic
11. Jejunal diverticulosis
12. Systemic amyloidosis

The following case illustrates this condition:

N.N. For over 12 years this 52 year old man had had chronic steatorrhea and weight loss associated with episodes of intestinal obstruction. Roentgenographic studies revealed striking dilatation of his duodenum and entire mesenteric small bowel (Fig. 4, *A–F*). A resected jejunal segment was found to contain normal ganglion cells, very minimal fibrosis of the lamina propria but no muscle atrophy or vascular changes. Because of the latter a diagnosis of scleroderma was felt to be unlikely. His skin was normal and there was no evidence of Raynaud's disease.

Diverticula. A certain amount of evidence has accumulated that suggests that some of the diverticula observed in the gastrointestinal tract develop because of an underlying motor dysfunction.

The pharyngoesophageal or Zenker's diverticulum is probably the result of dysfunction of the cricopharyngeal sphincter. Ellis *et al.*[4] found an abnormal temporal relationship between the peristaltic wave and the cricopharyngeal sphincter in

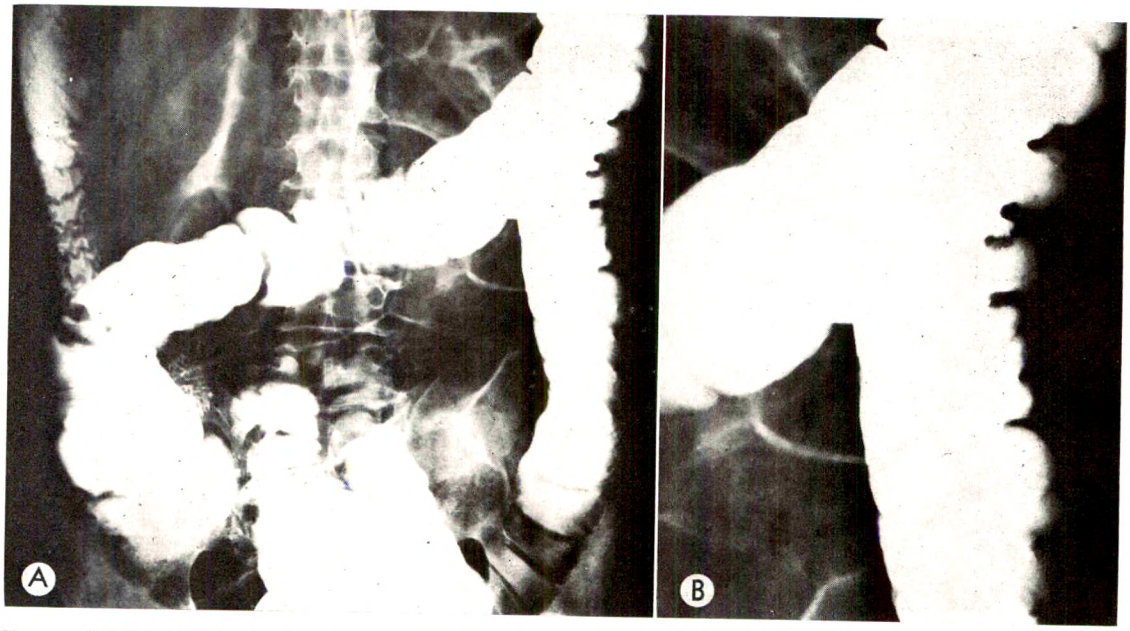


FIG. 4. (*A*) Normal barium enema roentgenogram, except for wide mouth diverticula in the proximal descending colon in a 52 year old man with steatorrhea and weight loss. Note the distended loops of the small bowel. (*B*) Detail view of the wide mouthed diverticula.

all of the 11 patients that were studied manometrically; *i.e.*, part or all of the period of pharyngeal contraction occurred after closure of the sphincter. The resultant high intraluminal pressure conceivably could force the mucosa to herniate through the anatomic weak spot of the cricopharyngeus, Killian's triangle, a gap lo-

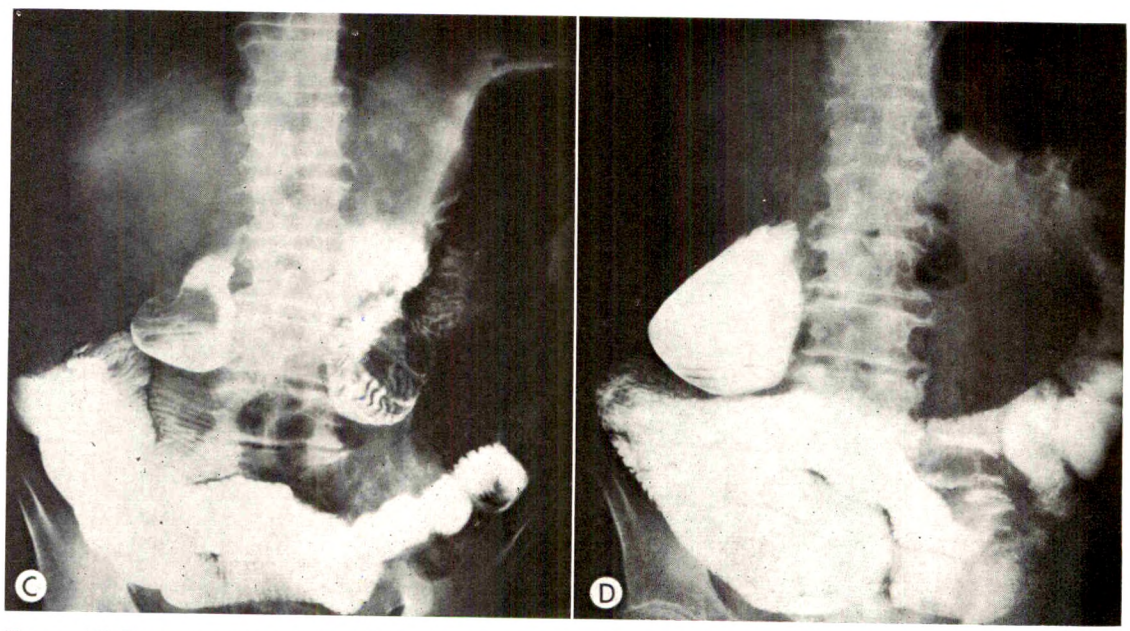


FIG. 4. (*C*) Roentgenogram 5 years later shows markedly dilated duodenum, jejunum and ileum with normal mucosal patterns. (*D*) Note striking dilatation of the duodenal loop in the roentgenogram taken 7 years later.

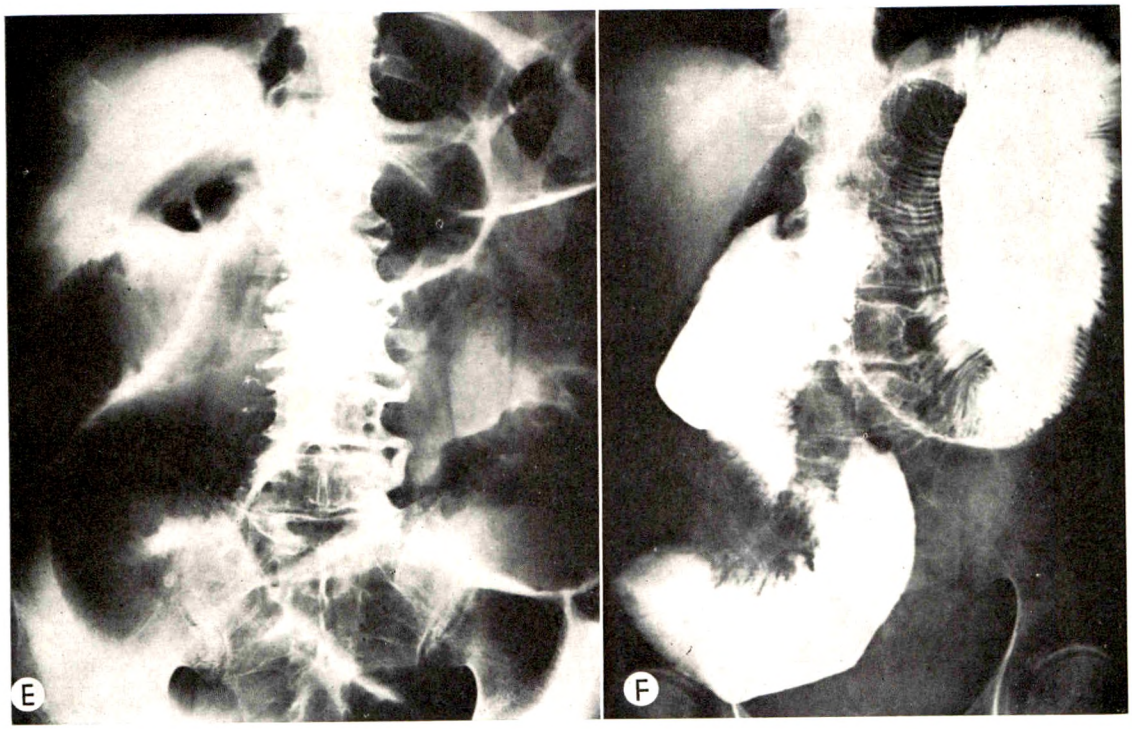


FIG. 4. (*E*) Eight years later, markedly dilated small intestine. (*F*) Dilated loop still present 11 years later.

cated posteriorly between the oblique and transverse muscle fibers. If the underlying mechanism of a Zenker's diverticulum is motor dysfunction of the cricopharyngeus, this should also be evident postoperatively, when the surgical procedure consists of a diverticulectomy only. In a small group of such patients I did find evidence of cricopharyngeal dysfunction (Fig. 5). The series is too small to comment on the frequency of this abnormality. Ellis *et al.*[4] have reported good results in treating patients with pharyngoesophageal diverticula by cricopharyngeal myotomy.

The pathogenesis of the lower esophageal or epiphrenic diverticulum is unknown but its association with motor dysfunction such as achalasia, and lower esophageal spasm is well known. A recent review of 80 epiphrenic diverticula by Bruggeman and Seaman,[2] revealed that most diverticula over 4 cm. were associated with esophageal motor dysfunction (Fig. 6, *A* and *B*). Thus, when patients with epiphrenic diverticula complain of dysphagia, the latter is more

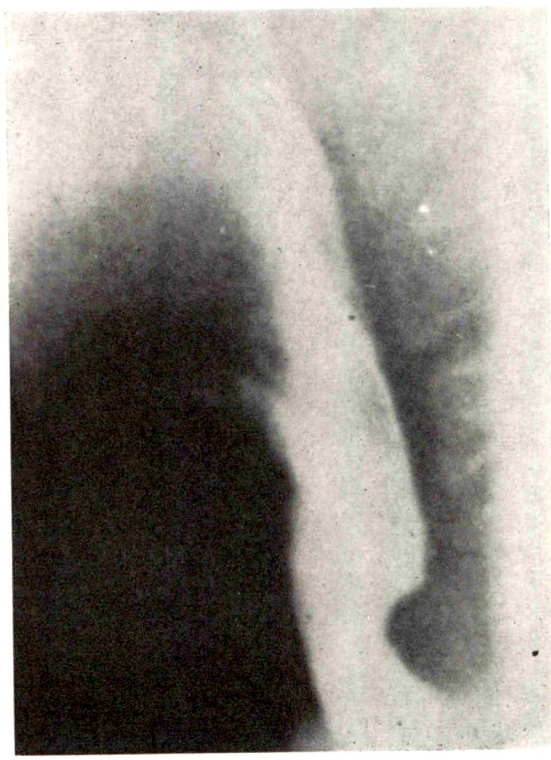


FIG. 5. Cricopharyngeal achalasia in patient following excision of a Zenker's diverticulum.

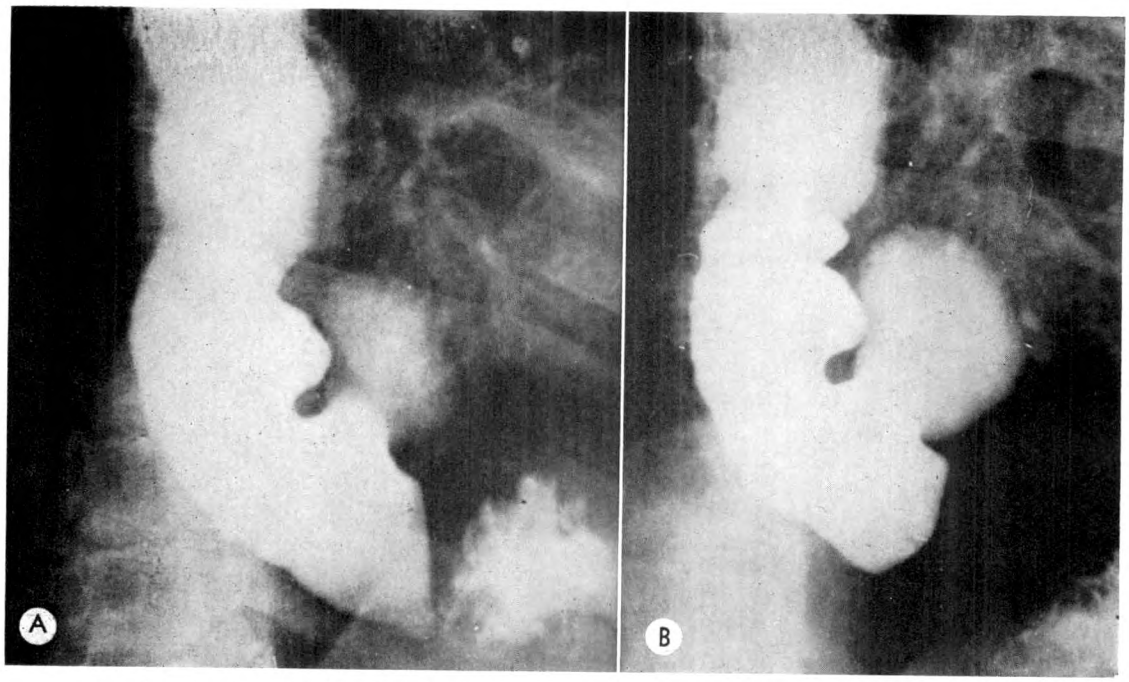


FIG. 6. (*A* and *B*) Epiphrenic diverticulum in a patient with esophageal achalasia.

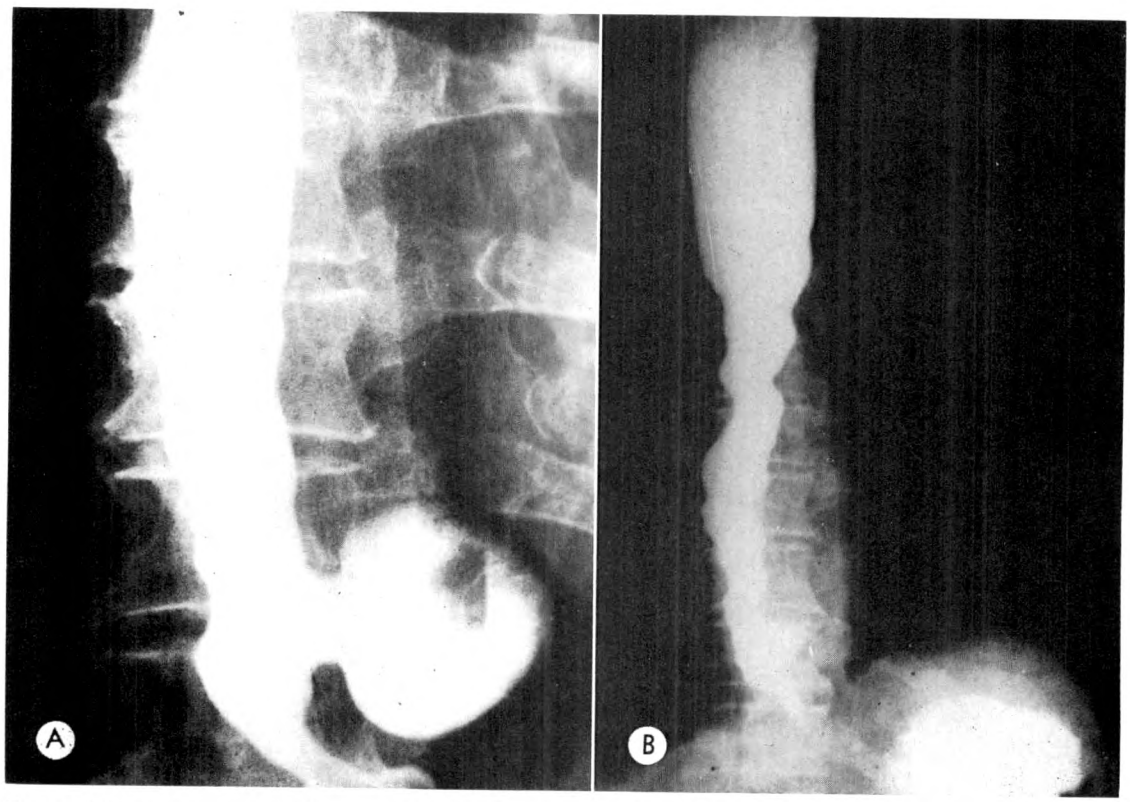


FIG. 7. (*A*) Lower esophageal spasm associated with an epiphrenic diverticulum. (*B*) Dysphagia persisted unchanged following excision of the diverticulum due to persistence of the diffuse esophageal spasm.

likely to be due to the underlying motor disorder than to the diverticulum.

The following case illustrates this point:

Seven years of constant interscapular pain associated with nocturnal regurgitation of food, was the chief complaint of this 61 year old man. Irregular spastic narrowing of his distal esophagus and a moderate sized epiphrenic diverticulum were demonstrated (Fig. 7, *A* and *B*). Following surgical excision of the diverticulum his symptoms continued unabated. Roentgenograms of his esophagus continued to exhibit the typical manifestations of diffuse lower esophageal spasm.

Altemeier *et al.*,[1] in reviewing 62 cases of jejunal diverticulosis, found that chronic, or recurrent jejunal obstruction was the most frequent complication. In the diverticula-bearing segment, muscular contractions were numerous but incoordinated and

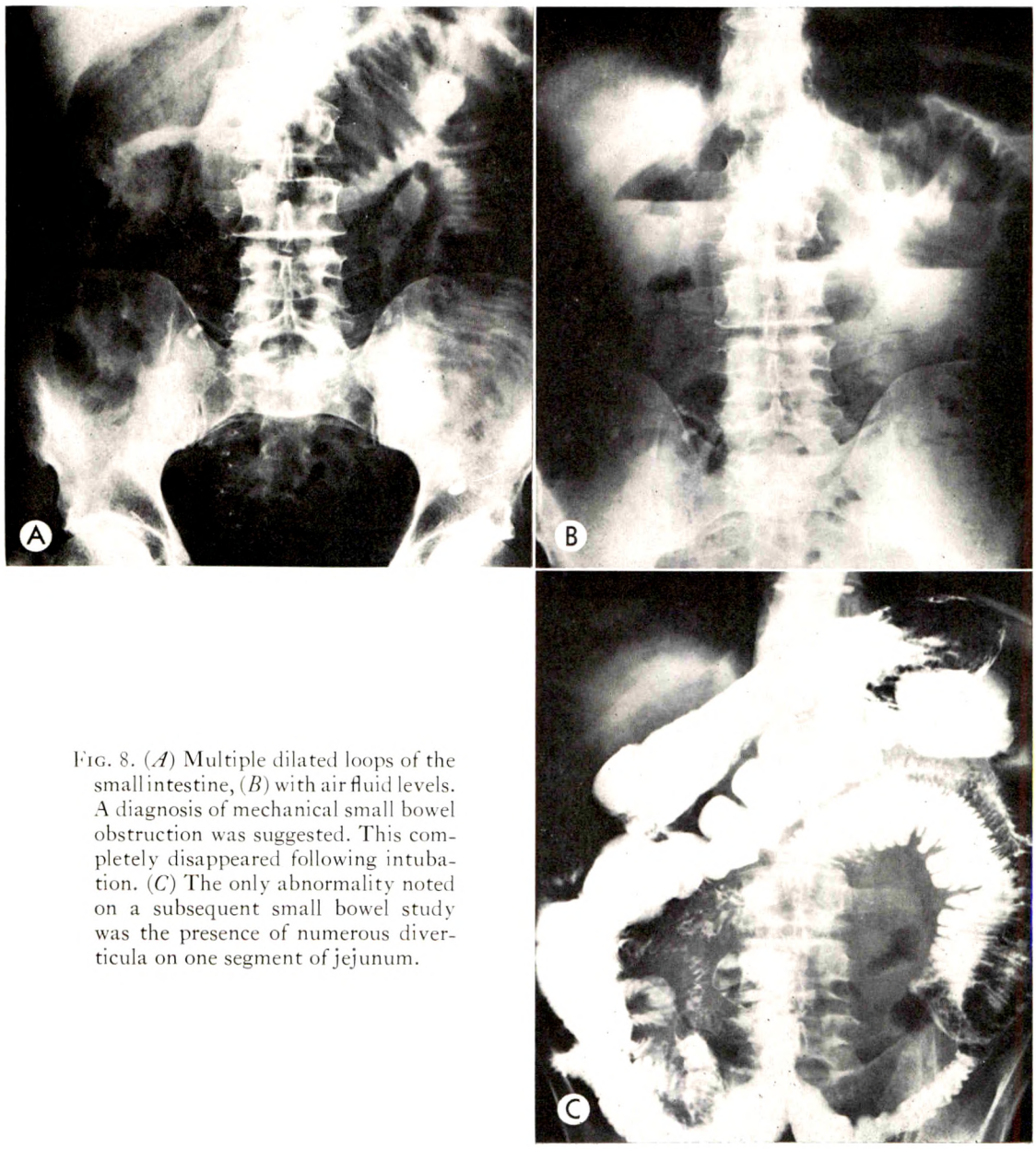


Fig. 8. (*A*) Multiple dilated loops of the small intestine, (*B*) with air fluid levels. A diagnosis of mechanical small bowel obstruction was suggested. This completely disappeared following intubation. (*C*) The only abnormality noted on a subsequent small bowel study was the presence of numerous diverticula on one segment of jejunum.

inefficient causing a functional obstruction. This jejunal dyskinesia, as Altemeier *et al.*[1] term it, was also observed at the time of surgery. The patients were cured by segmental resection of the excised segment which showed muscular hypertrophy. Phillips[11] reported 3 additional cases, relieved by resection or intestinal bypass and cited others in the literature. We have recently had a similar case (Fig. 8, *A–C*).

CONCLUSIONS

A simple classification of motor dysfunction of the gastrointestinal tract from the roentgenologic standpoint is proposed and a few pertinent examples cited.

Dynamic phenomena may be the major cause of the roentgen deformity in some types of organic disease.

Roentgenographic diagnosis of some forms of gastrointestinal disease can be made on the basis of altered motor function.

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REFERENCES

1. Altemeier, W. A., Bryant, L. R., and Wulsin, J. H. Surgical significance of jejunal diverticulosis. *A.M.A. Arch. Surg.*, 1963, *86*, 732–745.
2. Bruggeman, L., and Seaman, W. B. Epiphrenic diverticula—review of eighty cases. To be published.
3. Dyer, N. H., Dawson, A. M., Smith, B. F., and Todd, I. P. Obstruction of bowel due to lesion in myenteric plexus. *Brit. M. J.*, 1969, *1*, 686–689.
4. Ellis, F. H., Jr., Schlegel, J. F., Lynch, V. P., and Payne, W. S. Cricopharyngeal myotomy for pharyngo-esophageal diverticulum. *Ann. Surg.*, 1969, *170*, 340–350.
5. Golden, R. Functional obstruction of efferent loop of jejunum following partial gastrectomy. *J.A.M.A.*, 1952, *148*, 721–724.
6. Hiatt, R. B., Goodman, I., and Alavi, A. Hormonal control of intestinal motility. *Ann. Surg.*, 1967, *166*, 704–711.
7. Hiatt, R. B., Goodman, I., and Overweg, N. I. A. Intestinal motility: control mechanisms in basic electric rhythm of canine small intestine. *J. Surg. Research*, 1971, *11*, 454–463.
8. Hiatt, R. B., Goodman, I., and Overweg, N. A. Intestinal motor dysfunction (arrhythmia) as a major clinical problem. To be published.
9. Hightower, N. C., Jr. Motility of the alimentary canal of man. In: Disturbances of the Gastrointestinal Motility. Charles C Thomas, Publisher, Springfield, Ill., 1959.
10. Naish, J. M., Capper, W. M., and Brown, N. J. Intestinal pseudo-obstruction with steatorrhea. *Gut*, 1960, *1*, 62–65.
11. Phillips, J. H. Jejunal diverticulosis. *Brit. J. Surg.*, 1952–53, *40*, 350–354.
12. Theros, E. G. Value of radiologic-pathologic correlation in education of radiologist. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *107*, 235–258.

ACCELERATION OF THE BARIUM MEAL THROUGH THE SMALL INTESTINE BY THE C-TERMINAL OCTAPEPTIDE OF CHOLECYSTOKININ*

By PAVO HEDNER *and* GUNNE RORSMAN
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THE passage of barium meal through the small intestine is a time consuming examination. It is tiresome to the patient and it disturbs the routine in the Department of Roentgenology. Among several methods devised to decrease the time needed for the intestinal transit, the administration of cholecystokinin has proved to be one of the most effective.[9,12] However, the limited supply of extracted cholecystokinin has greatly reduced its use in routine clinical work.

In 1968, Mutt and Jorpes[10] were able to elucidate the structure of porcine cholecystokinin and observed that the C-terminal octapeptide of the molecule was highly active. This C-terminal octapeptide of cholecystokinin was synthesized by Ondetti *et al.*,[11] and in animal experiments was found to possess all the activities of extracted cholecystokinin including the stimulation of the propulsive peristalsis of the small intestine.[5,13] It has been investigated in man and found to be effective in evacuating the contrast medium filled gallbladder.[6]

As the synthetic C-terminal octapeptide of cholecystokinin (C8-CCK) is available in a pure form and offers the advantage of being highly active, it might be expected to be an alternative to extracted cholecystokinin for routine clinical use. Therefore, we have investigated whether C8-CCK, like extracted cholecystokinin, could be used for accelerating the passage of barium meal through the small intestine in man.

MATERIAL AND METHOD

Ten healthy subjects, 4 women and 6 men, aged 20–45 years, were investigated. After fasting for at least 6 hours, the subjects were given 200 ml. of a barium sulphate suspension (Mixobar, Astra A.B., Sweden). They were then asked to lie on their right side for 20 minutes. An intravenous infusion was then started, containing 0.13 μg. of C8-CCK per ml. of Ringer's solution. The infusion rate was kept at 3 ml. per minute by means of an automated infusion unit (Decca Type 460). Roentgenograms were made before the infusion and then every 5 minutes until the barium meal had reached the cecum.

Seven to 10 days later the investigation was repeated in the same manner, but C8-CCK was not given. Roentgenograms were now made at intervals that in each individual corresponded to the time needed in the first investigation (with C8-CCK) to recognize contrast medium in the cecum. Four roentgenograms were made at this time interval, and then, if needed, hourly until contrast medium was found in the cecum.

Before, and 7–10 days after the administration of C8-CCK the following laboratory tests were performed: Hemoglobin concentration, RBC, WBC, differential count, serum concentrations of Ca^{++}, Na^{+}, K^{+}, Cl^{-}, bicarbonate, phosphate, total protein, albumin, globulin, BUN, creatinine, bilirubin, and activity of GOT, GPT, LDH and alkaline phosphatases in serum.

C8-CCK (SQ 19 844) was obtained as a gift from Dr. M. A. Ondetti, The Squibb Institute for Medical Research, New Brunswick, N. J., U. S. A. The potency of this substance regarding the effect on the small intestine has been assayed to 36,000 Ivy dog units per mg.[5] The substance was dissolved in 0.9 per cent NaCl to a stock con-

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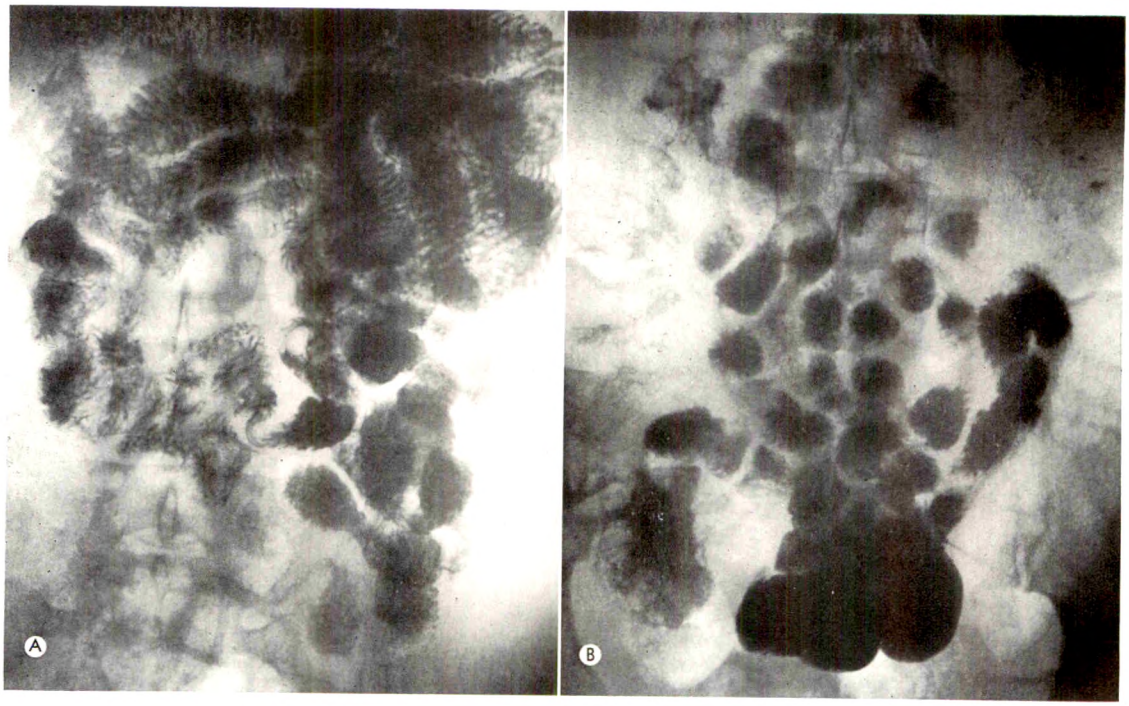


FIG. 1. Roentgenograms (*A*) 10 minutes and (*B*) 20 minutes after the beginning of infusion of C8-CCK.

centration of 0.33 μg. per ml., and sterile filtered.

RESULTS

Twenty minutes after the ingestion of the barium meal, with the subjects lying down and turned to the right, the contrast medium had regularly passed into the distal part of the duodenum. The infusion of C8-CCK was then started. During the infusion, the contrast medium reached the cecum within 20 minutes in all cases, leaving a contrast coating on the mucosal surface of almost the whole small bowel (Fig. 1, *A* and *B*).

Two of the subjects experienced a moderate abdominal discomfort for a few minutes during the infusion of C8-CCK, but otherwise no side reactions were observed.

When the investigation was repeated 7–10 days later without C8-CCK, the intestinal transit time was significantly longer, and the difference was considerable in several cases (Fig. 2).

The laboratory parameters listed above showed no significant changes following the administration of C8-CCK.

DISCUSSION

In an investigation of intestinal function, the unbiased passage time for the barium meal through the small intestine may be an important item of information. Therefore, an acceleration of the intestinal transit should be restricted to cases where only morphologic changes are questioned.

An acceleration of the intestinal transit can be induced by intramuscular neostigmine or by the ingestion of ice water. Both methods have been shown to produce a passage time for the barium meal of 60 minutes or less in about 75 per cent of 240 healthy subjects.[4] Compared to these results, cholecystokinin and related compounds seem to be more effective. Thus the intestinal transit time was found to be 15 minutes or less in 8 of 11 normal individuals following the administration of extracted cholecystokinin,[12] 20–40 minutes following caerulein,[3]

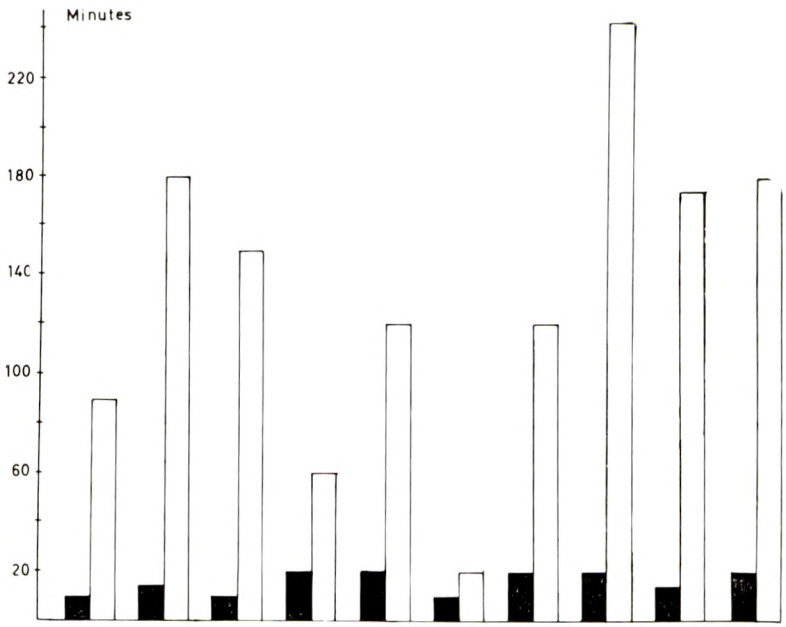


Fig. 2. Intestinal transit times in 10 healthy subjects with C8-CCK (black columns) and without C8-CCK (white columns), respectively.

and 20 minutes or less following C8-CCK in the present investigation.

The limited supply of extracted cholecystokinin prevents its use in routine clinical work. Therefore, increasing interest has been focused on its synthetic relatives, caerulein and C8-CCK.

Caerulein is a decapeptide that originally was extracted from the skin of the frog *Hyla caerulea*. Its structure is slightly different from the C-terminal part of porcine cholecystokinin.[1] C8-CCK is an octapeptide with a structure identical to that of the C-terminal part of porcine cholecystokinin.[10]

Cholecystokinin and related compounds seem to relax the proximal portion of the duodenum, but to stimulate its distal portion.[3] Thus C8-CCK can be expected to delay the passage through the proximal duodenum, if the administration is started too soon after the ingestion of the contrast medium. To obtain the striking decrease in the intestinal transit time that is possible with the aid of C8-CCK, we believe that it is important not to start the infusion until the barium meal has passed into the distal portion of the duodenum. This was regularly achieved within 20 minutes when the subjects were lying on their right side.

In vitro experiments have shown that cholecystokinin and C8-CCK act on a postsynaptic cholinergic neuron of the intestinal nerve plexus.[5,7] Thus the effect should be independent of the action on the gallbladder. However, some patients have been observed who did not decrease their intestinal transit time after cholecystokinin.[2] The mechanism for this is not known, but a common factor in these patients was found to be cholecystopathy.[2]

In animal experiments,[13] as in man,[6] C8-CCK has been found to have a shorter duration of action than extracted cholecystokinin. For visualization of the bile ducts in connection with cholecystography, the short duration of action of C8-CCK is no drawback,[6] but for acceleration of the intestinal transit intravenous infusion seems to be necessary. The use of C8-CCK as

described in the present investigation permitted the passage of the barium meal from the duodenum to the cecum within 20 minutes in all cases. With respect to the long time otherwise needed,[8,14] the use of C8-CCK indicates obvious advantages both to the patient and to the radiologist who can make the examination in an uninterrupted sequence with spot roentgenography of areas of interest, guided by television fluoroscopy.

SUMMARY

During the intravenous infusion of the synthetic C-terminal octapeptide of cholecystokinin (C8-CCK), the barium meal passed from the distal portion of the duodenum to the cecum within 20 minutes in 10 healthy subjects. A contrast medium coating was left on the mucosal surface of almost the whole small intestine.

This technique permits the examination of the small bowel in an uninterrupted sequence under fluoroscopy with spot roentgenography of areas of interest.

No harmful side effects were encountered.

Without C8-CCK, the corresponding transit times in the same subjects were 30–240 minutes.

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REFERENCES

1. Anastasi, A., Erspamer, V., and Endean, R. Isolation and structure of caerulein, active decapeptide from skin of Hyla caerulea. *Experientia*, 1967, *23*, 699–700.
2. Backlund, V. Kolecystokinin vid röntgenundersökningar. *Läkartidningen*, 1967, *64*, 2473–2476.
3. Bertaccini, G., and Agosti, A. Action of caerulein on intestinal motility in man. *Gastroenterology*, 1971, *60*, 55–63.
4. Friedenberg, M. J., McAlister, W. H., and Margulis, A. R. Roentgen study of small bowel in adults and children with neostigmine. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1962, *88*, 693–70.
5. Hedner, P. Effect of C-terminal octapeptide of cholecystokinin on guinea pig ileum and gallbladder in vitro. *Acta physiol. scandinav.*, 1970, *78*, 232–235.
6. Hedner, P., and Lunderquist, A. Use of C-terminal octapeptide of cholecystokinin for gallbladder evacuation in cholecystography. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *116*, 320–326.
7. Hedner, P., and Rorsman, G. Structures essential for effect of cholecystokinin on guinea pig small intestine in vitro. *Acta physiol. scandinav.*, 1968, *74*, 58–68.
8. Kim, S. K. Small intestinal transit time in normal small bowel study. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *104*, 522–524.
9. Morin, G., Besancon, F., Grall, A., Jouve, R., and Debray, C. Technique d'accélération du transit du grêle. *Arch. mal. app. digest.*, 1965, *54*, 1285–1290.
10. Mutt, V., and Jorpes, J. E. Structure of porcine cholecystokininpancreozymin. I. Cleavage with thrombin and with trypsin. *European J. Biochem.*, 1968, *6*, 156–162.
11. Ondetti, M. A., Pluscec, J., Sabo, E. F., Sheehan, J. T., and Williams, N. Synthesis of cholecystokinin—pancreozymin. I. C-terminal dodecapeptide. *J. Am. Chem. Soc.*, 1970, *92*, 195–199.
12. Parker, J. G., and Beneventano, T. C. Acceleration of small bowel contrast by cholecystokinin. *Gastroenterology*, 1970, *58*, 679–684.
13. Rubin, B., Engel, S. L., Drungis, A. M., Dzelzkalns, M., Grigas, E. O., Waugh, M. H., and Yiacas, E. Cholecystokinin like activities in guinea pigs and dogs of C-terminal octapeptide SQ 19844 of cholecystokinin. *J. Pharmacol. Sc.*, 1968, *13*, 443–454.

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ROENTGENOGRAPHIC ASPECTS OF INTESTINAL ISCHEMIA*

By STOVER L. SMITH, M.D., ROGER H. TUTTON, M.D., *and* SEYMOUR FISKE OCHSNER, M.D.
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THE diagnostic roentgenologist has become increasingly interested in intestinal ischemia because this disease is now recognized as being relatively common and because the roentgenologic findings are so important in its recognition.

It seemed worthwhile to present a workable classification of clinical varieties as seen by the roentgenologist, and to illustrate these in this brief review.

CLASSIFICATION

In Table 1 we have presented a simple working classification which is based on the presenting clinical and/or roentgenographic findings. The changes in the mesenteric circulation may be either occlusive or nonocclusive. The nonocclusive form may present early as an acute illness, or late as a stenosis or an incidental roentgenographic finding. The occlusive variety may present as an acute or chronic illness. These then are the 4 major categories. Even though on a pathogenetic basis there may be overlapping categories, it is usually possible to classify any patient into one of the above. By such classification the appropriate therapy can be suggested.

TABLE I

CLASSIFICATION OF INTESTINAL ISCHEMIA

I. Nonocclusive Intestinal Ischemia
  A. Early phase
  B. Late phase
II. Occlusive Intestinal Ischemia
  A. Acute
    a. Arterial embolism or thrombosis
    b. Surgical (ligature)
    c. Trauma to vessel
    d. Dissecting aneurysm
    e. Venous thrombosis
    f. Intestinal obstruction
  B. Chronic
    a. Arteriosclerosis
    b. Celiac axis syndrome
    c. Arteritis (scleroderma, polyarteritis, etc.)

NONOCCLUSIVE INTESTINAL ISCHEMIA

Early phase. Nonocclusive lesions account for more than 50 per cent of the cases seen clinically. The most common presenting complaints are abdominal pain and bleeding from the gastrointestinal tract (Fig. 1, *A* and *B*). Price and co-workers[8] have emphasized that the pain may initially be colicky and associated with spasm of early ischemia; later, it may become intense and more constant. Occult or gross blood per rectum is common, but hematemesis may occur. A decreased cardiac output is almost always the underlying pathophysiologic mechanism in these patients. Patients with congenital or acquired heart disease are therefore prime candidates for this type of complicating disease. It has been documented that arrhythmias such as ventricular tachycardia have caused transient intestinal ischemia.[1] Even though these patients may seem relatively well during an attack, the intestinal ischemic episode may become irreversible leading to death. Modern statistics indicate that these lesions have a higher incidence than previously realized, and the area of the splenic flexure is the commonest site of ischemic change.[6]

Late phase. The acute phase may resolve without residual clinical or roentgenographic findings. However, frequently there is residual stenosis or loss of normal haustral pattern. The degree of stenosis will determine whether or not there is intestinal obstruction (Fig. 2, *A* and *B*).

* Presented at the Seventy-second Annual Meeting of the American Roentgen Ray Society, Boston, Massachusetts, September 28–October 1, 1971.

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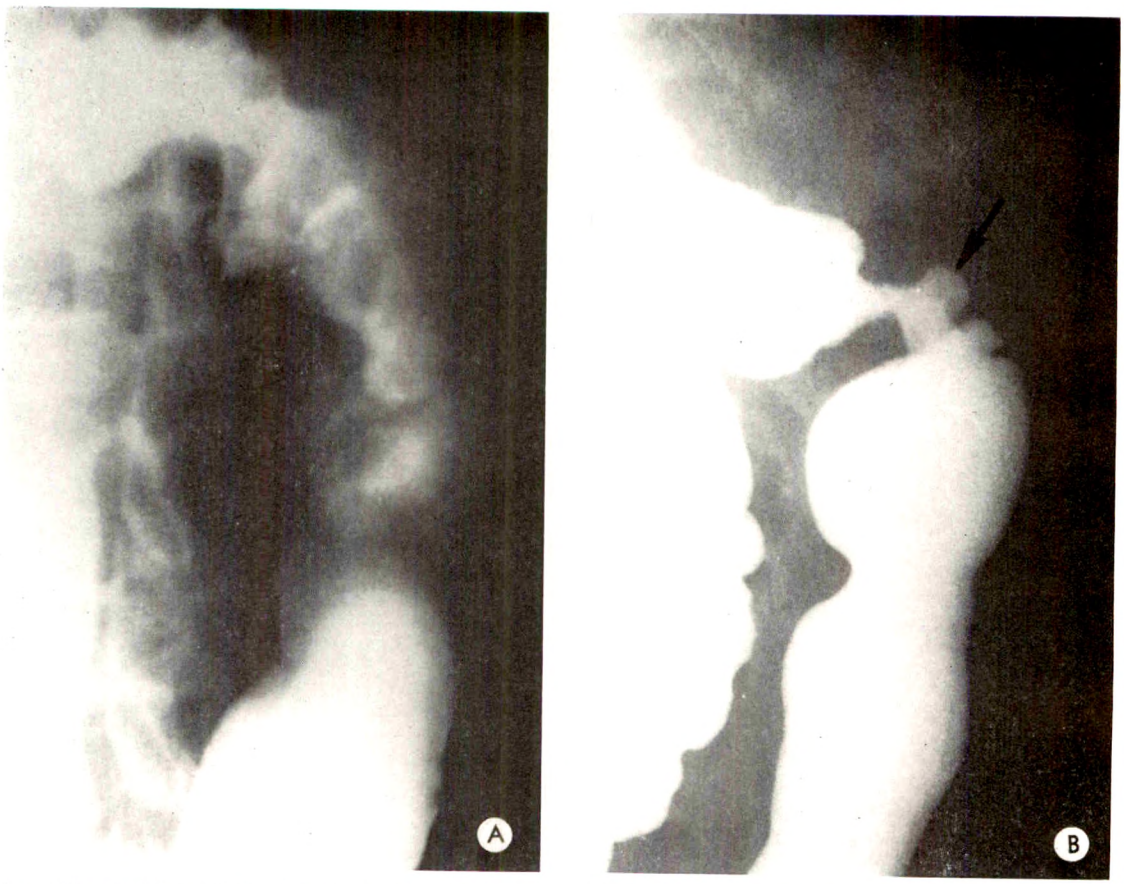


FIG. 1. Case 1. *Nonocclusive intestinal ischemia.* A 40 year old female presented with acute abdominal pain and rectal bleeding. The initial barium enema examination revealed "cobblestone" appearance confined to the splenic flexure (*A*). Within 3 weeks a localized stenosis and ulceration developed (*B*). There were no vascular occlusions and microscopic diagnosis was ischemic colitis. (Courtesy of Dr. Thomas Weatherall.)

OCCLUSIVE INTESTINAL ISCHEMIA

Acute. Clinically acute occlusive vascular disease is of sudden onset, with abdominal pain the dominant symptom. Vomiting, diarrhea, or tenesmus are frequently associated. Occasionally there is rectal bleeding.

Embolism is the most frequent cause of acute occlusive disease that results in intestinal ischemia. The usual source is some type of cardiac disease, of which arteriosclerosis is by far the most common etiologic factor. Arterial thrombosis of mesenteric vessels is usually preceded by atherosclerosis. Recent reports suggest that the "pill" used in contraception may be a cause of acute thrombosis of small vessels in young women.[4] Similar thrombosis may occur in association with arteritis. Acute venous thrombosis, by contrast, may be an isolated phenomenon, or may develop secondary to such conditions as intestinal obstruction, cirrhosis, portal hypertension, or abdominal tumors as, for example, pancreatic carcinoma.

Aneurysms of the aorta deserve special comment. When an aneurysm is removed surgically, the inferior mesenteric artery is frequently sacrificed. The surgeon usually checks the "back bleeding" from the orifice of this artery, to assure himself that there is satisfactory collateral filling of the artery. Even when this is done, however, ischemia of the distal colon may occur due to inadequate collateral circulation after ligation of the vessel (Fig. 3, *A–C*).

In dissecting aneurysms abdominal symp-

toms may be present, due to intestinal ischemia. The symptoms related to the abdomen may even predominate and obscure the supposedly classical thoracic symptoms. If the aorta is not grossly enlarged, or if the dissecting process is not suspected, the presence of aneurysm may be overlooked at the time of surgical exploration. Preoperative arteriography, of course, will greatly lessen the possibility of this mistake.

Chronic. In these patients, the clinical history usually features abdominal pain and loss of weight. Classically, the pain is postprandial, and has been termed "intestinal angina." In some patients a malabsorption pattern is present both roentgenographically and on laboratory studies. Weight loss is usually from food avoidance rather than from failure of absorption. Frequently, carcinoma of the pancreas is erroneously suspected.

Arteriosclerosis is the most common cause of chronic intestinal ischemia. There are several potential collateral arteries in the visceral circulation, and therefore stenosis or occlusion of all 3 of the major arteries supplying the gastrointestinal tract is usually required before the patient becomes symptomatic. This means that the celiac, superior mesenteric and inferior mesenteric arteries must be demonstrated with angiography before one can determine the full extent of the disease process.

As in other organs, the degree and extent of involvement are the determining factors in potential success of surgical correction. If the stenosis is localized, the prognosis is good. If the arteries are diffusely involved, the prognosis for surgical correction is poor (Fig. 4, *A* and *B*). Background disease processes are sometimes important, such as diabetes, which may be associated with early atherosclerosis. In some instances,

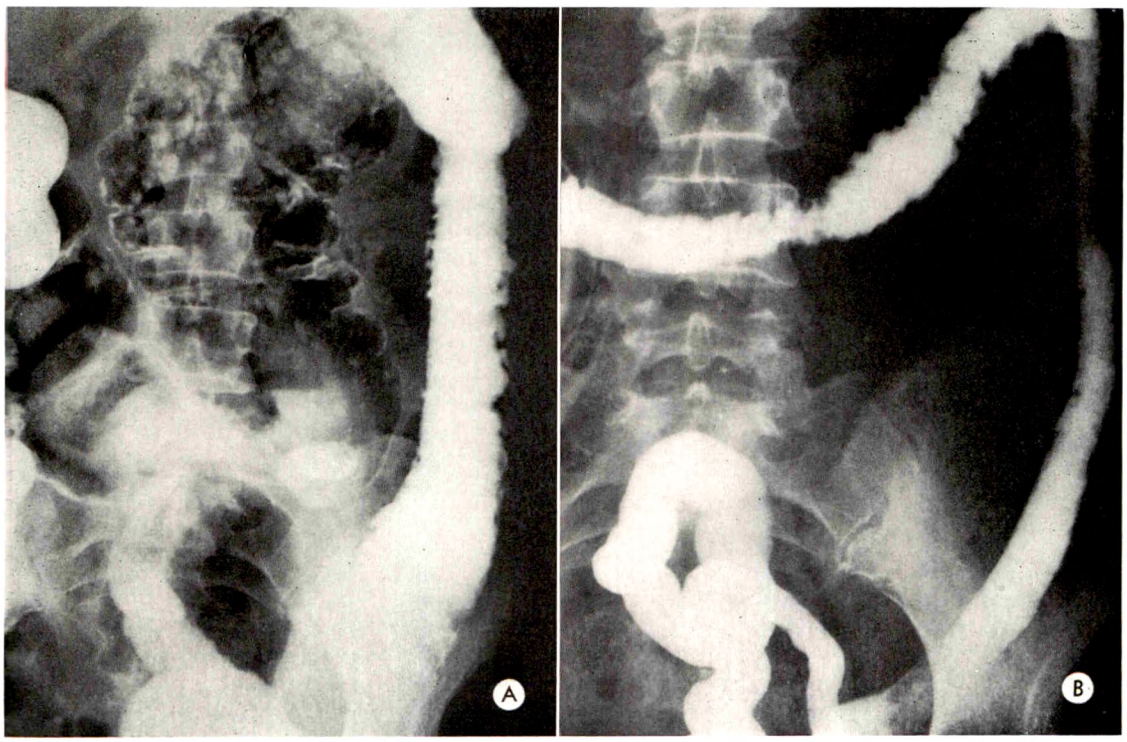


Fig. 2. Case 11. *Nonocclusive intestinal ischemia.* This 58 year old female with mitral valvular prosthesis developed abdominal pain, diarrhea and rectal bleeding following a percutaneous liver biopsy. A colon examination 17 days later demonstrated deep ulcerations in the descending colon and gaseous distention proximally (*A*). There was no major vessel occlusion, so a diverting colostomy was done. Six months later there was diffuse narrowing of the descending colon (*B*).

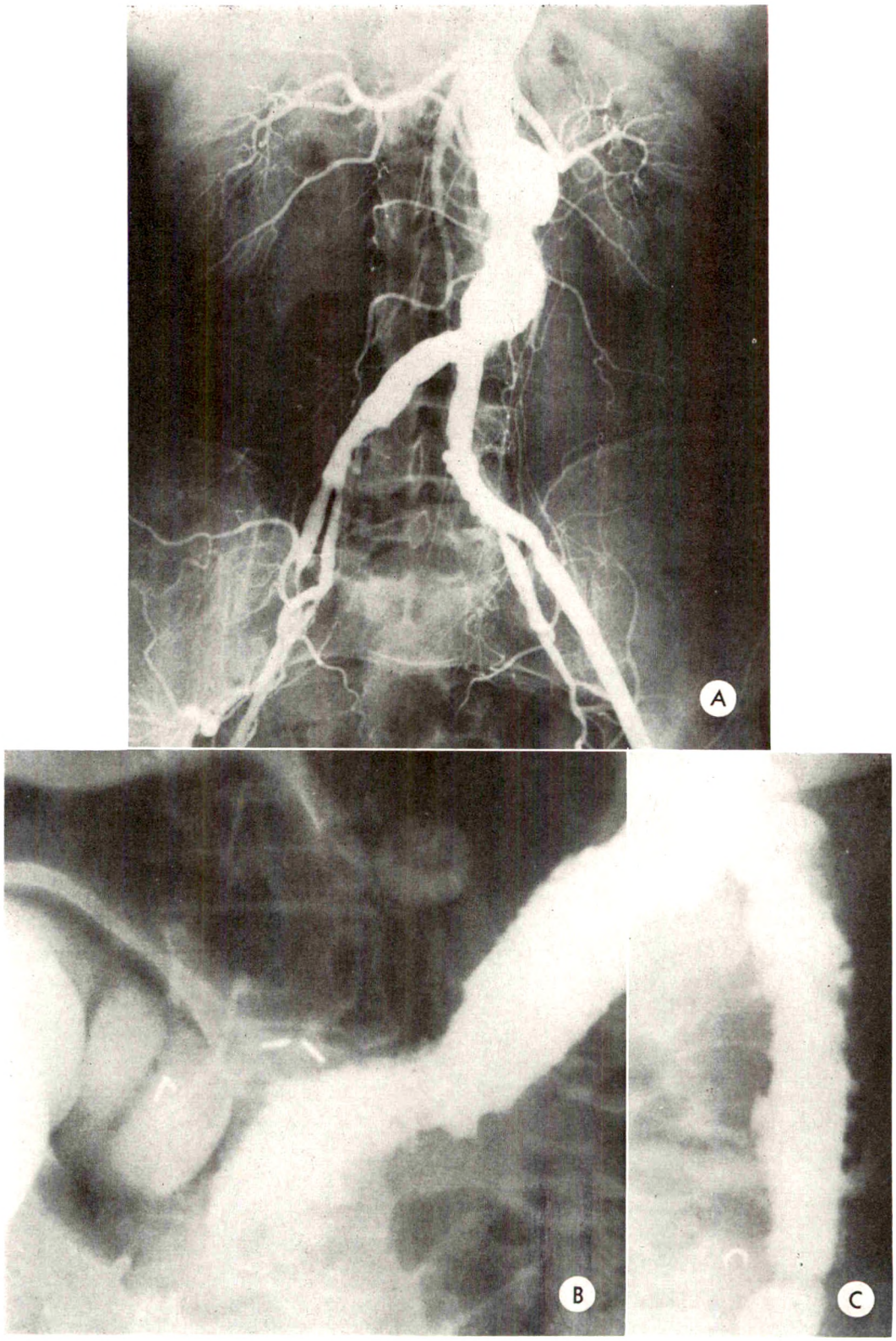


FIG. 3. Case III. *Acute occlusive intestinal ischemia.* Following aneurysectomy and Y grafting beyond right common iliac stenosis (*A*), this 65 year old female developed abdominal pain and diarrhea. Colon examination 12 days later revealed deep ulcerations and proximal dilatation of the normal bowel (*B* and *C*).

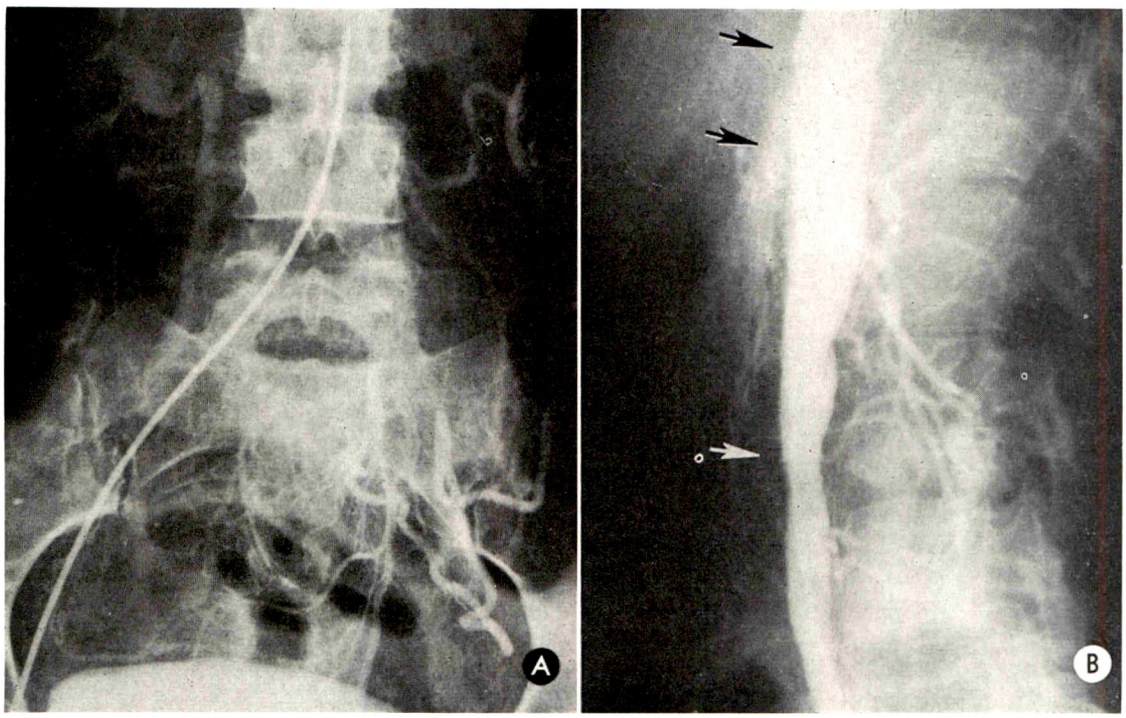


Fig. 4. Case IV. *Chronic occlusive intestinal ischemia.* A 41 year old female presented with abdominal pain for 12 months and a 40 pound weight loss. Abdominal aortogram demonstrated retrograde filling of the inferior mesenteric and marginal arteries via the superior hemorrhoidal artery (*A*). The lateral aortogram demonstrated lack of direct filling of celiac, superior mesenteric or inferior mesenteric arteries (*B*).

however, vascular occlusion may develop as early as the mid forties, without any known predisposing disease.

The celiac compression syndrome[5] remains a controversial and poorly understood form of intestinal ischemia. This form is usually seen in females below the menopausal age. It may be caused by a compression of the celiac axis by the median arcuate ligament, or reportedly neurofibrosis of the celiac ganglion.[3] Relief of symptoms has been gained by surgical intervention in the great majority of the reported 262 cases.[11] Rian and Larowe[10] have recently reported that 35 per cent of 162 patients, who had abdominal angiography for reasons other than intestinal angina, had "arcuate ligament compression." They concluded that in their series there was no correlation between the clinical symptoms and the presence of collaterals or degree of stenosis.

The type of arteritis that is seen in scleroderma, polyarteritis and rheumatoid arthritis has been implicated in some patients, but this is a relatively rare cause of ischemia. Fibromuscular dysplasia and retroperitoneal fibrosis are potential causes of intestinal ischemia.

ROENTGENOGRAPHIC FINDINGS

Plain roentgenograms. On roentgenograms made soon after the onset of an attack, a paucity of gas in the bowel is supposed to be characteristic. This "gasless abdomen" is not usually discovered, however, perhaps because roentgenographic examination is not performed early enough during the course of events. More often, a nonspecific type of paralytic ileus is found. The explanation for this development is that the ischemic segment of bowel becomes nonfunctional, acting in effect as a physiologic obstruction even with an open lumen. Rendich and Harrington[9] were among the first to stress this localized ileus, and later Frimann-Dahl[2] called attention to the

water density mass that may be present if the loop is filled with fluid.

The air within the lumen of an affected segment of intestine may provide information about the inner contour of the intestine. The most significant change is a nodular appearance of the mucosal surface, produced by localized areas of edema. When this appearance is present, in a patient with suggestive history, intestinal ischemia should be suspected. Nelson and Eggleston[7] pointed out that a narrowed, rigid loop may develop, exhibiting little change in position despite change in patient's position, and should suggest mesenteric vascular disease.

Another finding on plain roentgenograms is intramural gas, known also as pneumatosis intestinalis. This pneumatosis may be a relatively benign and unrelated process when saccular in appearance, but a linear appearance is usually indicative of severe disease and is associated with a poor prognosis. Occasionally, air has been found also in the portal veins indicating severe disease and intestinal necrosis.

Gastrointestinal studies with barium. Examination of the colon by barium enema, and of the small bowel by ingested barium, is well tolerated by patients who are not critically ill. The findings in the acute phases of both nonocclusive and occlusive types may be only of irritability of a portion of the intestine, with difficulty of filling and localized spasm. Usually, there are more conclusive findings that are related especially to the mucosal surface of the intestine.

In some patients, the inflamed mucosa may form mounds of edematous tissue that produce an irregular mucosal surface (Fig. 1*A*). When these heaped up mounds of edematous mucosa and submucosa are seen in profile, either by contrasting air or more commonly barium, the descriptive term of "thumbprinting" has been used. It is not specifically diagnostic of ischemic disease, but should raise consideration of the possibility of this disease. If the "thumbprinting" is numerous and small, the surface may present a "cobblestone appearance." Ulcer craters may be seen when they extend into the mucosa, or more deeply into the submucosa of the intestine (Fig. 2*A*). Wang and Reeves[12] were among the earliest to illustrate these ulcerations and to point out that regional ileitis and ulcerative colitis were among the differential diagnoses to consider.

If the patient survives the acute ischemic episode without surgery, the roentgenographic findings may: (1) return to normal; (2) return to normal lumen but with loss of normal haustra, or formation of sacculations; or (3) form varying degrees of stricture. Strictures may develop in 3–4 weeks (Fig. 1*B*), or it may be longer if the active process is longer. Strictures have been "discovered" on routine examinations which have resulted from previous ischemic episodes which did not cause the patient to seek medical attention. The narrowing is usually relatively smooth in contour, has tapering margins where it joins the normal intestine proximally and distally, and is not associated with tenderness or spasm.

Angiography. The use of angiography should be considered whenever the diagnosis of intestinal ischemia is suspected, and when the facilities and personnel for an adequate examination are available. Visualization of the visceral arteries, especially the celiac axis, superior mesenteric and inferior mesenteric arteries, is necessary to differentiate occlusive from nonocclusive ischemic disease. This also provides the pathologic and anatomic details necessary for more definite diagnosis and the most appropriate medical or surgical treatment. It is almost always necessary to supplement the frontal roentgenograms with lateral and, at times, oblique projections. Lateral projections are especially important so that the proximal portion of the celiac and mesenteric arteries are well visualized, and because many of the lesions involve the orifices and proximal few centimeters of these arteries.

SUMMARY

Until recent years, the general concept

of vascular disease involving the intestine was of overwhelming infarction leading to rapid death unless corrected surgically. While this type of involvement may occur, there has been increasing recognition of a whole spectrum of intestinal ischemic states. Some are mild and chronic. Long-standing symptoms such as abdominal pain, weight loss, disturbed intestinal peristalsis, malabsorption, or fatty diarrhea may be the result of ischemic intestinal abnormality.

The possibility of a vascular abnormality as a cause for such complaints should be more promptly recognized by clinicians and by radiologists. Plain roentgenograms and contrast studies of the bowel may be interpreted with greater understanding and discrimination if the patterns produced by ischemia are more widely recognized.

Arteriography then gives the diagnostic roentgenologist a very accurate method of identifying the presence of vascular disease, of localizing with precision, and studying the pattern of abnormal vascular flow. The ability to obtain this information about visceral vessels is of much more than academic interest. It permits prompt decision as to whether medical or surgical management is required and provides the vascular surgeon with the anatomic detail he needs ahead of time.

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REFERENCES

1. Corday, E., Irving, D. W., Gold, H., Bernstein, H., and Skelton, R. B. Mesenteric vascular insufficiency. *Am. J. Med.*, 1962, *33*, 365–375.
2. Frimann-Dahl, J. Roentgen examination in mesenteric thrombosis. Am. J. Roentgenol. & Rad. Therapy, 1950, *64*, 610–616.
3. Harjola, P. T., and Lohtiharju, A. Celiac axis syndrome. *Am. J. Surg.*, 1968, *115*, 864–869.
4. Hurwitz, R. L., Martin, A. J., Grossman, B. E., and Waddell, W. R. Oral contraceptives and gastrointestinal disorders. *Ann. Surg.*, 1970, *172*, 892–896.
5. Marable, S. A., Kaplan, M. F., Beman, F. M., and Molnar, W. Celiac compression syndrome. *Am. J. Surg.*, 1968, *115*, 97–102.
6. Marston, A., Phelis, M. T., Thomas, M. L., and Morson, B. C. Ischemic colitis. *Gut*, 1966, *7*, 1–15.
7. Nelson, S. W., and Eggleston, W. Findings on plain roentgenograms of abdomen associated with mesenteric vascular occlusion with possible new sign of mesenteric venous thrombosis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1960, *83*, 886–894.
8. Price, W. E., Rohrer, C. V., and Jacobson, E. D. Mesenteric vascular disease. *Gastroenterology*, 1969, *57*, 599–604.
9. Rendich, R., and Harrington, L. A. Roentgenologic observations in mesenteric thrombosis. Am. J. Roentgenol. & Rad. Therapy, 1944, *52*, 317–322.
10. Rian, R. L., and Larowe, P. C. Lateral radiographic anatomy of celiac axis and superior mesenteric artery. Personal communication.
11. Stanley, J. C. and Fry, W. J. Medial arcuate ligament syndrome. *A.M.A. Arch. Surg.*, 1971, *103*, 252–258.
12. Wang, C. C., and Reeves, J. D. Mesenteric vascular disease. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1960, *83*, 895–908.

HEREDITARY ANGIONEUROTIC EDEMA OF THE GASTROINTESTINAL TRACT*

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ANGIONEUROTIC edema is a disease characterized by edematous, frequently localized, swellings of the skin, mucous membranes, or viscera. Hereditary angioneurotic edema is by no means rare, but the radiologic literature contains very little information about the entity. Symptoms may start at any age. Visceral manifestations may be the first or the only presenting symptom.[8] Clinically, the patient may be thought to have an acute surgical abdomen. It is, therefore, essential for the radiologist to be aware of the condition and to include it in the differential diagnosis.

We have recently seen 4 patients with hereditary angioneurotic edema at the National Institutes of Health who presented with abdominal symptoms and were examined roentgenographically.

It is the purpose of this communication to present the roentgenographic features of this entity and differentiate it from other conditions involving the gastrointestinal tract.

REPORT OF CASES

Case I. The patient is a 45 year old female who developed episodic swelling of the hands and feet as a teenager. At age 28, for the first time, she experienced severe crampy abdominal pain and underwent an exploratory laparotomy for what was thought to be appendicitis. The appendix was normal, but intraperitoneal fluid containing eosinophils was present. Similar attacks of abdominal pain became more frequent, with episodes occurring at approximately monthly intervals. She was asymptomatic between attacks. At age 43, she had a severe attack and developed acute cardiovascular collapse. When seen at the NIH she was found to have deficiency of the serum inhibitor of C'1 esterase. On June 16, 1969, during an acute abdominal attack, abdominal roentgenograms demonstrated an abnormal small bowel pattern with apparent thickening of the bowel wall. On June 18, 1969, as the attack was subsiding, an upper gastrointestinal series demonstrated thickening of the jejunal valvulae conniventes and some separation of adjacent loops consistent with submucosal edema (Fig. 1, *A* and *B*). Her symptoms subsequently resolved.

Case II. The patient is a 24 year old male who has had recurrent attacks of abdominal pain, often accompanied by nausea, vomiting and malaise, lasting 2 to 5 days and occurring approximately 4 to 6 times yearly since infancy. He also has a history of recurrent swellings, primarily of the hands and feet. These episodes occur about 12 times a year and consist of tingling followed by cool nonerythematous swelling of the affected part with poor demarcation between the affected area and normal skin. There is little or no pain associated and the attacks last 24 to 36 hours. During one episode he developed swelling of the jaw followed by difficult breathing which progressed to complete respiratory obstruction requiring emergency tracheotomy. Upon admission to the NIH, the patient was found to have a marked C'1 esterase inhibitor deficiency consistent with the diagnosis of hereditary angioneurotic edema. On August 13, 1969, during one of his abdominal attacks, an upper gastrointestinal series demonstrated a segment of thickened, edematous duodenum and upper jejunum (Fig. 2, *A–C*). The involved loops were relatively fixed and separated by the edematous submucosa. Spiculation and "thumbprinting" were prominent. The roentgenographic findings were indistinguishable from submucosal hemorrhage or ischemia. However, a repeat examination 36 hours later, at which time the abdominal pain had subsided, demonstrated that the small bowel had returned to normal except for minimal dilatation (Fig. 3). Another attack of severe abdominal pain occurred on

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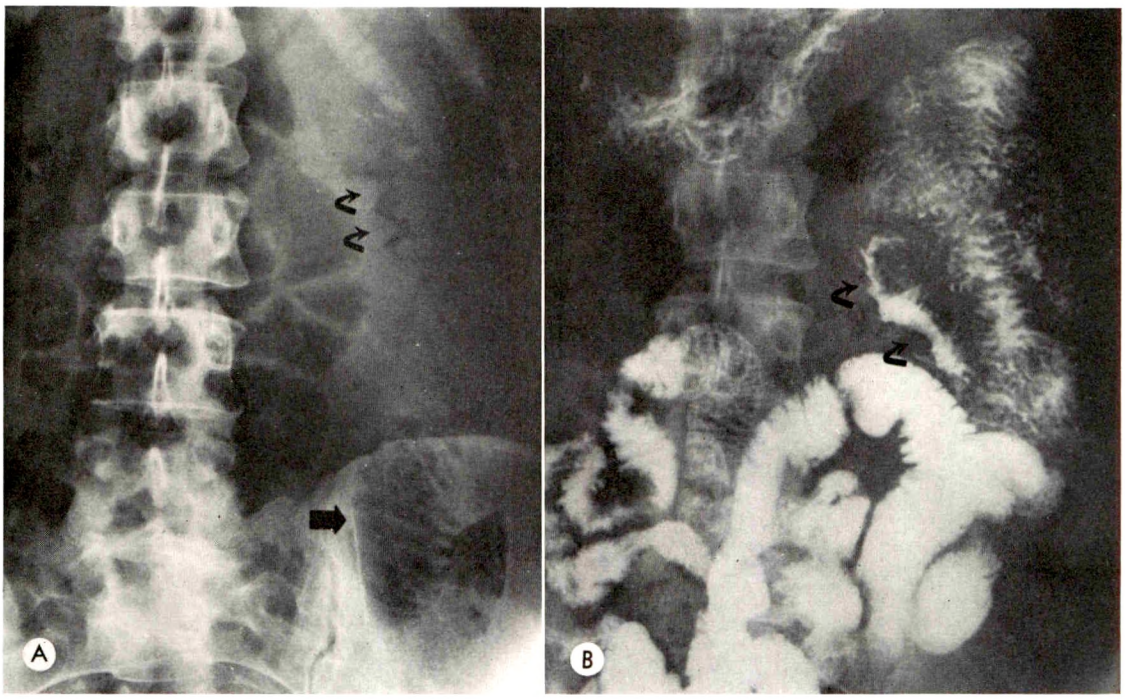


Fig. 1. Case I. (*A*) Supine abdominal roentgenogram demonstrates localized areas of abnormal small bowel, with segmental dilatation and thickening of the mucosal folds (large arrow) and "thumbprinting" (curved arrows). (*B*) Subsequent small bowel barium examination within 48 hours confirms segmental areas of abnormality (curved arrows).

October 28, 1970. An upper gastrointestinal series at that time demonstrated involvement of the distal jejunum and proximal ileum as well as proximal jejunal involvement similar to the previous episode (Fig. 4, *A* and *B*).

Case III. This 33 year old female has a long family history of hereditary angioneurotic edema extending through at least 4 generations. The dominant inheritance of this entity is well demonstrated in her family pedigree. The patient's symptoms began at age 7 and included episodes of vomiting and abdominal pain several times a year. The attacks increased significantly after marriage and consisted of severe crampy abdominal pain associated with protracted vomiting, lasting 12 to 24 hours. The episodes are frequently associated with whitish elevations of the skin surrounded by a red ring. She had attacks of laryngeal edema in 1968 and 1969. Hereditary angioneurotic edema has been documented by demonstration of a deficiency in the inhibitor of C'1 esterase.

A barium examination of the patient's small bowel in May 1971 demonstrated a localized area of abnormal jejunum, demonstrating a "stacked coin" appearance secondary to submucosal edema (Fig. 5). Marked spiculation of the barium column and separation of adjacent loops were present. The remainder of the visualized small bowel appeared normal. A barium enema examination 2 days previously had shown several areas of persistent "thumbprinting" (Fig. 6, *A* and *B*). Subsequent repeat barium examinations in approximately 10 days were normal.

Case IV. First seen at the NIH at age 46, this male has had a history of periodic attacks of edema first occurring at age 12, involving the hand and scrotum. These episodes progressively became more severe and frequent. The symptoms most commonly involved the skin, although he has had respiratory distress and moderately severe abdominal complaints on occasion. He was found to be deficient in C'1 esterase inhibitor. During an episode of mild abdominal discomfort, a barium enema examination demonstrated local submucosal swelling in the ileocecal region. Persistent scalloped defects were present along the medial aspect of the cecum. He underwent 2 upper

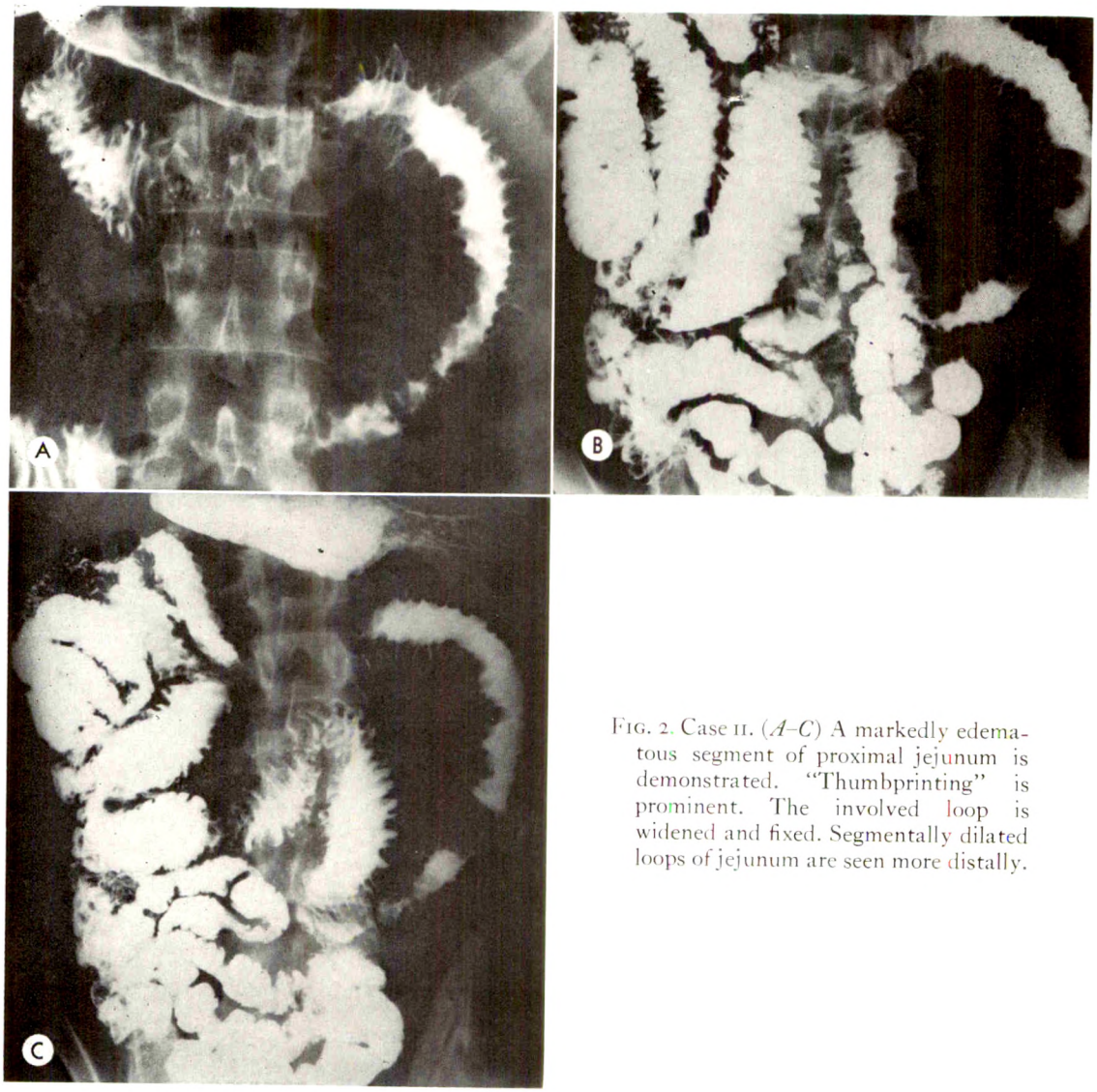


FIG. 2. Case II. (*A–C*) A markedly edematous segment of proximal jejunum is demonstrated. "Thumbprinting" is prominent. The involved loop is widened and fixed. Segmentally dilated loops of jejunum are seen more distally.

gastrointestinal barium examinations during his hospitalization. The first was normal, but there was slight thickening of the mucosal folds with edema in the second portion of the duodenum on the second examination.

DISCUSSION

Hereditary angioneurotic edema is inherited as a mendelian dominant trait with irregular penetrance. Initial onset may appear at any age. The clinical attacks are periodic and occur at irregular intervals. Swelling appears spontaneously and may involve any area of the body. Visceral manifestations are common and present as abdominal pain which may be severe and may be accompanied by nausea or vomiting. The most serious complication is that of laryngeal edema, which carries with it a very significant mortality rate. Crises may be precipitated by emotional stress or physical trauma.[4,8]

Patients with hereditary angioneurotic edema are now known to have deficiency of the serum inhibitor of C'1 esterase. However, individuals with the biochemical defect may not exhibit the trait clinically.[4] Recently a number of assay procedures

have been described which should facilitate diagnosis.[15,16]

The illustrations included are the most representative examples of the roentgenographic pathology exhibited by 4 patients examined during abdominal attacks. The findings consist of thickened mucosal folds in the small bowel with a "stacked coin" appearance, spiculation, "thumbprinting" of involved areas, and separation of adjacent loops of bowel as a result of mural and mesenteric thickening.[6] Involvement is segmental and may be in the stomach, small bowel or colon. These findings are present only when the patient is in visceral crisis and rapidly revert to normal as the attack subsides. This characteristic feature is important in differentiating hereditary angioneurotic edema from the numerous other causes of thickened bowel wall and prominent mucosal folds.

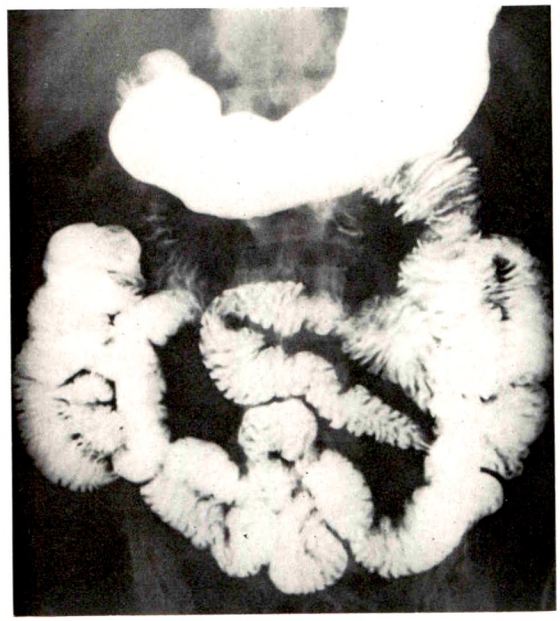


Fig. 3. Case II. Repeat small bowel barium examination 36 hours later demonstrates a dramatic return to a normal bowel pattern.

The most difficult differential diagnosis from a purely roentgenographic standpoint is that of vascular disease, especially segmental ischemic involvement or incomplete infarction. However, intramural intestinal bleeding from other causes such as excessive anticoagulant therapy, Henoch-Schoenlein purpura, a bleeding diathesis, or trauma can be equally difficult to distinguish.[11] Unlike hereditary angioneurotic edema, these patients frequently exhibit associated gastrointestinal bleeding. They do not have

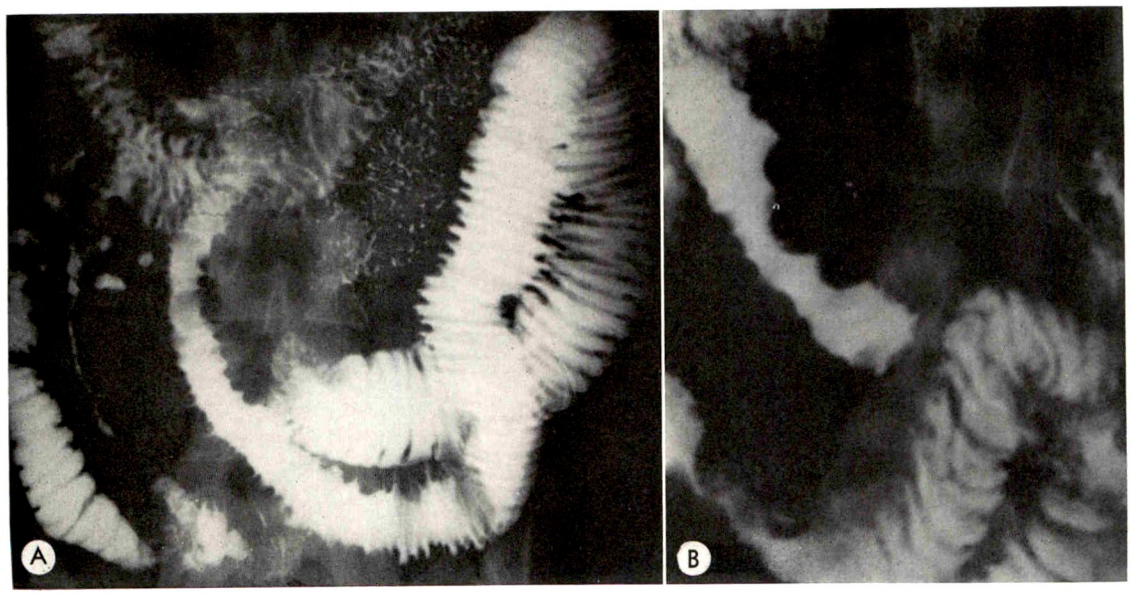


Fig. 4. Case II. (*A*) Thickened valvulae conniventes developed in a portion of distal jejunum during a subsequent attack. Areas of "thumbprinting" are present in this area. (*B*) Several isolated segments of distal ileum show fixation and significant "thumbprinting" from the edema.



FIG. 5. Case III. Thickened valvulae conniventes and intramural edema are present in a localized segment of distal jejunum. The patient exhibited tenderness to palpation localized in this area. The remainder of the small bowel is normal.

the characteristic family history or a history of previous angioneurotic reactions. Of most importance is the rapid and complete reversibility of the gastrointestinal roentgenographic findings in angioneurotic edema.

Generalized small bowel edema from hypoproteinemia, intestinal lymphangiectasia and the protein losing enteropathies tends to be more diffuse and does not exhibit the localized, segmental nature of hereditary angioneurotic edema. "Thumbprinting" is not usually associated with these entities. Other diseases that must be considered in the thickened fold gamut include regional enteritis, Whipple's disease, gastrointestinal amyloidosis, radiation enteritis, lymphoma, macroglobulinemia and mastocytosis.

Severe pain in the absence of a rigid abdomen, a history of recurring attacks, a family history of similar episodes and laboratory data should help the radiologist with the diagnosis.

SUMMARY

Four patients with hereditary angioneurotic edema who presented with ab-

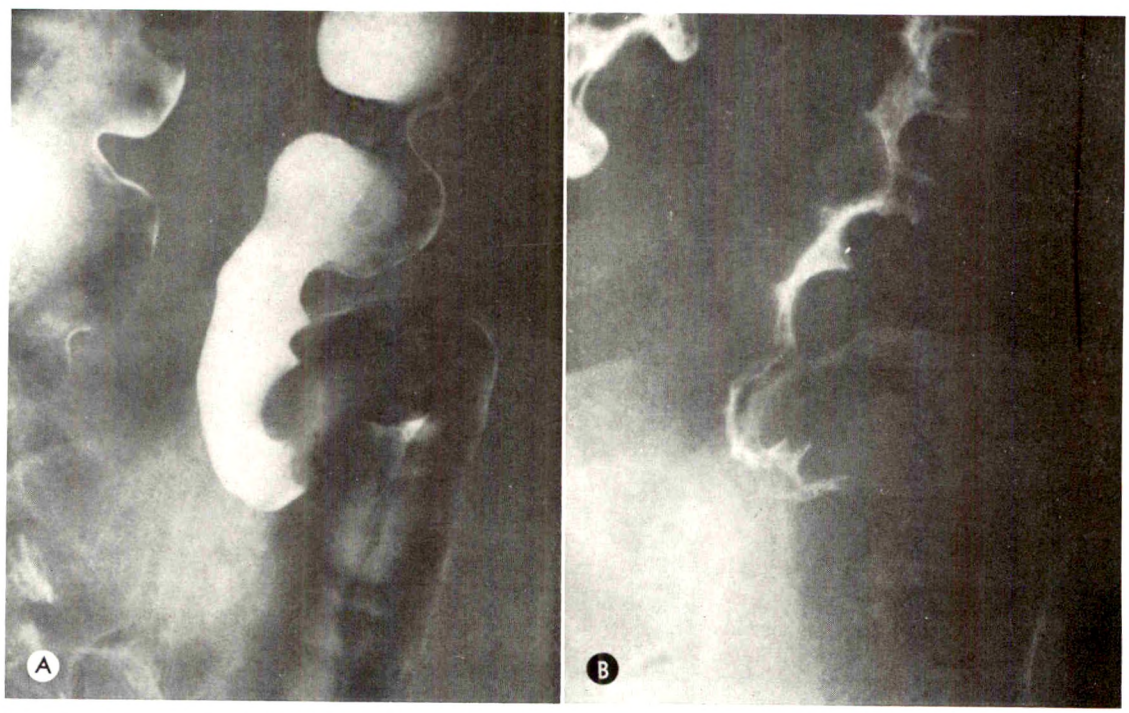


FIG. 6. Case III. (*A* and *B*) Persistent, localized "thumbprinting" is demonstrated in the descending colon. The remainder of the colon appears within normal limits.

dominal symptoms and were examined roentgenographically, at the National Institutes of Health, are reported.

The roentgenographic features of this entity are described and differentiation from other conditions involving the gastrointestinal tract is discussed.

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REFERENCES

1. Austen, K. F., and Sheffer, A. L. Detection of angioneurotic edema by demonstration of reduction in second component of human complement. *New England J. Med.*, 1965, *272*, 649–656.
2. Becker, E. L., and Kagen, L. Permeability globulins of human serum and biochemical mechanism of hereditary angioneurotic edema. *Ann. New York Acad. Sc.*, 1964, *116*, 866–873
3. Biering, A. Abdominal pains in angioneurotic edema *Acta med. scandinav.*, 1956, *153*, 373–382.
4. Dennehy, J J. Hereditary angioneurotic edema. *Ann. Int. Med.*, 1970, *73*, 55–59.
5. Donaldson, V. H., and Rosen, F. Hereditary angioneurotic edema: clinical survey. *Pediatrics*, 1966, *37*, 1017–1027.
6. Ellis, K., and McConnell, D. J. Hereditary angioneurotic edema involving small intestine. *Radiology*, 1969, *92*, 518–519.
7. Johnson, T. H., and Caldwell, K. W. Angioneurotic edema of colon. *Radiology*, 1971, *99*, 61–63.
8. Landerman, N. S. Hereditary angioneurotic edema: case reports and review of literature. *J. Allergy*, 1962, *33*, 316–329.
9. Love, R. J. Gastrointestinal crises of angioneurotic edema. *Brit. J. Surg.*, 1932, *20*, 170–172.
10. Marshak, R. H., Khilnani, M., Eliasoph, J., and Wolf, B. S. Intestinal edema. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *101*, 379–387.
11. Marshak, R. H., and Lindner, A. E. Radiology of Small Intestine. W. B. Saunders Company, Philadelphia, 1970.
12. Osler, W. Hereditary angioneurotic edema. *Am. J. Med. Sc.*, 1888, *95*, 362–367.
13. Reimann, H. A. Hereditary periodic edema: interrelation of familial periodic disorders. *Am. J. Med. Sc.*, 1962, *243*, 727–739.
14. Renton, C. J. Recurrent oedema of small intestine. *Brit. J. Surg.*, 1965, *52*, 536–539.
15. Ruddy, S., and Austen, K. F. Stoichiometric assay for fourth component of complement in whole human serum using EAC'la^{GP} and functionally pure human second component. *J. Immunol.*, 1967, *99*, 1162.
16. Sun-Chik Fong, J., Good, R. A., and Gewurz, H. Simple diagnostic test for hereditary angioneurotic edema. *J. Lab. Clin. Med.*, 1970, *76*, 836–844.
17. Thompson, J. S. Urticaria of angioedema. *Ann. Int. Med.*, 1968, *69*, 361–380.
18. Trigg, J. W. Hereditary angioneurotic edema: report of case with gastrointestinal manifestations. *New England J. Med.*, 1961, *264*, 761–763.
19. Watson, I. M. Angioneurotic edema. *J.A.M.A.*, 1926, *86*, 1332–1333.

FUNCTIONAL EVALUATION OF SMALL BOWEL OBSTRUCTION BY SUCCESSIVE ABDOMINAL ROENTGENOGRAMS

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SMALL bowel obstruction classically produces distention of the intestine proximal to the site of obstruction. The distended bowel usually contains both fluid and gas, but at times may be predominantly gas-filled or fluid-filled.

The appearance of this distended bowel on the recumbent and erect abdominal roentgenograms has been used to evaluate the presence of a simple obstruction or one complicated by volvulus, strangulation, perforation, or peritoneal inflammation. According to Frimann-Dahl,[6] U-shaped loops in the erect roentgenogram with short fluid levels of different heights in the same loop are an indication of a simple dynamic obstruction with increased muscular activity proximal to a mechanical block, while long fluid levels and straight flaccid loops are a manifestation of decreased bowel activity which occurs with strangulating obstructions, mesenteric vascular thrombosis and peritonitis. In long-standing simple mechanical bowel obstruction, however, long fluid levels may be seen and probably denote exhaustion of bowel muscle. Mellins and Rigler[9] in their study of strangulating obstruction include long fluid levels in their group of signs which are less frequent in occurrence and less specific in character. They state that these signs may suggest the possibility of strangulation and should initiate a search for the more specific signs that they describe, such as the "coffee-bean sign" or the "pseudotumor sign." Lasser[7] states that in his experience he has been unable to utilize effectively the length of the air fluid levels or the presence of levels at the same heights or different heights within the same loop in differentiating mechanical obstruction from paralytic ileus.

It is generally accepted that in association with bowel vascular compromise, either due to strangulation or vascular occlusion, there is considerable inhibition of bowel activity. This has been demonstrated experimentally in rabbits by Frimann-Dahl[5] and in dogs by Dixon *et al.*[2] This inhibition of bowel activity has been evaluated in patients by various techniques. Frimann-Dahl[6] advocates fluoroscopy of the abdomen in the erect position to evaluate the changes in the air fluid levels as a result of bowel activity. Nelson and Christoforidis[10] recommend the administration of oral barium sulfate suspensions to demonstrate the small bowel abnormality. They state that the presence of peristaltic activity in or proximal to dilated loops of small intestine is important because it almost certainly means that the dilated loops are due to a mechanical obstruction. They contend that peristaltic activity can be recognized with ease in the dilated barium-filled loops in contrast to the invisibility of peristalsis in air containing loops of small intestine. They further state that although clearly recognizable peristaltic activity in dilated loops of small intestine indicates some mechanical obstruction, it is usually not possible to determine if hypotonic or hypomotile small intestine is due to adynamic ileus or whether it is due to the fatigued adynamic state which often occurs proximal to a simple nonstrangulating mechanical obstruction, which has been present for several days. In strangulating obstruction, both Nelson and Christoforidis[10] and Dixon *et al.*[3,4] advocate

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administration of oral barium sulfate suspensions, since there is atonicity of the stomach and small intestine with retention of barium, while in simple mechanical obstruction the barium passes to the obstructed segment. A similar conclusion was reached by Vest[11] using water soluble contrast material. He noted prolonged retention of the contrast substance in the stomach with failure or marked delay of opacification of the small bowel in cases with strangulating closed loop obstruction. Dixon *et al.*[2] have suggested that this inhibition of activity is probably the result of sympathoadrenal discharge resulting in activation of inhibitory alpha and beta adrenergic receptors in the intestine.

Recently, Bryk and Rosenkranz[1] reported another method of evaluating intestinal motility in a patient with an acute abdomen, the technique of successive abdominal roentgenograms. In this method, 2 supine abdominal roentgenograms are made at 5 minute intervals with no change in patient position and in the same phase of respiration. Intestinal activity is evaluated by visual comparison of the size, shape, orientation and distribution of the gas and fluid in the intestinal loops. This is best accomplished by dividing the abdomen into multiple quadrants and comparing these one at a time.

The 5 minute interval used in this study was chosen somewhat arbitrarily. In auscultation of the abdomen, it is generally agreed that a prolonged evaluation is necessary to gain an impression of the intestinal sounds. Usually, a period of from 2–5 minutes is adequate. Five minutes, therefore, appeared to be a reasonable interval which did not excessively prolong the examination for the patient. It was extremely important that the patient remain in the same position throughout the 5 minute period, since patient movement can alter the position of the small bowel loops, thus causing changes not due to peristaltic activity.

One hundred patients with small bowel obstruction were studied with this technique.

The purpose of this report is to evaluate its utility in providing functional information on the state of the small bowel, especially from the standpoint of differentiating cases of simple obstruction from those with complicated obstruction. The results of this technique are also compared to the functional information provided by the other signs seen on the plain roentgenogram, previously mentioned as being useful.

METHOD

All the cases of small bowel obstruction included in this study were examined by 2 supine successive abdominal roentgenograms exposed at 5 minute intervals and one erect or decubitus study. The diagnosis of small bowel obstruction was either proven at laparotomy or was the final clinical diagnosis in those who were treated conservatively with improvement.

The cases were divided into 4 groups based on the degree of activity on the successive roentgenograms and on the appearance of the fluid levels and the dilated loops in the erect roentgenograms.

The following changes in the small bowel loops were noted as indications of activity on the successive roentgenograms: (1) fluid gas shifts with opacification of the gas-filled lumen by replacement with fluid or replacement of the fluid-filled lumen by gas; (2) fluid gas shifts with changes in the distribution and configuration of the floating bubbles in the fluid-filled loops; (3) constrictions in the contours of the gas-filled loops; and (4) changes in the arrangement and position of the loops.

Small bowel activity was graded as 1+ to 4+ using the following criteria for grading the degree of activity:

1+ Activity.

(a) Minimal fluid gas shifts within loops with minimal changes in the configuration or arrangement of the floating gas bubbles in the fluid-filled loops or minimal shifts of gas and fluid at the periphery of the gas-filled loops (Fig. 1, *A* and *B*).

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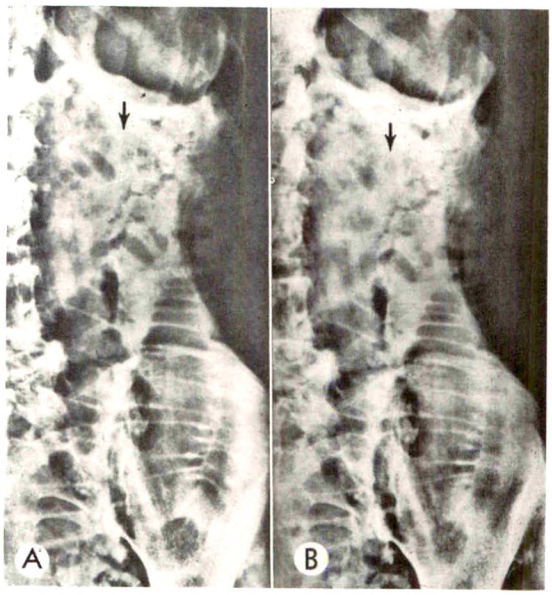


Fig. 1. (*A* and *B*) Small bowel obstruction due to an incarcerated inguinal hernia with a congested hemorrhagic loop. Supine successive roentgenograms: 1+ activity. The dilated fluid and gas-filled loops show no significant change except for minimal alterations in the configuration and arrangement of the gas bubbles in the fluid-filled loops (arrows).

(b) No constrictions in the contours of the gas-filled loops.
(c) No change in the arrangement or position of the loops.

2+ Activity.

(a) Moderate fluid gas shifts within loops with considerable change in distribution or configuration of the floating bubbles in the fluid-filled loops (Fig. 2, *A* and *B*) and moderate shifts of gas and fluid at the periphery of the gas-filled loops (Fig. 3, *A* and *B*).
(b) No constrictions in the contours of the gas-filled loops.
(c) No change in the arrangement or position of the loops.

3+ Activity.

(a) Constrictions in the contours of the gas-filled loops (Fig. 4, *A* and *B*), or
(b) Marked gas fluid shifts within loops with complete or almost complete opacification of the major portion of the gas-filled lumen by replacement with fluid (Fig. 5, *A* and *B*).
(c) No change in the arrangement or position of the loops.

4+ Activity.

Changes in loop arrangement and position (Fig. 6, *A* and *B*).

Cases were also classified by the roentgen appearance of the dilated loops and the fluid levels in the erect or decubitus roentgenograms. The cases were thus divided into 4 groups:

Group 1.

(a) Short fluid levels (*i.e.*, the maximum length of the levels is no more than 1.5 cm. longer than the maximum diameter of the loops).
(b) U-shaped loops.
(c) Activity 2+ to 4+ (Fig. 7, *A–C*).

Group 2.

(a) Long fluid levels (*i.e.*, the maximum length of the levels is 2 cm. or more

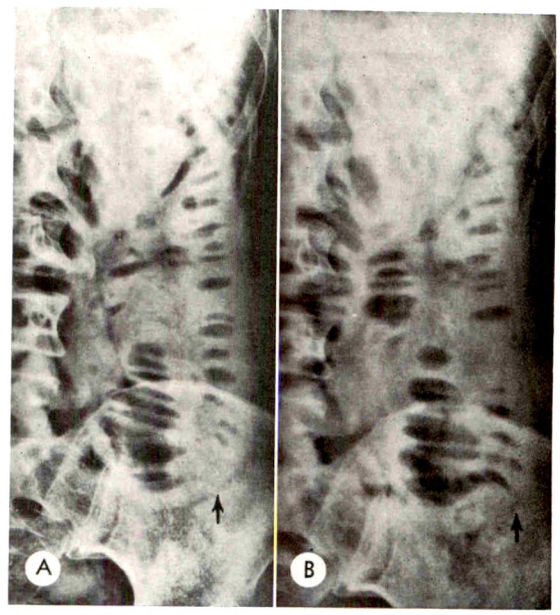


Fig. 2. (*A* and *B*) Small bowel obstruction due to adhesions. Supine successive roentgenograms: 2+ activity. The dilated loops are predominantly fluid-filled and contain multiple floating gas bubbles. Note the considerable changes in the distribution and configuration of these floating bubbles (arrows).

longer than the maximum diameter of the loops).

(b) Flaccid loops.

(c) Activity 2+ to 4+ (Fig. 8, *A–C*).

Group 3.

Same as Group 1, except activity 1+ (Fig. 9, *A–C*).

Group 4.

Same as Group 2, except activity 1+ (Fig. 10, *A–C*).

The criterion of differential heights of the fluid levels was not used, because it appeared difficult to evaluate in cases with multiple dilated and overlapping loops whether the 2 fluid levels of different heights or of the same heights were in the same or different loops.

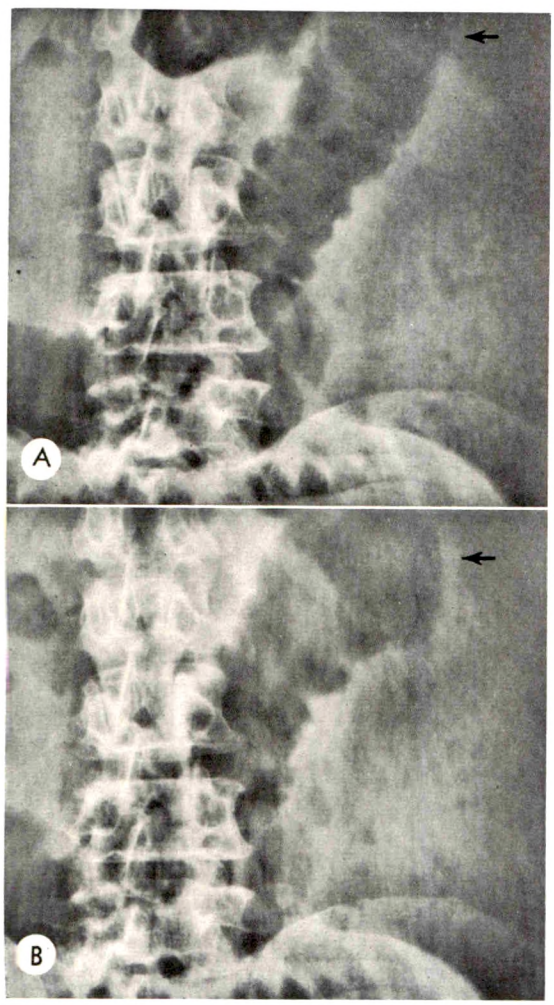


Fig. 3. (*A* and *B*) Small bowel obstruction due to adhesions. Supine successive roentgenograms: 2+ activity. Note the change at the periphery of the gas-filled loop due to a shift of gas into the fluid-filled portion of the loop in *B* (arrows).

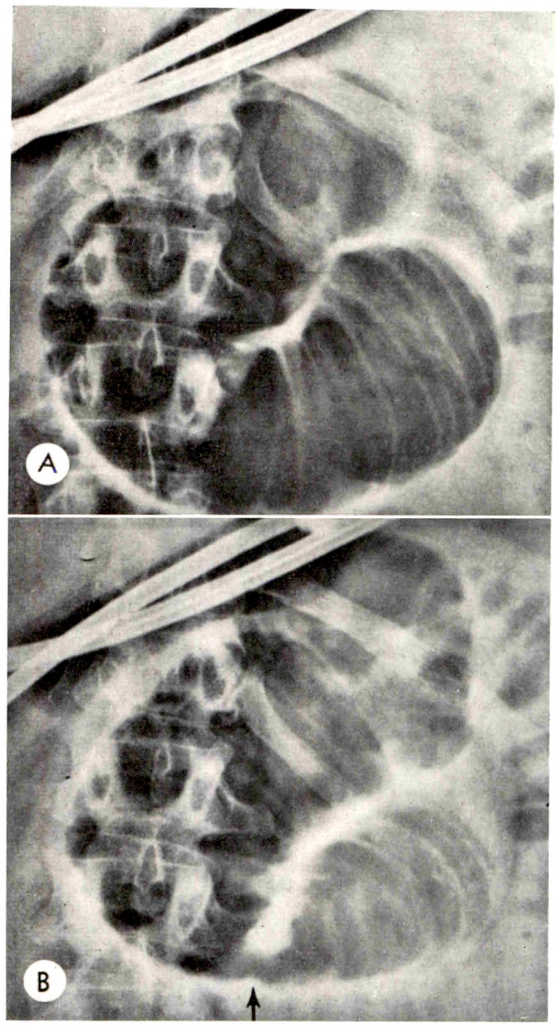


Fig. 4. (*A* and *B*) Small bowel obstruction due to an incarcerated inguinal hernia. Supine successive roentgenograms: 3+ activity. Note the marked constriction in the lower portion of the gas-filled loop (arrow).

These 4 groups were then compared from the standpoint of the findings at laparotomy and the incidence of improvement on nonsurgical therapy.

Comparisons were also made of the distribution of the degrees of activity in Group 1 *versus* Group 2. In addition, in an attempt to evaluate whether the size of the dilated loops was a factor in activity, the degree of activity was compared with the

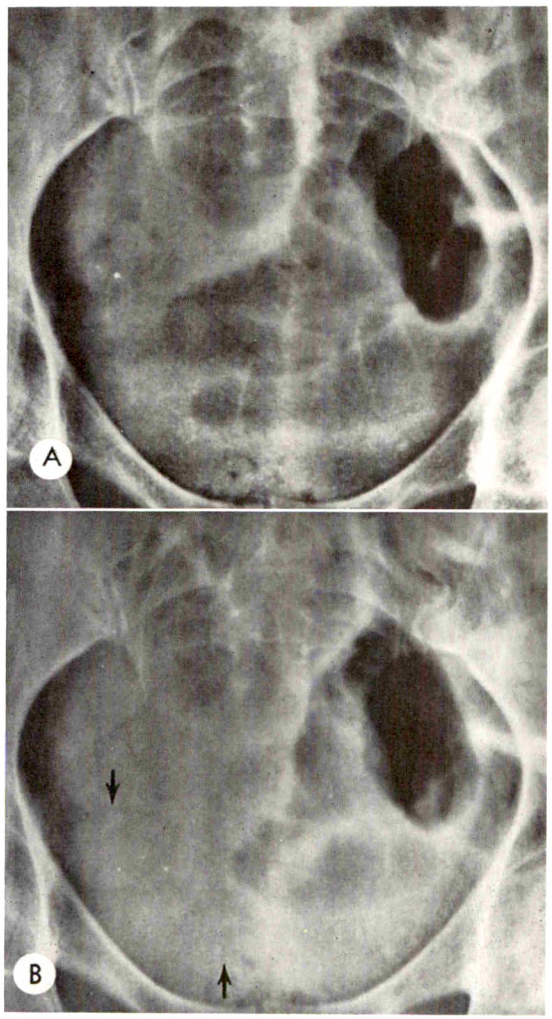


Fig. 5. (*A* and *B*) Small bowel obstruction due to adhesions. Supine successive roentgenograms: 3+ activity. Note the opacification in *B* of the previously gas-filled loops in the right side of the pelvic cavity by a marked gas-fluid shift (arrows).

maximum size of the bowel loops as measured in the supine roentgenogram, and similarly the maximum size of the bowel loops was compared with the degree of activity. In those cases with multiple successive roentgenographic studies, the consistency or variation in activity between studies was also determined. Since the level of serum potassium has been reported to be a factor in intestinal activity,[8] this value was correlated with the intestinal activity, the fluid levels and the type of obstruction.

It must be noted that on the surgical service at the Jewish Hospital and Medical Center of Brooklyn, the policy is to explore patients with small bowel obstruction. The cases that improved on medical management were generally those who showed rapid improvement on upper gastrointestinal drainage, while the patient was being medically prepared for surgery, or where there were other medical indications that caused the surgeon to attempt nonoperative decompression.

RESULTS

Table 1 tabulates the causes of obstruction and associated complications in the 4

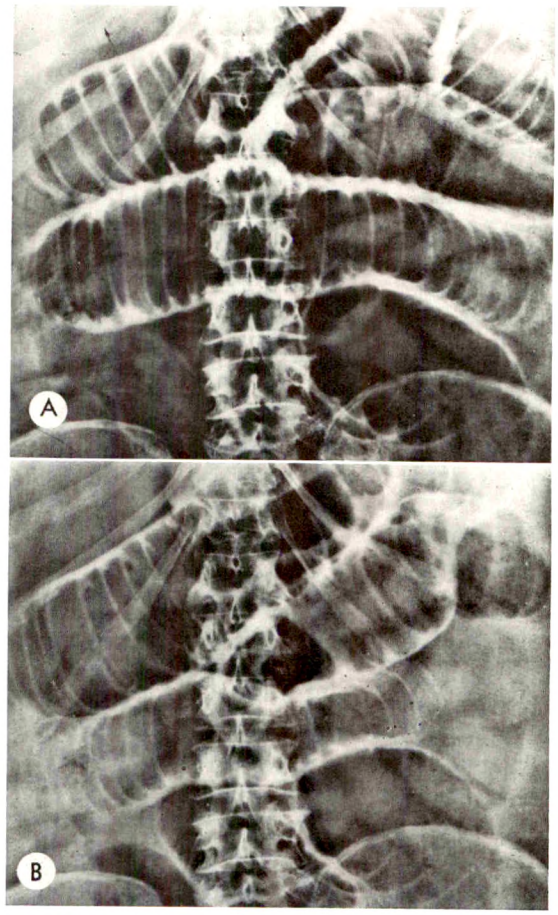


Fig. 6. (*A* and *B*) Small bowel obstruction due to carcinomatosis. Supine successive roentgenograms: 4+ activity. There is free air in the peritoneal cavity, introduced during abdominal paracentesis, outlining the outer wall of a number of the loops. Note the marked changes in loop arrangement and position.

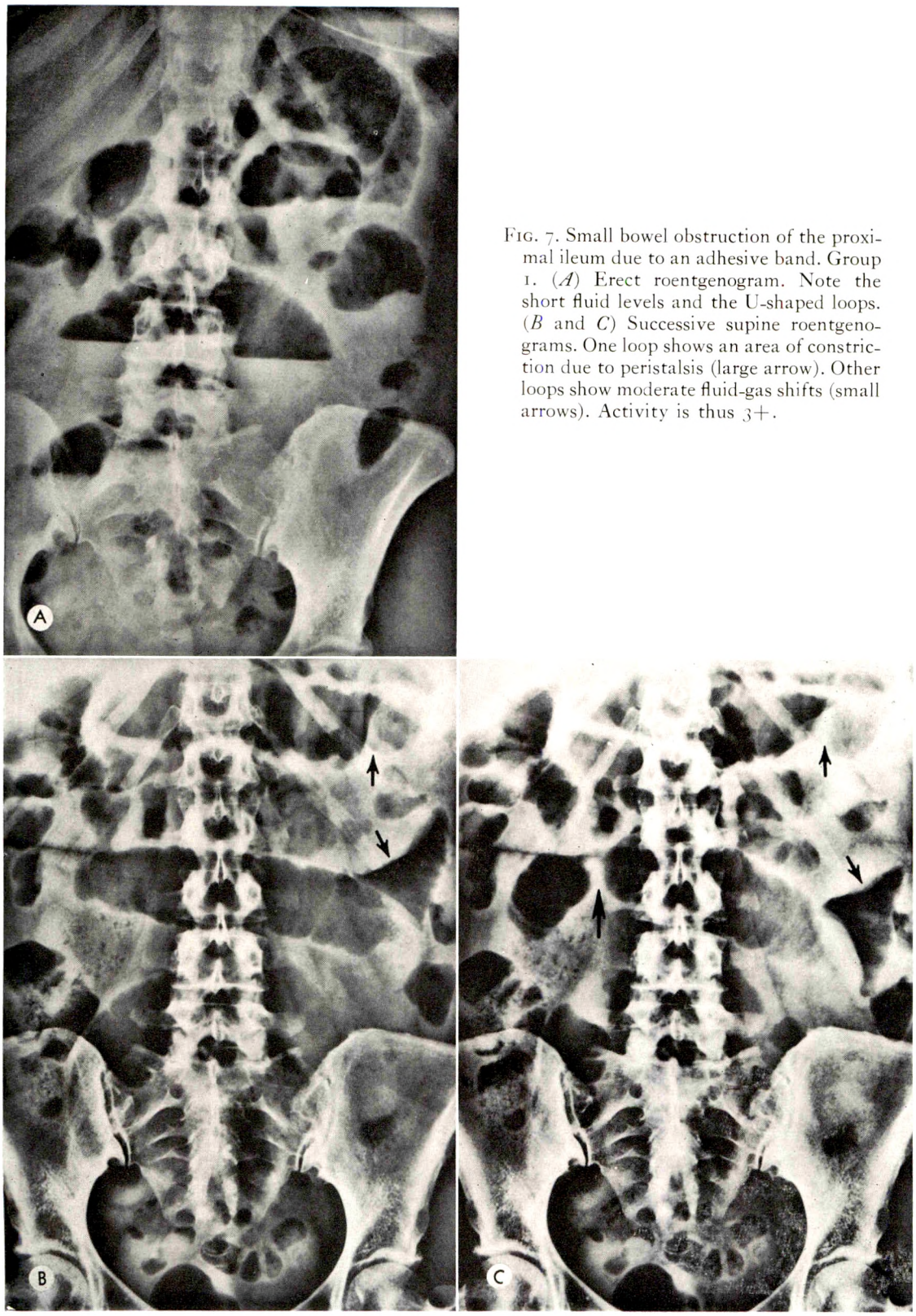


Fig. 7. Small bowel obstruction of the proximal ileum due to an adhesive band. Group 1. (*A*) Erect roentgenogram. Note the short fluid levels and the U-shaped loops. (*B* and *C*) Successive supine roentgenograms. One loop shows an area of constriction due to peristalsis (large arrow). Other loops show moderate fluid-gas shifts (small arrows). Activity is thus 3+.

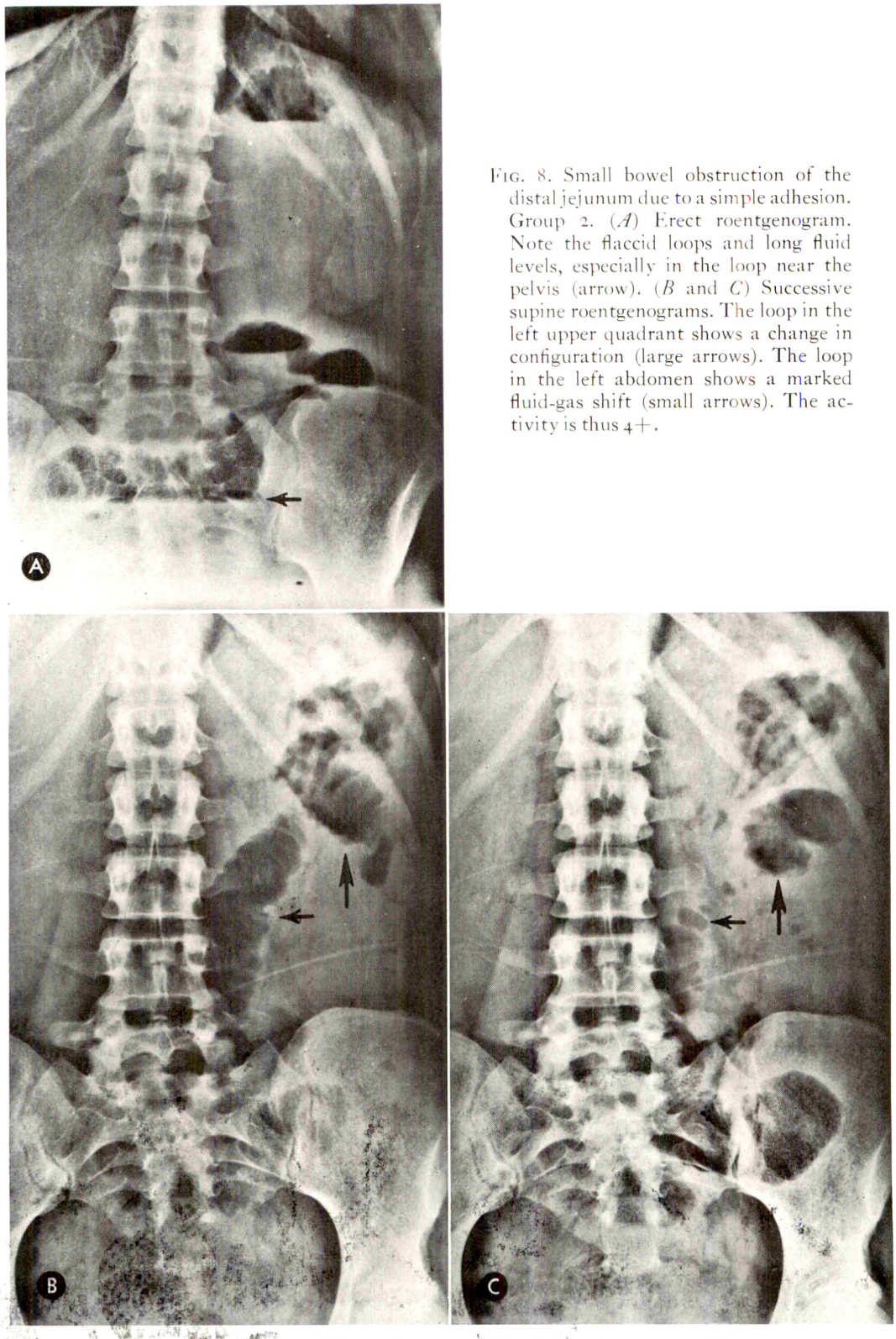


Fig. 8. Small bowel obstruction of the distal jejunum due to a simple adhesion. Group 2. (*A*) Erect roentgenogram. Note the flaccid loops and long fluid levels, especially in the loop near the pelvis (arrow). (*B* and *C*) Successive supine roentgenograms. The loop in the left upper quadrant shows a change in configuration (large arrows). The loop in the left abdomen shows a marked fluid-gas shift (small arrows). The activity is thus 4+.

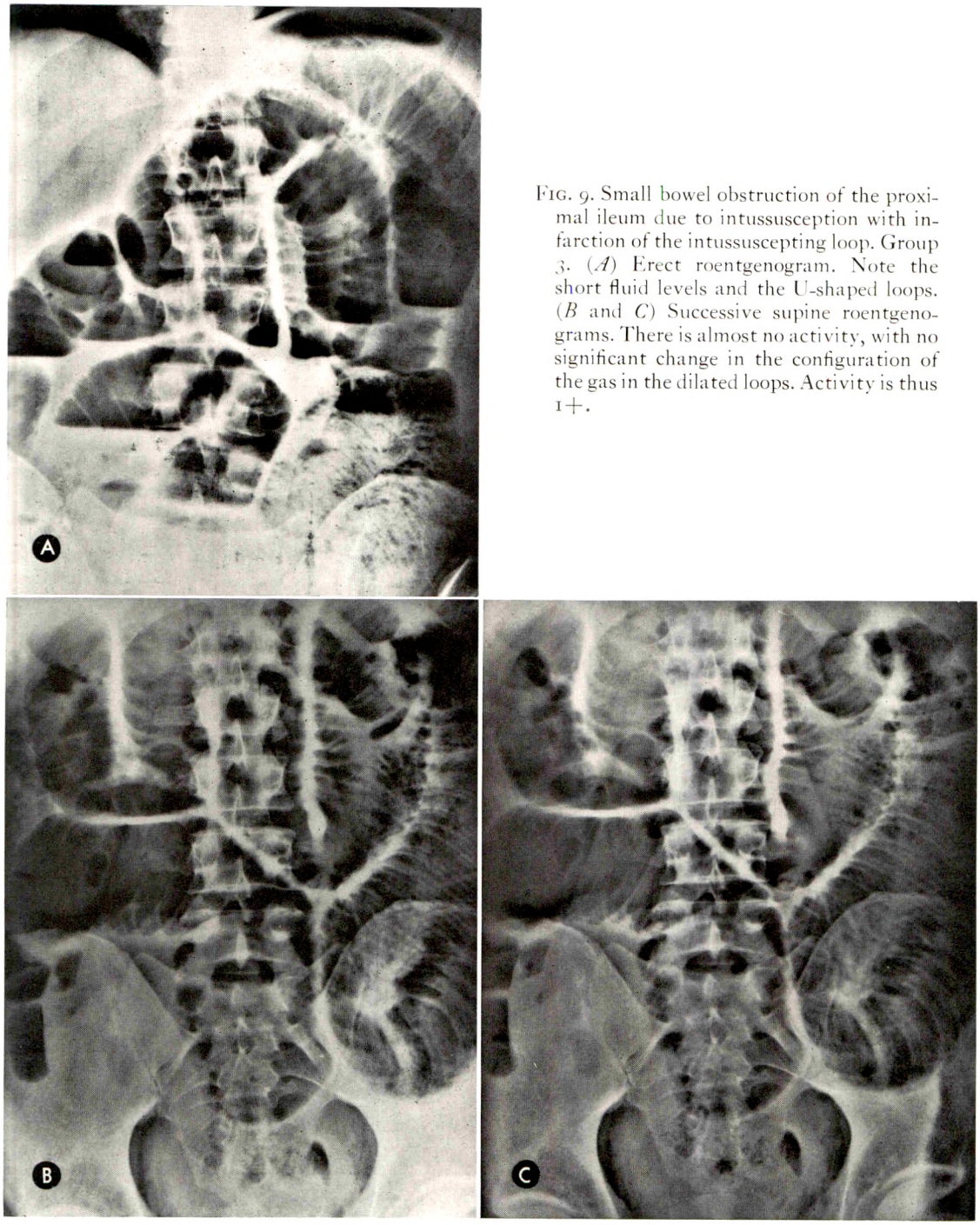


FIG. 9. Small bowel obstruction of the proximal ileum due to intussusception with infarction of the intussuscepting loop. Group 3. (*A*) Erect roentgenogram. Note the short fluid levels and the U-shaped loops. (*B* and *C*) Successive supine roentgenograms. There is almost no activity, with no significant change in the configuration of the gas in the dilated loops. Activity is thus 1+.

groups. There were 50 cases in Group 1, 25 cases in Group 2, 16 cases in Group 3, and 9 cases in Group 4. Table II compares Groups 1 and 3 combined, with Groups 2 and 4 combined; that is, cases showing U-shaped loops and short fluid levels with cases showing flaccid loops and long levels. The figures indicate no significant difference in the incidence of simple *versus* complicated obstruction or in the incidence of improvement on medical management. Table III compares Groups 1 and 2 com-

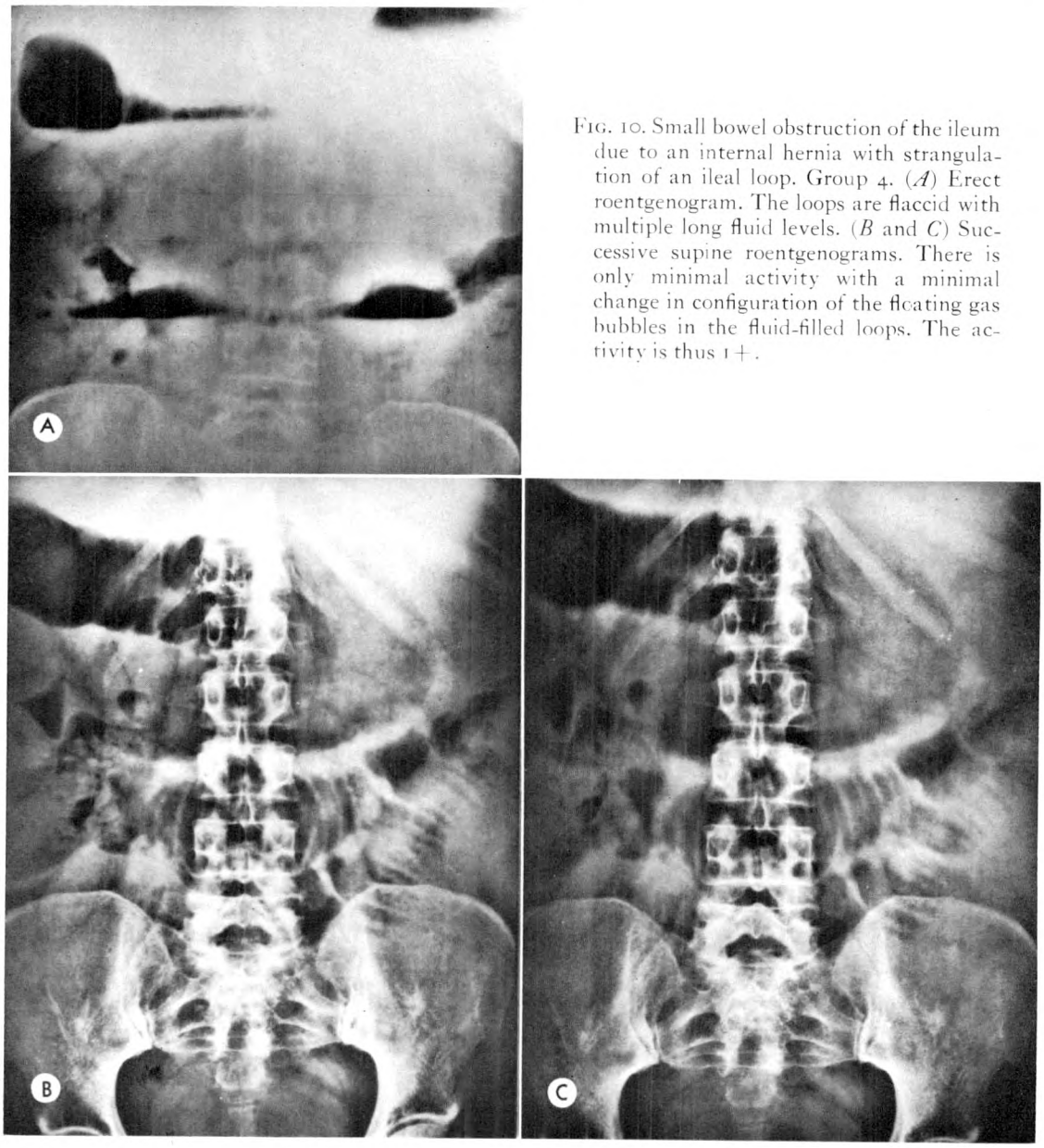


Fig. 10. Small bowel obstruction of the ileum due to an internal hernia with strangulation of an ileal loop. Group 4. (*A*) Erect roentgenogram. The loops are flaccid with multiple long fluid levels. (*B* and *C*) Successive supine roentgenograms. There is only minimal activity with a minimal change in configuration of the floating gas bubbles in the fluid-filled loops. The activity is thus 1+.

bined, with Groups 3 and 4 combined; that is, cases with normal or increased activity *versus* those with reduced activity. It shows that, whereas the incidence of complicated obstruction (*i.e.*, strangulated bowel, volvulus, and/or congested hemorrhagic loop and intraperitoneal abscess) was 72 per cent in Groups 3 and 4, it was only 8 per cent in Groups 1 and 2. Using the chi square test, these data are statistically significant at the 0.010 level. Similarly, it shows that while 22 per cent of the cases in Groups 1 and 2 improved on medical management, none of those in Groups 3 and 4 showed such improvement. Using the chi square test, these data are statistically significant at the 0.025 level.

Table IV summarizes the degree of activity noted in those that improved on medical management. It shows that except for 1+ activity, there was no significant difference in the incidence of improvement in the other activity categories.

Similarly, a comparison of the degrees of

TABLE I

CAUSES OF OBSTRUCTION AND ASSOCIATED COMPLICATIONS
GROUPS 1, 2, 3, AND 4

Cause of Obstruction and Associated Complications	Group 1	Group 2	Group 3	Group 4
Strangulated bowel	1	0	6	3
Volvulus and/or congested hemorrhagic loop	2	1	3	4
Intraperitoneal abscess	1	1	2	0
Incarcerated hernia	5	1	2	1
Carcinomatosis	7	2	1	1
Simple adhesive bands	21	10	2	0
Miscellaneous causes of simple obstruction	4	3	0	0
Improvement on medical treatment	9	7	0	0
Total	50	25	16	9

activity seen in Groups 1 and 2 (Table v) shows no significant difference in activity between these 2 groups, with a similar distribution of degrees of activity noted in those cases with short fluid levels and U-shaped loops and those with long fluid levels and so-called flaccid loops. Thus, the only degree of activity which appeared significant was 1+ or reduced activity, while once significant activity was present, its degree was of no importance.

Tables VI and VII evaluate whether the

TABLE II

CAUSES OF OBSTRUCTION AND ASSOCIATED COMPLICATIONS
GROUPS 1 AND 3 VERSUS GROUPS 2 AND 4

Cause of Obstruction and Associated Complications	Total Groups 1 and 3		Total Groups 2 and 4	
	No. of Cases	Per Cent	No. of Cases	Per Cent
Strangulated bowel	7	11	3	9
Volvulus and/or congested hemorrhagic loop	5	7	5	15
Intraperitoneal abscess	3	5	1	3
Incarcerated hernia	7	11	2	6
Carcinomatosis	8	12	3	9
Simple adhesive bands	23	34	10	29
Miscellaneous causes of simple obstruction	4	6	3	9
Improvement on medical treatment	9	14	7	20
Total	66	100	34	100

TABLE III

CAUSES OF OBSTRUCTION AND ASSOCIATED COMPLICATIONS
GROUPS 1 AND 2 VERSUS GROUPS 3 AND 4

Cause of Obstruction and Associated Complications	Total Groups 1 and 2		Total Groups 3 and 4	
	No. of Cases	Per Cent	No. of Cases	Per Cent
Strangulated bowel	1	1	9	36
Volvulus and/or congested hemorrhagic loop	3	4	7	28
Intraperitoneal abscess	2	3	2	8
Incarcerated hernia	6	8	3	12
Carcinomatosis	9	12	2	8
Simple adhesive bands	31	41	2	8
Miscellaneous causes of simple obstruction	7	9	0	0
Improvement on medical treatment	16	22	0	0
Total	75	100	25	100

size of the dilated loops of bowel was in any way a factor in the degree of activity noted. There appears to be a slightly higher incidence of 1+ activity in cases with loops 6.5 to 8 cm. in maximum diameter, but because of the number of cases studied, the figures are not statistically significant.

Eighteen cases had multiple studies, 15 had 2 studies and 3 had 3 studies. In 6 cases there were differences in degree of activity comparing the studies in the same patient, but this was only in those with 2+ to 4+ activity.

The correlation of the level of the serum potassium with the criteria evaluated in this study was poor. There was a slight difference in the incidence of low serum potassium (3.5 mEq./l. or less) comparing the cases of 1+ activity with those of 2+ to 4+ activity. Thirty-eight per cent of the cases with 1+ activity had a low serum potassium at the time of the roentgenographic study, as compared to 25 per cent of those with 2+ to 4+ activity. Comparing cases with simple obstruction and those with complicated obstruction, the incidence of low serum potassium was 27 per cent *versus* 33 per cent. Comparing cases with short fluid levels and those with long fluid levels, the incidence was 30 per cent *versus* 27 per cent. These figures indicate no correlation using these parameters.

DISCUSSION

This report indicates that the length of the fluid levels and the shape of the small

TABLE IV

IMPROVEMENT ON MEDICAL TREATMENT
VERSUS DEGREE OF ACTIVITY

	Degree of Activity			
	1+	2+	3+	4+
Number of cases improved on medical treatment	0	4	5	7
Total number of cases with this degree of activity	25	17	30	28
Per cent improved	0	24	17	25

Table V

DEGREE OF BOWEL ACTIVITY, GROUP 1 VERSUS GROUP 2

Group	Degree of Activity						Total
	2+		3+		4+		
	No. of Cases	Per Cent	No. of Cases	Per Cent	No. of Cases	Per Cent	
Group 1	10	20	21	42	19	38	50
Group 2	7	28	9	36	9	36	25

Table VI

DEGREE OF ACTIVITY VERSUS MAXIMUM SIZE OF BOWEL LOOPS

Degree of Activity	Size of Loops								Total
	3.0–4.5 cm.	Per Cent	5.0–6.5 cm.	Per Cent	6.5–8.0 cm.	Per Cent	Above 8.0 cm.	Per Cent	
1+	8	32	11	44	6	24	0	0	25
2+	9	53	5	30	3	17	0	0	17
3+	15	50	13	44	1	4	1	3	30
4+	10	36	14	50	3	11	1	3	28

Table VII

MAXIMUM SIZE OF BOWEL LOOPS VERSUS DEGREE OF ACTIVITY

Size of Loops	Degree of Activity								Total
	1+	Per Cent	2+	Per Cent	3+	Per Cent	4+	Per Cent	
3.0–4.5 cm.	8	19	9	21	15	36	10	24	42
5.0–6.5 cm.	11	26	5	11	13	30	14	33	43
6.5–8.0 cm.	6	46	3	23	1	8	3	23	13
Above 8.0 cm.	0	0	0	0	1	50	1	50	2

bowel loops on roentgenograms in the erect position are not reliable in evaluating the functional state of the bowel. The length of the fluid levels may be related to the amount of fluid in the dilated loops rather than to their state of activity. It was noted that the dilated predominantly fluid-filled distal jejunal and ileal loops usually showed long fluid levels, while the predominantly gas-filled jejunal loops showed short levels. The reasons for the presence of predominant gaseous distention of loops in some cases of simple obstruction and predominant fluid-filled loops in other cases are unclear. Williams[12] contends that the lack of gas is due to the fact that certain patients swallow little or no air. He states that patients with fluid-filled obstructions of the

ileum tend to appear stoical, whereas patients with gaseous distention of the obstructed intestine are usually those who moan, groan, grunt or swallow consistently. The presence or absence of vomiting may also be a factor, since vomiting will tend to empty the upper jejunal loops of gas and fluid.

In contrast, the evaluation of the degree of activity on the successive roentgenograms, using the criteria discussed above, appears to be a satisfactory method of evaluating bowel activity. It appears to be fairly consistent in any individual case. A 1+ activity was associated with complicated obstructions in 72 per cent of the cases, while a 2+ to 4+ activity was associated with complicated obstructions in only 8 per cent of the cases. This technique was also prognostically significant, since only cases with 2+ to 4+ activity improved on nonoperative management. Thus, determination of the degree of activity by roentgen means appeared to be another useful criterion in determining patient management, suggesting immediate surgery in those with reduced activity and an attempt at nonoperative management in those with increased activity.

It must be noted that 8 per cent of the cases in this study with 2+ to 4+ activity did have strangulation or other complications at laparotomy. It is possible that these patients were seen early in their course before reduced activity became manifest. It must be stressed that the successive studies are only an evaluation of activity at the time of the study, and that if the clinical condition of the patient suggests change, the activity should be reevaluated by a repeat study. It is known from experimental studies[3] that the inhibition of activity in strangulating obstruction may be delayed. Another possibility is that in some patients these complications do not decrease bowel activity. Of interest in this regard is the fact that Dixon *et al.*[2] showed in experimental studies that in strangulating obstruction, the decrease in activity was probably the result of sympathoadrenal discharge resulting in activation of inhibitory alpha and beta adrenergic receptors in the intestine. This inhibition was prevented or relieved by alpha and beta adrenergic blockade using drugs. Thus, it is conceivable that in some patients the mechanism does not operate or improves spontaneously, depending on the degree of bowel compromise and associated complications.

The cause of reduced activity in those cases in whom simple obstructions were found is not completely clear. It is possible that these were cases of prolonged distention with a fatigued adynamic state that can occur proximal to a simple mechanical obstruction which has been present for several days. This is suggested by the slightly increased incidence of markedly dilated bowel loops (*i.e.*, those with maximum diameters of 6.5 to 8 cm.) in this group of cases. Another possibility is that the reduced activity in these cases was secondary to dehydration and electrolyte imbalance, especially hypokalemia,[8] which have been shown to affect bowel activity. The current study indicates that electrolyte abnormalities are frequently present in cases of small bowel obstruction, and that there is a slightly increased incidence of reduced serum potassium in cases with reduced activity.

This report does not discuss the use of successive roentgenograms in the differential diagnosis of small bowel obstruction and nonobstructive distention which entirely or predominantly involves the small bowel and is due to inflammatory intra-abdominal conditions. This subject is still under study, but preliminary results indicate that bowel activity is present in most cases of nonobstructive small bowel distention, except in association with diffuse peritonitis or postoperatively.[1]

SUMMARY

One hundred cases of small bowel obstruction were studied using the technique

of 2 successive supine abdominal roentgenograms exposed at an interval of 5 minutes.

The following significant results were noted: (1) the presence of activity in the dilated small bowel loops, as indicated by changes in their appearance in successive roentgenograms, was a more reliable indicator of a dynamic obstruction than the length of the fluid levels and the configuration of the loops in the erect position; and (2) reduced activity in the dilated loops was indicative of a poor prognosis for nonsurgical therapy and was frequently associated with complications such as strangulation, volvulus, hemorrhagic congestion, perforation or peritoneal inflammation.

This technique is a simple and useful addition to the routine single recumbent and erect abdominal roentgenograms obtained in patients with obstructive symptoms.

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REFERENCES

1. Bryk, D., and Rosenkranz, W. Functional evaluation of acute abdomen by radiological means. *CRC Crit. Rev. Radiol. Sc.*, 1971, *2*, 1–45.
2. Dixon, J. A., Harman, C. G., Nichols, R. L., and Englert, E. Intestinal motility following luminal and vascular occlusion of small intestine. *Gastroenterology*, 1970, *58*, 673–678.
3. Dixon, J. A., Nichols, R. L., and Hunter, D. C., Jr. Strangulation obstruction of intestine: early detection. *A.M.A. Arch. Surg.*, 1964, *88*, 527–532.
4. Dixon, J. A., and Nichols, R. L. Roentgen diagnosis of strangulation obstruction of intestine. *Surg., Gynec. & Obst.*, 1966, *122*, 50–54.
5. Frimann-Dahl, J. Experimental ileus in rabbits. *Acta radiol.*, 1951, *35*, 101–114.
6. Frimann-Dahl, J. Roentgen Examinations in Acute Abdominal Diseases. Charles C Thomas, Publisher, Springfield, Ill., 1960.
7. Lasser, E. C. Dynamic Factors in Roentgen Diagnosis. Williams & Wilkins Company, Baltimore, 1967.
8. Lowman, R. M. Potassium depletion states and postoperative ileus: role of potassium ion. *Radiology*, 1971, *98*, 691–694.
9. Mellins, H. Z., and Rigler, L. G. Roentgen findings in strangulating obstructions of small intestine. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1954, *71*, 404–414.
10. Nelson, S. W., and Christoforidis, J. Use of barium sulfate suspensions in diagnosis of acute diseases of small intestine. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *104*, 505–528.
11. Vest, B. Roentgenologic diagnosis of strangulating closed-loop obstruction of small intestine. *Surg., Gynec. & Obst.*, 1962, *115*, 561–567.
12. Williams, J. L. Obstruction of small intestine. *Radiol. Clin. North America*, 1964, *2*, 21–31.

LACTOSE-BARIUM SMALL BOWEL STUDY*

EFFICACY AS A SCREENING METHOD

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LOS ANGELES, CALIFORNIA

THE recently introduced roentgenologic method originated by Haemmerli *et al.*,[6] and also described by Laws *et al.*,[7] along with the impression of several investigators, that disaccharidase deficiency may be a common problem in most racial groups, have led to the suggestion by Preger and Amberg[10] that lactose be used with barium in small bowel examinations routinely to screen for lactase deficiency.

The authors have screened 100 consecutive patients undergoing small bowel examination, regardless of whether lactase deficiency was suspected or not, in order to test this hypothesis.

The hydrolysis of sugars within the bowel lumen depends upon the presence of specific disaccharidases, principally lactase, sucrase and maltase. When these are lacking a physiologic chain of events begins which ends with the production of explosive diarrhea. The majority of intolerances to milk products result from this inability to handle lactose. The problem is, however, that the offending agent, and hence the cause of the problem goes unrecognized.

Centrifuged mucosal hemogenate and fluorescent antibody studies have shown that the various disaccharidase activities are located in the brush border of the intestinal mucosal cells. This activity is uniformly high over the entire length of the small intestine with somewhat lower values in the duodenum and distal ileum.[9] Although various types of disaccharide malabsorption exist, lactose is the most common and the deficiency may be either on a hereditary or acquired basis. Acquired lactose malabsorption may occur as an isolated problem or may be part of a secondary, generalized disaccharide malabsorption as is seen in patients with various primary intestinal disorders.

In these so-called "fermentative diarrheas" the same basic mechanism for the production of diarrhea exists. A rather marked osmotic effect is exerted in the colon by the undigested sugars. In addition to accumulation of water with dilution of colon content there is a direct irritating effect on the colon mucosa from lactic and acetic acids produced from partial bacterial breakdown.

With the addition of barium to the lactose meal, roentgenologic visualization of the shortened transit time, dilated bowel lumen, and dilution of intestinal contents can be seen. Prior to the lactose-barium meal no reliable practical screening method had been devised. A recent breath analysis examination which involves determination of the concentration of hydrogen in expired air following in-take of carbohydrates may prove useful for this purpose.[3]

The association of disaccharidase deficiencies with other disease entities has been pointed out by Littman and Hammond[8] and includes the following: infectious diarrhea, sprue, cystic fibrosis, ulcerative colitis, regional enteritis, giardiasis, and peptic ulcer. It has been felt that a successful method of detecting lactase deficiency would inadvertently lead to the diagnosis of other malabsorption diseases.

PATIENT MATERIAL

One hundred unselected patients undergoing small bowel roentgenologic examinations at the White Memorial Hospital in Los Angeles in 1970 received 50 gm. of lactose mixed with their nonflocculating

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barium meal. Sixty-two per cent of the patients were female. The age distribution mean was 53 years. Five per cent were Oriental, 15 per cent Negro, 25 per cent Latin-American, and 55 per cent non-Latin Caucasians.

METHOD

Subsequent to the usual overnight fasting, as a routine all patients received barium-lactose mixture with a uniform lactose concentration; *i.e.*, 50 gm. lactose per 8 oz. of barium sulfate. Those patients undergoing upper gastrointestinal study in conjunction with small bowel examination received lactose for fluoroscopy, and timing of the study was begun from the completion of upper gastrointestinal fluoroscopy. Exposure interval selection was under direct supervision of a radiologist in all cases. Most commonly the sequence consisted of 15, 30, 45, 60 and 90 minute roentgenograms followed by roentgenograms at hourly intervals thereafter until visualization of the proximal colon.

ROENTGEN FINGINGS

Thirty-three patients (33 per cent) had abnormal examinations (Fig. 1). Of these, 8 had malabsorption patterns which fell into 2 categories: rapid transit; and normal or slowed transit. Varying criteria of malabsorption were present in each. Transit was considered quickened if barium reached the colon on the 15 minute roentgenogram. Only 2 patients had rapid transit, neither of whom showed the other characteristic findings of the triad, that is dilatation of small intestinal loops and dilution of barium. Neither experienced urgent diarrhea. Six persons had normal or delayed transit but none of these showed an abnormal dilution of barium in the distal small bowel or colon. Segmentation, dilatation, flocculation and thickened valvulae were frequent findings. Diagnoses in the 6 were intestinal lipodystrophy, cirrhosis, (2 cases) partial small bowel resection, nephrotic syndrome, and metastatic ovarian cyst adenocarcinoma.

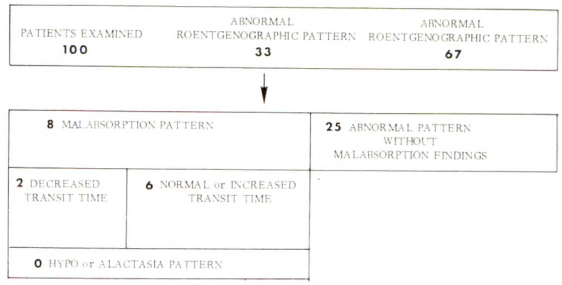


Fig. 1.

Eleven patients had a partial or complete obstructive pattern. Five showed displaced small bowel loops, mostly from adjacent neoplastic processes. Crohn's disease was present in 3 and none of these examinations showed appreciable dilatation, dilution or intestinal hurry; in fact, one presented with constipation and weight loss. Other diagnoses were Meckel's diverticulum, diverticulosis, right upper quadrant hernia and gastroenteritis.

DISCUSSION

Of the 8 examinations suggesting malabsorption, none fits into the characteristic pattern of lactose malabsorption. In those with intestinal hurry, the other 2 criteria for diagnosis of intestinal hypolactasia were lacking. One patient was a 91 year old female with massive rectal bleeding, the other was found to have arachnoiditis with resulting pituitary insufficiency and secondary adrenal insufficiency. In the group with normal or delayed transit, it is unlikely that lactose intolerance played a prominent part of the malabsorption picture. No control studies were done in this series with plain barium, however. The patient with cystadenocarcinoma had relief of intestinal symptoms with milk.

Gravelle and Marsden[5] reported on 63 patients with proctocolitis examined by lactose-barium mixture, 5 of whom were shown to have abnormal patterns confirmed as hypolactasia. Lactase deficiency has also been reported in granulomatous colitis and irritable colon syndrome.[11] The 3 cases of Crohn's disease in this series

did not show a roentgenographic pattern of hypolactasia.

CONCLUSIONS

Although in certain cultures and racial groups, the incidence of lactase deficiency may be very high, the results of our screening would not seem to bear this out for residents in Southern California. It is doubted that the statement by Preger and Amberg,[10] "From what is known today, it must be considered the most common small bowel disorder" is true for our population group. At least this could not be confirmed by the roentgenologic screening method.

When performed as a routine examination, a great deal of care and supervision by the interpreting radiologist is required to personally assure him that lactose was actually added to the barium meal. It was also found that considerable personal control was required to get accurate timing of early roentgenograms, especially when the upper gastrointestinal and small bowel studies were combined. These aggravating difficulties, added to the apparent low productivity of the method as a screening procedure, suggest to the authors that the lactose-barium meal should be reserved for those patients who have clinical suspicion of lactase deficiency. When used in this way there should be great value for the roentgenologic method. It is certainly much superior to fecal analysis, lactose tolerance test, and dietary tests in terms of acceptance to both patient and physician.

SUMMARY

The practicability of the roentgenologic lactose-barium method as a routine screening method for lactase deficiency has been tested on 100 unselected patients from the Los Angeles area of Southern California.

Lactase deficiency has not been found to show the frequency in the study population that has been recently suggested.

The method should be reserved for those showing clinical suspicion of lactase deficiency.

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REFERENCES

1. Besigye, E., and Kajubi, S. K. Experience with radiological method of diagnosing lactase deficiency. *East African M. J.*, 1969, *46*, 564–571.
2. Bowdler, J. D., and Walker-Smith, J. A. Role of radiology in diagnosis of lactose intolerance in child. *Ann. radiol.*, 1969, *12*, 467–472.
3. Calloway, D. H., Murphy, E. L., and Bauer, D. Determination of lactose intolerance by breath analysis. *Am. J. Digest. Dis.*, 1969, *14*, 811–815.
4. Cook, G. C., and Howells, G. R. Lactosuria in African with lactase deficiency. *Am. J. Digest. Dis.*, 1968, *13*, 634–637.
5. Gravelle, I. H., and Marsden, R. T. H. Radiological assessment of hypolactasia in ulcerative colitis. *Brit. J. Radiol.*, 1969, *41*, 416–418.
6. Haemmerli, U. P., Kistler, H., Ammann, R., Marthaler, T., Semenza, G., Auricchio, S., and Prader, A. Acquired milk intolerance in adult caused by lactose malabsorption due to selective deficiency of intestinal lactase activity. *Am. J. Med.*, 1965, *38*, 7–30.
7. Laws, J. W., Spencer, J., and Neale, G. Radiology in diagnosis of disaccharidase deficiency. *Brit. J. Radiol.*, 1967, *40*, 594–603.
8. Littman, A., and Hammond, J.B. Diarrhea in adults caused by deficiency in intestinal disaccharidases. *Gastroenterology*, 1965, *48*, 237–249.
9. Prader, A., and Auricchio, S. Defects of intestinal disaccharide absorption. *Ann. Rev. Med.*, 1965, *16*, 345–358.
10. Preger, L., and Amberg, J. R. Sweet diarrhea. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *101*, 287–295.
11. Weser, E., Rubin, W., Ross, L., and Sleisenger, M. H. Lactase deficiency in patients with irritable-colon syndrome. *New England J. Med.*, 1965, *273*, 1070–1075.

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SELECTIVE VASOCONSTRICTOR INFUSION IN THE MANAGEMENT OF ARTERIO-CAPILLARY GASTROINTESTINAL HEMORRHAGE*

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SELECTIVE vasoconstrictor infusion into the bleeding artery or its feeder, causing local vasoconstriction with diminished blood flow and thereby permitting stable clot formation, offers rational, nonoperative means for controlling both variceal and arterial bleeding into the gastrointestinal tract.[2,6–9,11]

A 3 year experience forms the basis for the following report on the vasoconstrictive therapy of arteriocapillary bleeding.

MATERIAL AND TECHNIQUE

Selective arterial vasoconstriction was used to treat 38 of our patients with arteriocapillary bleeding from various gastrointestinal lesions (Table 1). In all cases, diagnostic studies were done first and therapy was undertaken only if active bleeding was shown by the arteriographic demonstration of extravasated contrast material in the lumen of the gastrointestinal tract. In the presence of upper gastrointestinal hemorrhage, selective celiac, and sometimes superior mesenteric arteriography was done. In questionable cases, selective injections into the left gastric, hepatic or gastroduodenal arteries followed. Selective inferior and superior mesenteric arteriography was performed when lower gastrointestinal bleeding was clinically evident. With the bleeding identified, the angiographic catheter was either left in place for infusions, or if indicated and possible, advanced closer to the bleeding point. In the Mallory-Weiss syndrome or bleeding into the upper stomach, selective left gastric artery catheterization was attempted; selective common hepatic catheterization was done for bleeding lesions of the gastric antrum and pylorus. In 2 patients with bleeding duodenal ulcers, the catheter was positioned in the gastroduodenal artery. For bleeding into the large bowel, the catheter was left in either the superior or inferior mesenteric artery, depending on the site of the lesion. In 1 patient with bleeding into the terminal ileum, the catheter was advanced deep into the ileocolic artery.

Epinephrine was the vasoconstrictor in 30 patients; in 6 of these with colonic bleeding, its use was immediately preceded by the injection of 3–5 mg. of propranolol. Epinephrine dosage varied from 8–16 micrograms per minute for the left gastric, gastroduodenal or inferior mesenteric arteries to 20–30 micrograms per minute for the celiac and superior mesenteric arteries. Vasopressin, the vasoconstrictor in 8 patients, was given at doses of 0.05–0.1 units per minute in the left gastric or ileocolic arteries and 0.2–0.3 units per minute in the celiac or superior mesenteric arteries. Infusions lasted from 20 to 60 minutes, depending mainly on the patient's condition and response to treatment. The longer infusions were given to patients with arteriosclerosis or impaired hemocoagulation. Repeat angiography was then performed. If this showed no further bleeding, a final 20 minute infusion was given to counteract the vasodilating effect of the contrast medium. The catheter was then removed in patients treated early in this series; in later cases, it was filled with heparin solution and left in the perfused artery for 1–3 days to facilitate treatment

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TABLE I

RESULTS OF SELECTIVE VASOCONSTRICTIVE THERAPY IN 38 PATIENTS WITH ACTIVE ARTERIO-CAPILLARY GASTROINTESTINAL BLEEDING (DATA THROUGH DECEMBER 31, 1971)

Disease	No. of Treated Patients	Complete Control	Limited Control	No Control
Mallory-Weiss syndrome	2	2	—	—
Gastritis	9	7	—	2
Stress ulcer	6	5	1	—
Peptic ulcer	13	2	2	9
Colitis	2	2	—	—
Colon diverticulum	4	3	1	—
Colon angioma	2	2	—	—
Total	38	23	4	11

in the event of early recurrent bleeding. If the postinfusion angiogram showed persistent bleeding, further infusion was done if it appeared to be worthwhile on the basis of the initial response and safe from the clinical point of view.

All patients had necessary supportive therapy, the electrocardiogram was continuously monitored, and the blood pressure was measured at frequent intervals during infusions.

RESULTS

The effectiveness of selective arterial vasoconstrictive therapy was dependent upon the underlying pathology (Table 1).

MALLORY-WEISS SYNDROME

In 2 patients with mucosal tears at the esophago-cardiac junction, 20 minute vaso-

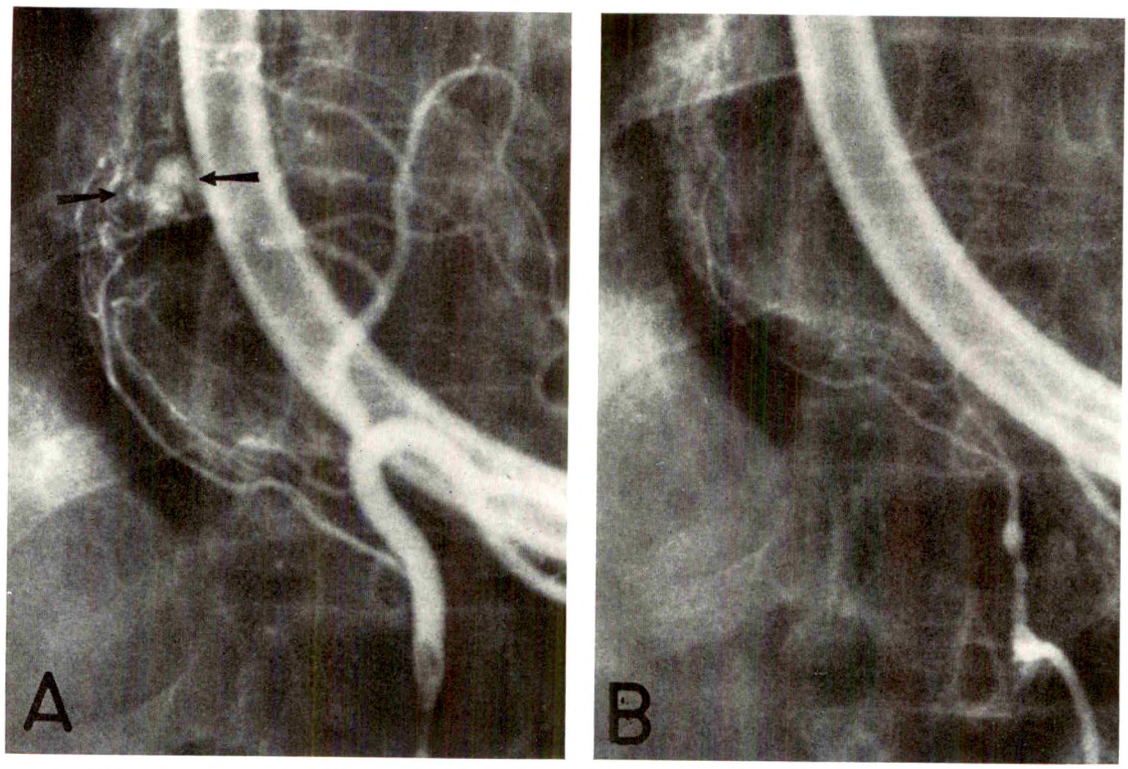


FIG. 1. A 62 year old man with a hiatal hernia, Mallory-Weiss syndrome, and severe gastrointestinal bleeding. Selective left gastric arteriography. (*A*) Control study demonstrating extravasation of contrast medium in a hiatal hernia (arrows). (*B*) Following a 20 minute infusion of epinephrine (10 micrograms per minute) there is extensive constriction of the left gastric artery and its branches, and extravasation is no longer occurring.

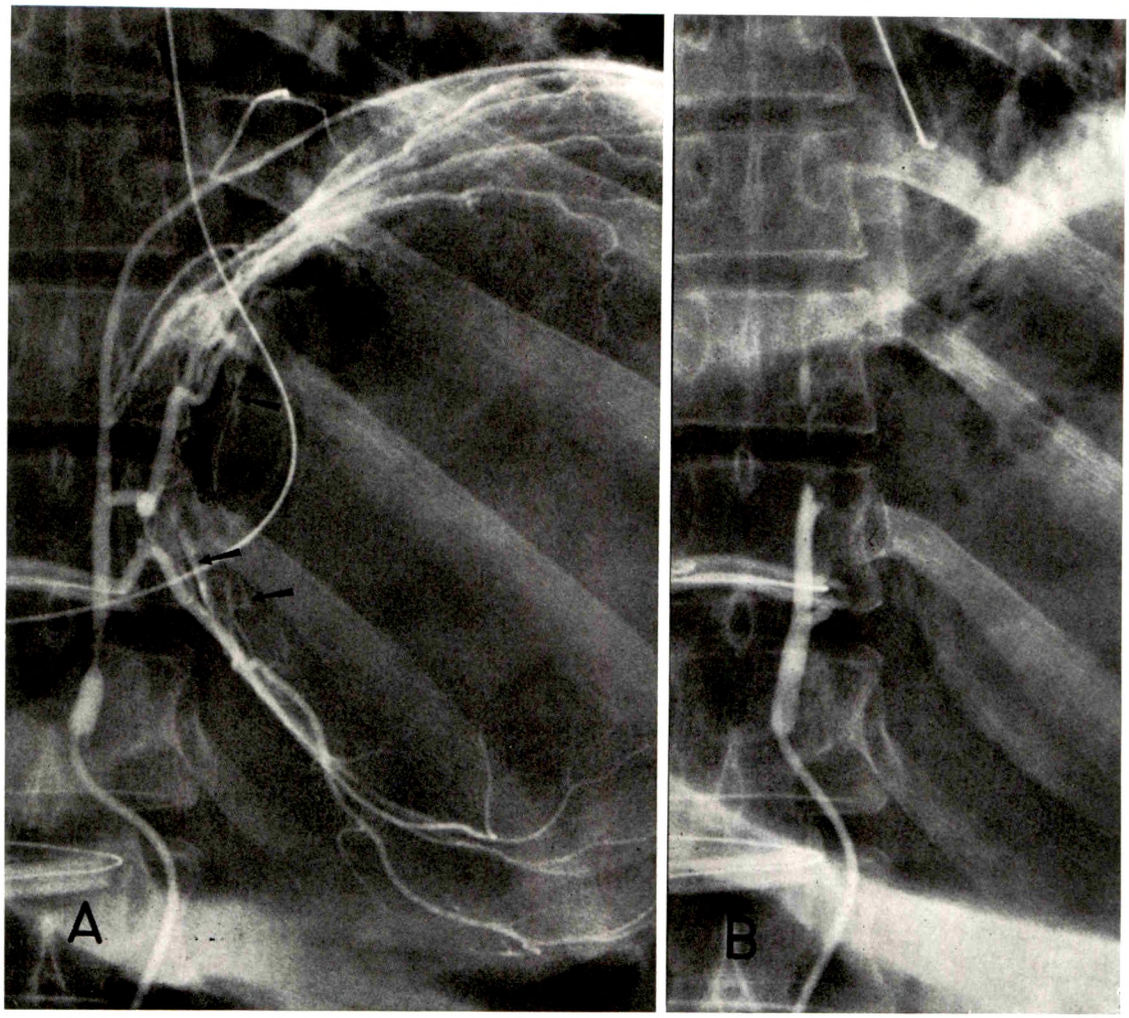


FIG. 2. A 39 year old man with erosive gastritis and severe gastrointestinal bleeding. Selective left gastric arteriography. (*A*) Control study demonstrates several extravasations of contrast medium on the lesser curvature of the stomach, which is distended by blood (arrows). (*B*) Following a 30 minute infusion of epinephrine (12 micrograms per minute), there is extensive vasoconstriction of all left gastric artery branches and no extravasation of contrast medium.

constrictive infusions promptly, completely and permanently stopped the bleeding. On follow-up angiograms there was pronounced vasoconstriction without the extravasation of contrast agent which had been present on the pre-infusion study (Fig. 1, *A* and *B*). Surgery was not necessary and bleeding did not recur.

GASTRITIS

Nine patients with various types of hemorrhagic gastritis were treated, and in 7 the bleeding stopped after infusions lasting from 20 minutes to 3 hours (Fig. 2, *A* and *B*). In 1 of these 7, the bleeding recurred 14 hours later, and surgery disclosed a duodenal ulcer which had eroded into the duodenal artery. It appeared that the prior gastric bleeding had been due to mucosal trauma from a gastric tube. Dual bleeding sites were also encountered in another patient whose angiographically documented gastric bleeding stopped after the infusion, but in whom persistent,

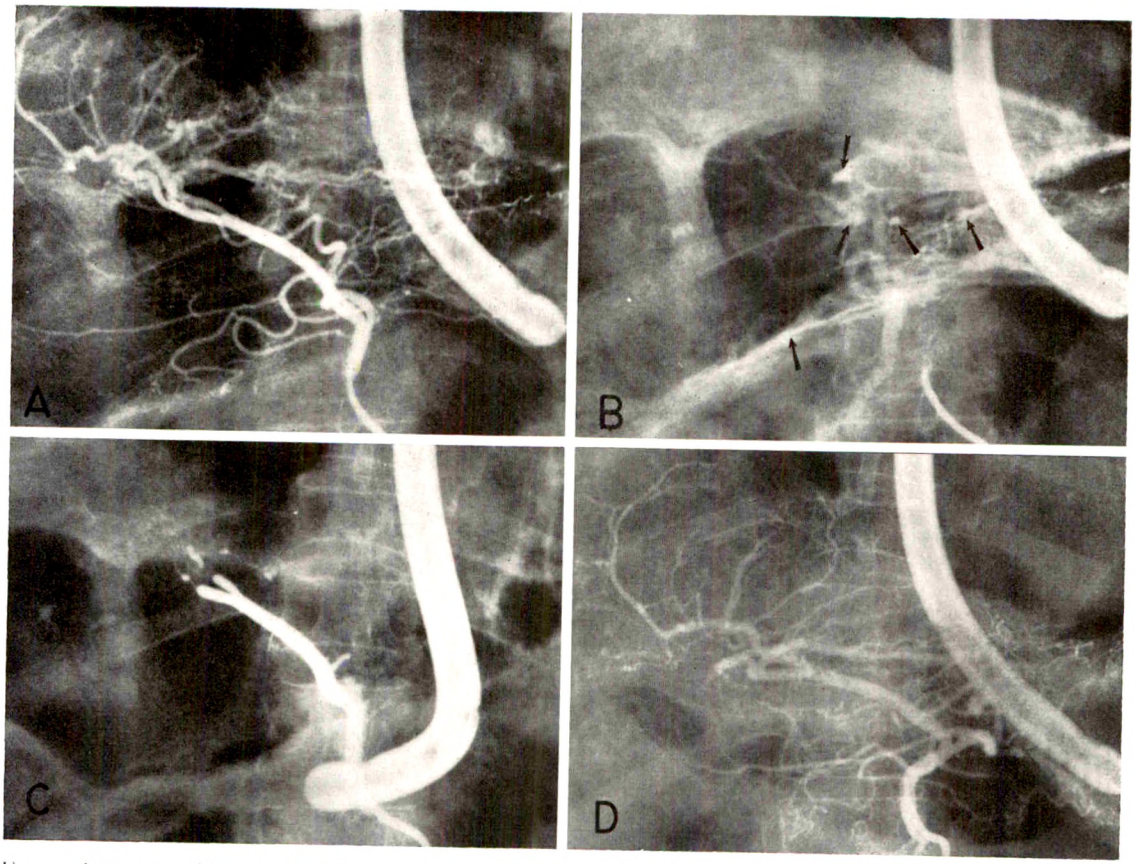


FIG. 3. A 51 year old woman with multiple superficial gastric stress ulcers causing severe bleeding. Selective left gastric arteriography. (*A* and *B*) Control study demonstrates several small extravasations of contrast medium in the stomach (arrows). The left gastric artery also feeds an aberrant left hepatic artery. (*C*) Following a 30 minute infusion of epinephrine (12 micrograms per minute) there is extensive constriction of all branches of the left gastric artery and extravasation of contrast agent is no longer occurring. (*D*) Forty-eight hours after the vasoconstrictive infusion, contrast agent injected through the catheter remaining in the left gastric artery shows normal findings.

clinically evident bleeding led to death before surgery could be done. Autopsy disclosed that in addition to the expected, erosive gastritis, an aortic aneurysm had ruptured into the lower duodenum. The ninth patient of this group had terminal renal disease with uremic gastritis. The prolonged infusion of epinephrine led to minimal vasoconstriction with little or no decrease in bleeding.

STRESS ULCERS

Stress ulcers, mostly postsurgical, led to bleeding in 6 of our patients. Selective vasoconstrictor control was achieved in 5 of these 6 (Fig. 3, *A–D*). In 3 patients, 20 to 60 minute infusions sufficed, while in 2 patients with hemocoagulation defects, longer infusions (of 8 and 24 hours) were necessary to control the bleeding. In the remaining patient, bleeding from stress ulcers occurred shortly after a transplant nephrectomy. Severe hypertension and a defective clotting mechanism were also present. A 2 hour epinephrine infusion caused only moderate vasoconstriction and a decrease in the bleeding, although this subsequently stopped completely following the combined delivery of epinephrine and packed platelets through the catheter. Surgery was avoided in all 6 patients with stress ulcers.

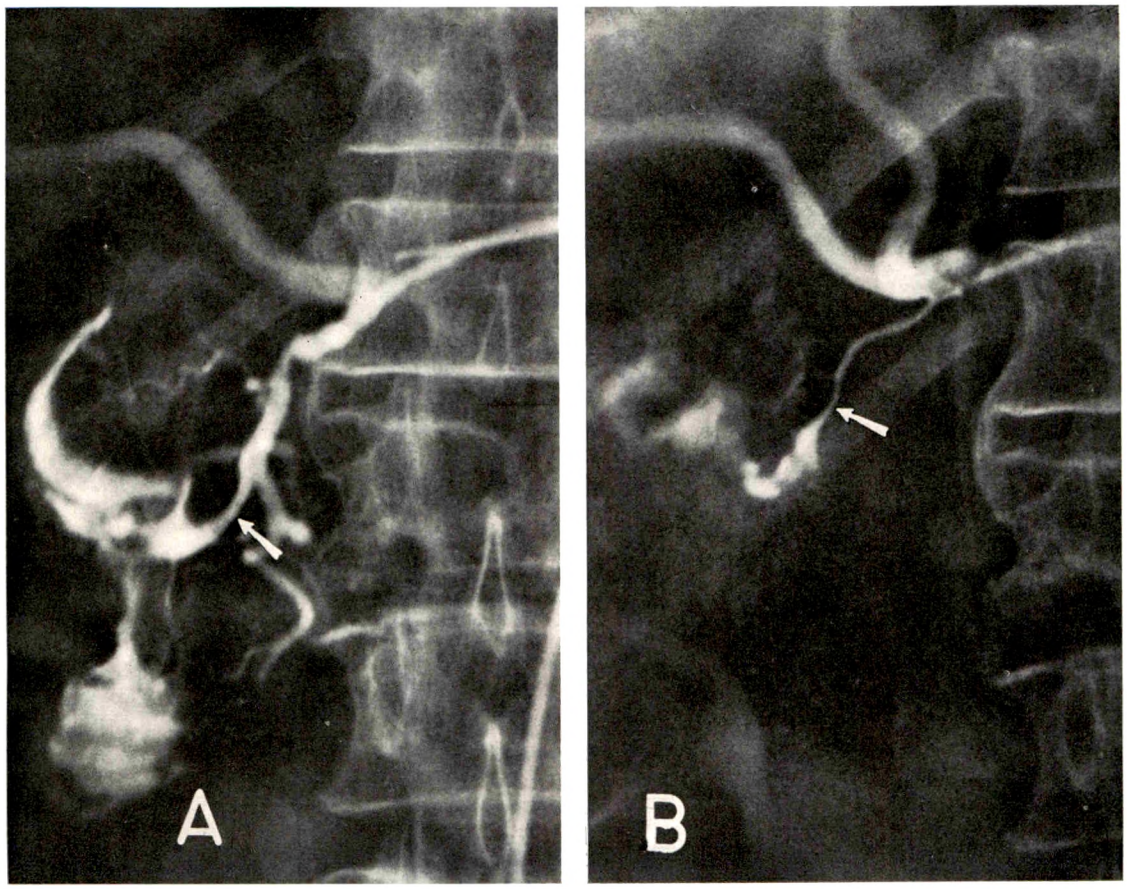


Fig. 4. A 74 year old man with severe gastrointestinal bleeding from a huge duodenal ulcer penetrating into the pancreas and eroding duodenal artery. (*A*) Control gastroduodenal arteriogram reveals extensive extravasation of contrast medium into the duodenum from a cut-off duodenal artery (arrow). (*B*) Follow-up hepatic arteriogram after 30 minute infusion of epinephrine into the gastroduodenal artery (10 micrograms per minute) shows vasoconstriction of gastroduodenal artery, but wide open duodenal artery (arrow) and persistent extravasation of contrast medium.

PEPTIC ULCERS

Selective vasoconstrictors were less successful in bleeding peptic ulcers. In only 2 of 13 treated patients was bleeding controlled and emergency surgery avoided. In 2 other patients, bleeding decreased substantially during and shortly after infusion, but recurred later, requiring surgery. No control was gained through vasoconstrictor infusions in the remaining 9 patients of this group. In 3 of these with cirrhosis and severe coagulation defects, bleeding stopped during infusion, but recurred immediately thereafter. Two of the 3 were operated on; the third was successfully treated by nonoperative selective embolization of the gastro-epiploic artery through the catheter.[10] In the other 6 vasoconstrictor failures, there was poor constriction of perfused vessels in 2 patients and in the other four, all vessels except the bleeder constricted (Fig. 4, *A* and *B*). In 2 of the latter, the constriction of nonbleeders effectively shunted blood into the eroded artery; thus, bleeding actually increased after infusion. In summary, despite vasoconstrictor infusions, 10 of our 13 patients with peptic ulcers required emergency surgery for bleeding.

COLONIC BLEEDING

Bleeding from colonic lesions (colitis,

angioma or diverticulum) stopped promptly in response to 20 to 30 minute vasoconstrictor infusions in all 8 treated patients (Fig. 5, *A–D*; and 6, *A* and *B*). Bleeding recurred and led to emergency surgery in 1 patient 6 hours after an infusion given

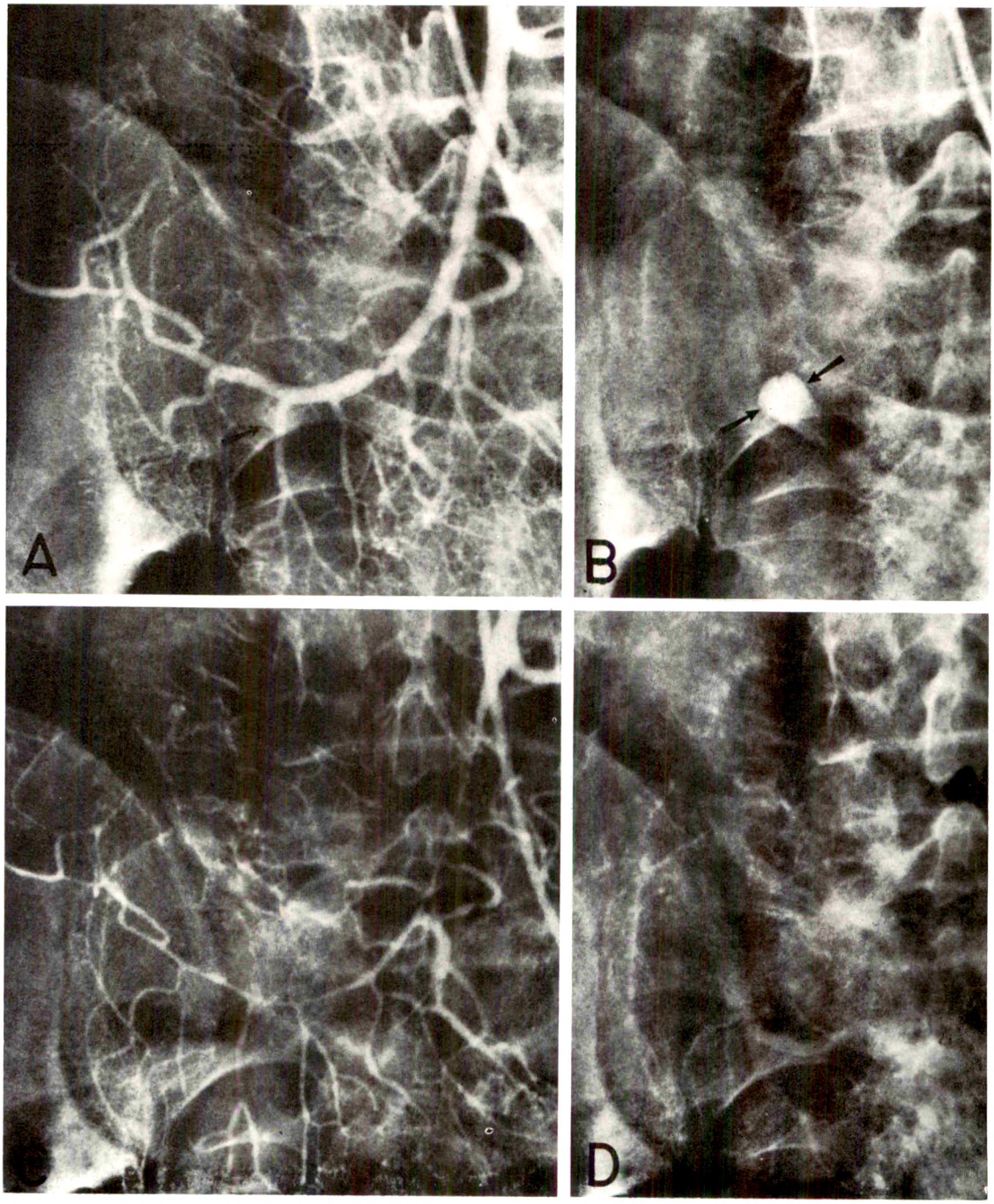


FIG. 5. A 64 year old man with a total colectomy and severe gastrointestinal bleeding. Selective ileal arteriography. (*A* and *B*) Control study demonstrates extravasation of contrast medium from terminal ileum branches (arrows). (*C* and *D*) Following a 20 minute infusion of vasopressin (0.1 unit per minute), there is extensive vasoconstriction of ileal branches and extravasation is no longer occurring.

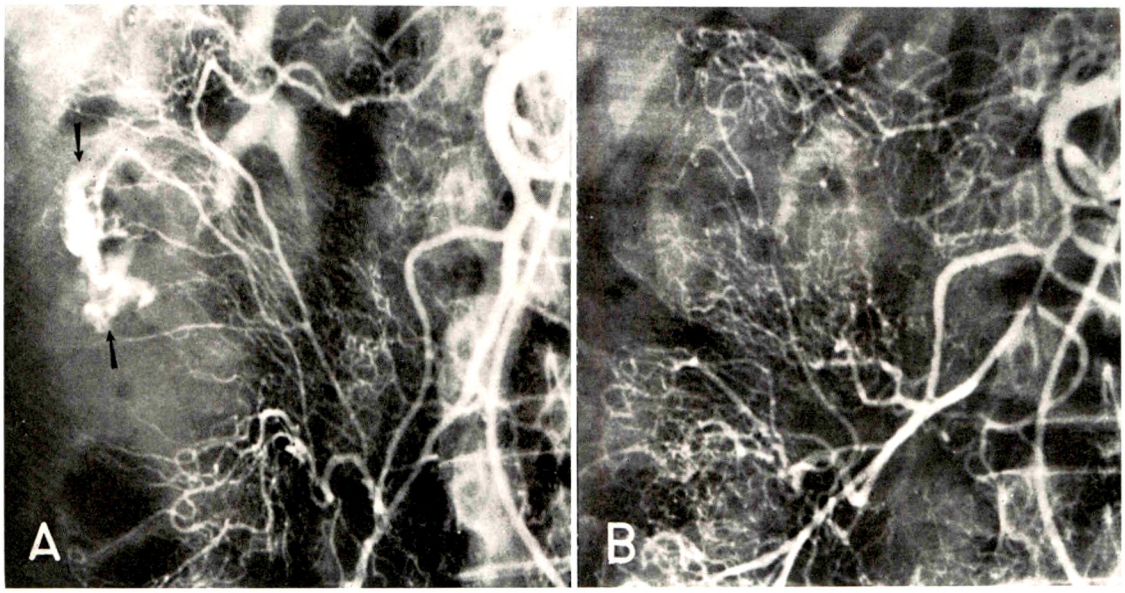


Fig. 6. A 75 year old man with severe gastrointestinal bleeding from colic diverticulum. Selective superior mesenteric arteriography. (*A*) Control study demonstrates extravasation of contrast medium into the ascending colon (arrows). (*B*) Study done 20 minutes after 30 minute infusion of vasopressin (0.3 unit per minute), shows no more extravasation.

early in this series. We now leave the catheter in the artery and expect to be able to control recurrent bleeding such as this. Four of our 8 patients had elective surgery later—not because of bleeding, but to treat the underlying lesion.

DISCUSSION

Factors bearing upon the conduct and results of selective vasoconstrictive therapy for arterio-capillary gastrointestinal bleeding include the patient's general condition, the adequacy of supportive therapy, the establishment of a positive arteriographic diagnosis, the nature and location of the bleeding lesion, the selectivity of infusion, the choice of and response to vasoconstrictor, and the presence of an adequate clotting mechanism.

The *positive diagnosis* of bleeding by arteriography through the demonstration of direct extravasation of contrast medium into the gastrointestinal lumen is the *sine qua non* of selective vasoconstrictive therapy in arterio-capillary bleeding. This is not the case in variceal bleeding, where a presumptive angiographic diagnosis is usually reached through the exclusion of arterial bleeding and the finding of esophageal or gastric varices. The diagnostic value of arteriography in arterial bleeding is closely related to the technique used. Selective celiac, superior or inferior mesenteric arteriography can show bleeding of as little as 0.5 cc. per minute.[1,4] This is usually sufficient in one-point bleeding such as the Mallory-Weiss syndrome, peptic ulcers and angiomata or diverticula of the large bowel. With the diffuse bleeding of gastritis and sometimes stress ulcers, so-called "superselective" catheterization and angiographic injection into gastric branch arteries often has to be done. In 3 of our patients celiac arteriography was negative, but left gastric arteriography clearly showed multiple small extravasations. More than a single visualized bleeding point can be present in a given patient; bleeding into the stomach may be secondary to the mucosal trauma caused by a gastric tube! In 2 patients we treated secondary gastric lesions, missing in one the rupture of an aortic aneurysm into the duodenum, and in the other a large duo-

denal ulcer which temporarily stopped bleeding at the time of angiography.

The *selectivity of infusions* is an important determinant of success or failure; vasoconstrictors should be infused as close as possible to the bleeding point. Selective left gastric infusions should be given to control bleeding from Mallory-Weiss tears or upper gastric lesions. Gastroduodenal artery infusions should be done for duodenal bleeding and ileocolic artery infusions for bleeding from the cecum or terminal ileum. "Superselectively" given vasoconstrictors are locally effective in low doses, and systemic effects may be minimized or avoided. Infusions into the main trunk of the celiac or superior mesenteric artery require higher doses to control the bleeding; furthermore, infusions into the celiac artery are less likely to control upper gastric bleeding, since celiac catheters often tend to slip or deliver injections beyond the orifice of the left gastric artery. Special problems are presented by bleeding from lesions in areas supplied by two arterial systems such as the middle part of the stomach (supplied by the right and left gastric and gastro-epiploic arteries), the descending duodenum (supplied by branches of the celiac and superior mesenteric arteries) and the middle transverse colon (from superior and inferior mesenteric arteries). In such cases, 2 catheters can be used to simultaneously infuse the dual feeder systems.

Various *vasoconstrictors* have been evaluated for their possible use in selective therapy.[5,8] Epinephrine was our initial choice. Its effects last only during the infusion, after which a brief, 2 to 3 minute period of reactive vasodilatation is promptly followed by a return to normal.[8] Epinephrine is a good vasoconstrictor for the celiac artery and its branches; in the mesenteric circulation, however, it produces undesirable beta effect dilatation. Although this can be blocked by pre- or concomitantly given propranolol, the latter is unsuitable for use in elderly patients because of its effects on cardiac function. Therefore, we now use vasopressin for mesenteric infusions, following Baum's lead. It is a predominantly peripheral vasoconstrictor with a prolonged action which persists after the infusion and exhibits no reactive hyperflow.[5,8]

Animal studies of selectively infused phenylephrine (neo-synephrine) and methoxamine (vasoxyl) revealed inconstant effects and a wide variation in effective dose from one individual to another. We have used neither clinically.

The *character*, and to a lesser extent, the *location of a bleeding lesion* have much to do with its treatment. In our experience, vasoconstrictive therapy is most effective in small artery and capillary bleeding such as that of gastritis, mucosal tears, superficial stress ulcers, and colitis or diverticulitis. It is less likely to stop the bleeding of peptic ulcers, especially when larger arteries are involved. Bleeding lesions supplied by catheterizable end arteries such as the left gastric, ileocolic and inferior mesenteric, are more likely to respond than lesions with dual arterial supplies such as those in the middle part of the stomach, descending duodenum and mid-transverse colon.

The infused bleeder (or feeder) arteries must be capable of a *vasoconstrictive response*, a capability which can be seriously limited by atherosclerosis. In 2 of our patients with severe renal disease, virtually no vasoconstriction occurred. Furthermore, if failure to constrict is limited to the arteries supplying a bleeding peptic ulcer, the normal response of other infused branches can lead to increased bleeding. The adequacy of response in a given patient should always be evaluated arteriographically and treatment continued only if sufficient vasoconstriction can be achieved.

A *coagulation mechanism* capable of forming a stable blood clot is necessary for selective vasoconstrictor-induced hemostasis. In patients with hemocoagulation

defects, vasoconstriction may give temporary control of bleeding during infusion with recurrence immediately afterwards. Even though not curative, vasoconstrictor infusions can still be of value by reducing preoperative blood loss.

Other nonoperative hemostatic approaches appear to have promise. Diffuse bleeding from capillaries may respond to combined selective vasoconstrictor infusion and platelet transfusion. The production of intravascular obstruction by the injection of autogenous clots[10] and the use of injectable elastometric catheters to produce controlled reversible intraluminal occlusion[3] are under investigation in our experimental laboratory.

The *risks* associated with the proper and appropriate use of selective vasoconstriction should approximate those of visceral angiography and general supportive therapy. A few of our patients noted intestinal hypermotility and abdominal cramps. Minimal elevations of blood pressure were occasionally recorded during infusions. In 1 patient, transient hypotension and bradycardia developed with the combined use of propranolol and epinephrine, a technique we no longer use. Electrocardiograms showed no evidence of myocardial ischemia in any patient, nor were there ischemic changes in the distribution of directly infused arteries. Neither local nor distant complications resulted from leaving the catheter in place for as long as 2 days in arteries as small as the left gastric (Fig. 3, *A–D*).

CONCLUSIONS

1. The selective intraarterial infusion of vasoconstrictors provides a new and effective nonoperative technique for managing small vessel arterial and capillary bleeding such as that due to gastritis, superficial stress ulcers, Mallory-Weiss tears and a variety of large bowel lesions.
2. Angiographic demonstration of bleeding, its character and location, the obtainable degree of infusion selectivity, vascular response to vasoconstrictor administration and the patient's clotting mechanism are important factors in the conduct of treatment.
3. Vasopressin is the drug of choice for perfusion of mesenteric vessels. Both vasopressin and epinephrine are effective in the celiac circulation, but until vasopressin's effect on the liver has been established, epinephrine should be used for celiac and hepatic infusions.
4. To date, selective intraarterial vasoconstrictor infusions have been of limited value in the management of large artery bleeding due to chronic peptic ulceration.

SUMMARY

Thirty-eight patients with active, arteriocapillary gastrointestinal bleeding were treated by the selective arterial infusion of epinephrine or vasopressin.

Vasoconstrictive therapy had a success rate of 80 per cent in small vessel bleeding. Complete control of bleeding was achieved in 20; temporary control in 3; and no control in 2 of 25 patients with gastritis, superficial stress ulcers, the Mallory-Weiss syndrome or colic lesions.

Selective vasoconstriction had limited value in the management of large artery bleeding from peptic ulcers, affording complete control in 2, temporary control in 2, and no control in 9 (70 per cent) of 13 patients.

Individual factors of importance in the conduct of treatment include: the patient's general condition, the adequacy of supportive therapy, the angiographic demonstration of the bleeding, the character and location of the bleeding lesion, the obtainable degree of infusion selectivity, choice of the vasoconstrictor, vascular response achieved, and the patient's clotting mechanism.

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REFERENCES

1. Baum, S., Nusbaum, M., Blakemore, W. S., and Finkelstein, A. K. Preoperative radiographic demonstration of intra-abdominal bleeding from undetermined sites by percutaneous selective celiac and superior mesenteric arteriography. *Surgery*, 1965, *58*, 797–805.
2. Baum, S., and Nusbaum, M. Control of gastrointestinal hemorrhage by selective mesenteric arterial infusion of vasopressin. *Radiology*, 1971, *98*, 497–505.
3. Dotter, C. T., Rösch, J., Lakin, P. C., Lakin, R., and Pegg, J. E. Injectable flow-guided coaxial catheters for selective angiography and controlled vascular occlusion. To be published.
4. Nusbaum, M., and Baum, S. Radiographic demonstration of unknown sites of gastrointestinal bleeding. *Surg. Forum*, 1963, *14*, 374–375.
5. Nusbaum, M., Baum, S., Sakiyalak, P., and Blakemore, W. S. Pharmacologic control of portal hypertension. *Surgery*, 1967, *62*, 299–310.
6. Nusbaum, M., Baum, S., Kuroda, K., and Blakemore, W. S. Control of portal hypertension by selective mesenteric arterial drug infusion. *A. M. A. Arch. Surg.*, 1968, *97*, 1005–1012.
7. Nusbaum, M., Baum, S., and Blakemore, W. S. Clinical experience with diagnosis and management of gastrointestinal hemorrhage by selective mesenteric catheterization. *Ann. Surg.*, 1969, *170*, 506–514.
8. Rösch, J., Gray, R. K., Grollman, J. H., Jr., Ross, G., Steckel, R. J., and Weiner, M. Selective arterial drug infusions in treatment of acute gastrointestinal bleeding. *Gastroenterology*, 1970, *59*, 341–349.
9. Rösch, J., Dotter, C. T., and Rose, R. W. Selective arterial infusions of vasoconstrictors in acute gastrointestinal bleeding. *Radiology*, 1971, *99*, 27–36.
10. Rösch, J., Dotter, C. T., and Brown, M. J. Selective arterial embolization—new method for control of acute gastrointestinal bleeding: case report. *Radiology*, 1972, *102*, 303–306.
11. Slezak, P., Lewander, R., and Fredricsson, B. Pharmacologically controlled visceral bleeding by means of intraarterial catheter. *Radiology*, 1969, *93*, 1379–1381.

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PSEUDOTUMORS IN LARGE HIATUS HERNIAS

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THE normal freely movable gastric mucosa is capable of prolapse through natural and surgically created orifices.[1,4] Roentgenographically, these various forms of mucosal prolapse may be visualized as pseudotumors created by the displaced mucosal folds. Such pseudotumors may be difficult to differentiate from true masses, especially in the lower esophagus and in the gastric fundus.[5,7,18] The prolapsed mucosa may occasionally be the source of gastrointestinal hemorrhage. Antegrade esophagogastric mucosal prolapse,[2,6,11–14] retrograde gastroesophageal prolapse,[2,3,8,16,17] gastroduodenal mucosal protrusion,[9] and herniation of redundant mucosa through a gastrojejunostomy[10,15] have been well described. However, the pseudotumor created by the prolapse of gastric mucosa into a hiatus hernia has received, with a single exception,[2] little attention in the radiologic literature. It appears, therefore, appropriate to report the roentgen findings in this entity based on 5 recently observed cases.

REPORT OF CASES

CASE I. The first hospital admission of this 70 year old female was in 1964 because of congestive heart failure. A routine gastrointestinal series revealed a large sliding hiatus hernia measuring 9 cm. in diameter. A lobulated filling defect about 3 cm. in maximum diameter was noted in the hernia (Fig. 1). At a second hospital admission in March 1969, the patient was anemic with a hematocrit of 28 per cent. After 2 blood transfusions the hematocrit rose to 38 per cent. Study of the bone marrow showed a hypochromic anemia consistent with gastrointestinal bleeding. A repeat gastrointestinal series again showed the sliding hiatus hernia with the filling defect within it (Fig. 2). The patient was readmitted in December 1969, because of 3 episodes of tarry stools associated with weakness and dyspnea. On a repeat gastrointestinal series, the filling defect, as seen within the hernia, was larger measuring 4 cm. in maximum diameter (Fig. 3). Surgical exploration of the hernia through a gastrotomy showed only large hypertrophic gastric rugae and no evidence of tumor.

CASE II. This 74 year old female was admitted to the hospital because of sudden syncope followed by mental confusion. She had a history of angina pectoris of many years' duration. On a routine upper gastrointestinal study, a large sliding hiatus hernia measuring 11 cm. in diameter was noted. On a roentgenogram taken in the right anterior oblique position, a lobular, well defined, pseudotumor-like filling defect

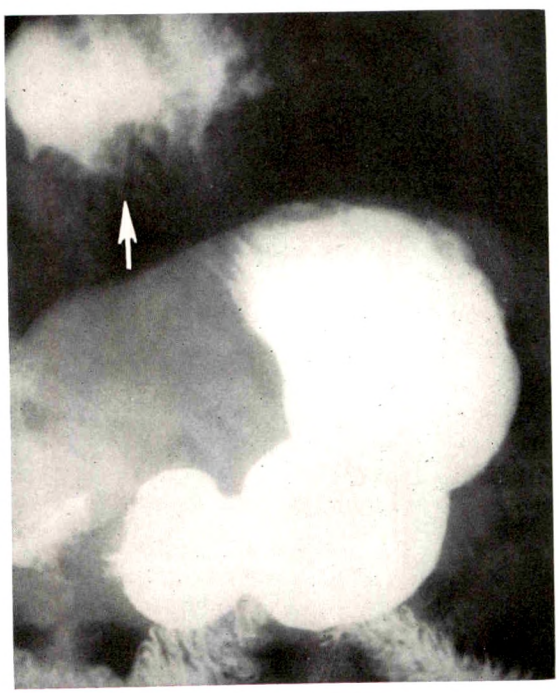


FIG. 1. Case I. Study in 1964. Prone right oblique roentgenogram. Note the large sliding hiatus hernia with a filling defect at the base of the hernia (arrow).

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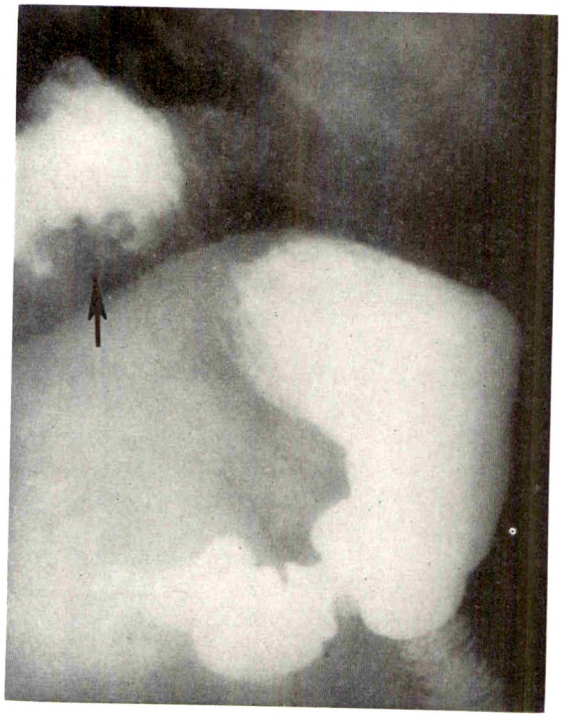


FIG. 2. Case 1. Repeat study in March 1969. Right lateral roentgenogram. Note the filling defect at the base of the sliding hiatus hernia (arrow). The filling defect has a lobulated appearance suggesting that it consists of multiple gastric rugae.

measuring 2.5 cm. in diameter was seen within the hernia. The patient was not anemic and had no complaints referable to the gastrointestinal tract.

CASE III. This 70 year old female was referred for an upper gastrointestinal series because of epigastric pain. Roentgenograms of the stomach in the prone oblique position showed a sliding hiatus hernia measuring 7 cm. in diameter with a well defined lobulated filling defect within it measuring 3.5 cm. in diameter (Fig. 4). In addition, a peptic ulcer located at the neck of the hernia on the anterior wall was observed. Cineroentgenographic studies of the hernia demonstrated the development of the filling defect within the hernia as a result of retrograde prolapse of the redundant gastric mucosa (Fig. 5, *A–C*).

CASE IV. This 59 year old female was admitted because of upper gastrointestinal bleeding. She had a history of heartburn relieved by antacids. On admission she had tarry stools and a hematocrit of 29 per cent. A gastrointestinal series showed a sliding hiatus hernia 7 cm. in diameter with a pseudotumor within it measuring 2 cm. in maximum diameter. Cineroentgenographic studies showed prolapse of the gastric mucosa causing the tumor-like filling defect in the hernia. During the study the pseudotumor was seen to disappear spontaneously as a result of antegrade movement of the prolapsed folds.

CASE V. This 78 year old female was admitted because of increasing dyspnea as a result of longstanding coronary heart disease. She had no complaints related to the gastrointestinal tract and her hematocrit was within normal limits. A routine gastrointestinal study showed a sliding hiatus hernia 14 cm. in diameter. A pseudotumor, 2.5 cm. in diameter, within the hernia was seen on the spot roentgenograms in the prone right anterior oblique position.

ROENTGEN FINDINGS

All 5 cases had moderate to large sized hiatus hernias, which were of the sliding type with the esophagogastric junction displaced into the thorax. The hernias measured 7 to 14 cm. in maximum diameter on the barium filled roentgenograms. The pseudotumor was best demonstrated in the prone right anterior oblique or lateral view

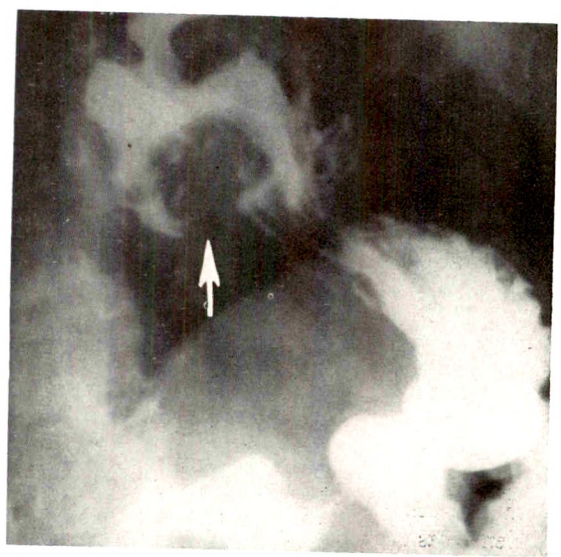


FIG. 3. Case 1. Repeat study in December 1969. Prone right oblique roentgenogram. The filling defect in the hiatus hernia (arrow) appears somewhat larger and more sharply defined. The small collections of barium within it, however, still suggest that it consists of a mass of gastric rugae.

and involved the right posterior aspect of the herniated stomach. It appeared to consist of large gastric rugae present in the neck and base of the hernia which prolapsed intermittently into the lumen of the herniated stomach. This usually produced a somewhat lobulated filling defect in the right posterior aspect of the hernia into which the gastric rugae could be traced.

In the 5 cases studied, the maximum size of these pseudotumors varied from 2 to 4 cm. in maximum diameter. The cineroentgenographic studies in 2 cases (Cases III and IV) best demonstrated the development and reduction of the pseudotumor and confirmed the impression that it was due to retrograde prolapse of gastric rugae into the hernia. In 1 case (Case I) multiple studies were available, 1 study in 1964 and 2 studies in 1969. The size of the filling defect in the hiatus hernia varied from examination to examination, appearing slightly larger on the later studies taken in December, 1969. In all cases there appeared to be prominence of the gastric rugae at the neck of the hernia and within the hernial sac. The physiologic factors responsible for the prolapse and its reduction could not be ascertained from the cineroentgenographic studies. In only 1 case was the pseudotumor associated with additional gastric pathology. This case showed a benign ulcer of the anterior wall of the stomach at the neck of the hernia (Case III).

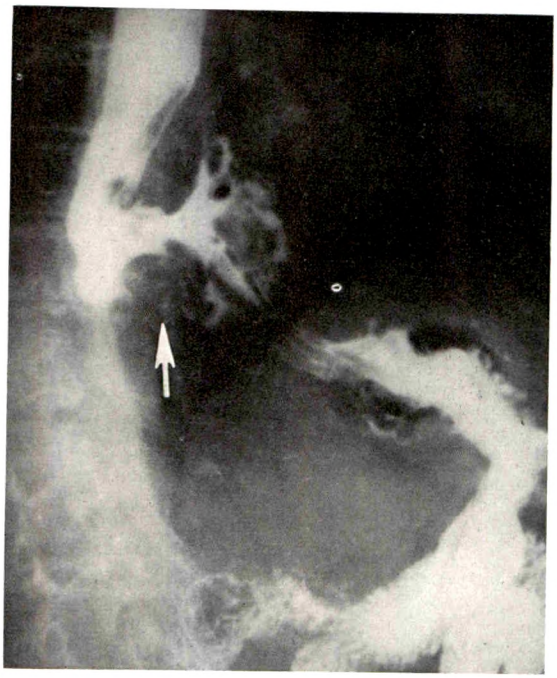


FIG. 4. Case III. Prone right oblique roentgenogram. Note the lobulated filling defect along the right posterior aspect of the large sliding hiatus hernia (arrow).

DISCUSSION

Prolapse of gastric mucosa into a sliding hiatus hernia is apparently not rare. We have seen 5 cases in a relatively short period of 1½ years. All 5 cases were elderly females. The tendency to mucosal prolapse increases with age, presumably due to the loose attachment of the gastric mucosa to

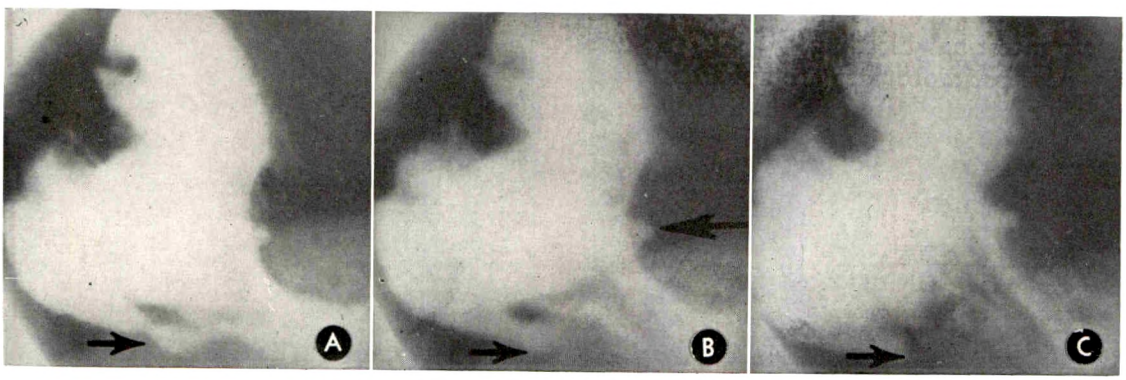


FIG. 5. Case III. (*A–C*) Selected frames from a cineroentgenographic study of the hiatus hernia. Prone right oblique view. Note the prominent folds along the right posterior aspect of the neck and base of the hernia which prolapse into the lumen of the hernia to form the pseudotumor (small arrows). There is a small ulcer at the neck of the hernia on its left anterior wall (large arrow).

the submucosa.[1,4] In our Case 1, large redundant folds were observed through a gastrotomy at laparotomy. In this patient, surgical exploration was prompted by the repeated gastrointestinal hemorrhages and the persistence of the mass-like filling defect in the hiatus hernia. No mass was found at operation because of probable spontaneous reduction of the herniated mucosa during surgery. It is likely, at least in a small proportion of the cases, that the compressed prolapsed mucosa is the source of occult bleeding or even tarry stools, particularly in cases where no other lesion can be detected. This occurred in our Cases 1 and IV.

Cineroentgenographic studies along with meticulous spot filming during fluoroscopy are the most useful methods in the exploration of the nature of the transient or persistent tumor-like filling defects in hiatus hernias.

SUMMARY

Five case reports are presented of large sliding hiatus hernias in elderly females in whom pseudotumors were noted roentgenographically near the neck of the hernias.

These pseudotumors were due to large gastric rugae present in the neck and base of the hernia which prolapsed intermittently into the lumen of the herniated stomach.

The nature of these pseudotumors was best demonstrated on cineroentgenographic studies. It is likely that in 2 of these cases this abnormality was the source of upper gastrointestinal bleeding.

These pseudotumors are apparently not rare, especially if cineroentgenographic studies of large hiatus hernias are performed.

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REFERENCES

1. Brooks, F. M., Stevens, L. W., Pendergrass, E. P., and Bassols, F. Experimental studies on motility of gastric mucosa in dogs. Am. J. Roentgenol. & Rad. Therapy, 1948, *59*, 482–491.
2. de Lorimier, A. A., and Warren, J. P. Prolapse of mucosa at esophagogastric junction. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1960, *84*, 1061–1069.
3. Feldman, M. Retrograde extrusion or prolapse of gastric mucosa into esophagus. *Am. J. M. Sc.*, 1951, *222*, 54–60.
4. Forsell, G. Studies of mechanism of movement of mucous membrane of digestive tract. Am. J. Roentgenol. & Rad. Therapy, 1923, *10*, 87–104.
5. Kaye, J. J., and Stassa, G. Mimicry and deception in diagnosis of tumors of gastric cardia. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *110*, 295–303.
6. Klinefelter, E. W. Invagination of esophagus in hiatus hernia. *Radiology*, 1956, *67*, 562–567.
7. Lame, E. L. Gastric cardia and fundus, extrinsic and benign tissues simulating gastric cancer. *Radiology*, 1960, *75*, 703–711.
8. Lannon, J., and Culiner, A. Retrograde intussusception of lesser curvature of stomach, pylorus and first part of duodenum into esophagus. *Brit. J. Surg.*, 1945, *33*, 392–393.
9. Levin, E. J., and Felson, B. Asymptomatic gastric mucosal prolapse. *Radiology*, 1951, *57*, 514–520.
10. Levine, M., Boley, S. J., Mellins, H. Z., and Schwartz, S. S. Gastrojejunal mucosal prolapse. *Radiology*, 1963, *80*, 30–38.
11. Odegaad, H. Invagination of esophagus in hiatus hernia. *Acta radiol.*, 1959, *51*, 443–448.
12. Palmer, E. D. Mucosal prolapse of esophagogastric junction. *Am. J. Gastroenterol.*, 1955, *23*, 530–537.
13. Sarasin, R., and Hoch, A. Die Invagination der Oesophagusschleimhaut in den Magen. *Schweiz. med. Wchnschr.*, 1951, *81*, 1207–1209.
14. Sarasin, R., and Hoch, A. Les invaginations oesophago-gastriques. *Arch. mal. app. digest.*, 1952, *41*, 434–441.
15. Seaman, W. B. Prolapsed gastric mucosa through gastrojejunostomy. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *110*, 304–313.
16. Walker, B. Q. Variable roentgenographic appearance of invagination of esophagus: based on thirteen cases. *Cleveland Clin. Quart.*, 1966, *33*, 35–37.
17. Wells, J. Herniation of gastric mucosa into esophagus: report of case. Am. J. Roentgenol. & Rad. Therapy, 1947, *58*, 194–195.
18. Wohl, G. T., and Shore, L. Lesions of cardiac end of stomach simulating carcinoma. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1959, *82*, 1048–1057.

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A CRESCENT-SHAPED COLLECTION OF RESIDUAL CHOLECYSTOGRAPHIC CONTRAST MATERIAL: A NEW SIGN OF BENIGN GASTRIC ULCER?*

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DURING the last 7 year period, while performing gallbladder studies which immediately precede many of our gastroduodenal fluoroscopic examinations, I have encountered 8 patients, each of whom had an interesting crescent-shaped collection of residual cholecystographic contrast material in the upper part of the abdomen near the midline or slightly to the left of it. In each case a subsequent barium study showed that this unusual collection of contrast material was trapped in the depths of a large undermined benign ulcer of the gastric antrum, usually on the greater curvature. Unfortunately I have been able to locate the films of only 4 of these 8 patients, the others having been discarded or lost. In the 8 observed cases the presence of a large gastric ulcer was verified during the barium studies of the stomach which were done either immediately after the cholecystographic examination or on the following day.

Although this finding is not very common, it is somewhat intriguing and easily understood. I thought it might be worthwhile to alert radiologic colleagues to this interesting finding, because its presence makes it possible to correctly predict the discovery of a large benign gastric ulcer when the stomach is examined during subsequent barium studies.

THE FIRST ENCOUNTER

Such a crescent-shaped collection of residual cholecystographic contrast material (Telepaque*) was first noted[2] in a patient who had a nonvisualized gallbladder (Fig. 1*A*). During the barium study of the stomach which immediately followed cholecystography the crescent-shaped collection of Telepaque was shown to be located in a large gastric ulcer (Fig. 1, *B–D*) on the greater curvature of the antrum. During the initial part of the fluoroscopic examination with the patient in the erect position, I could not clearly see a communication between the intraluminal barium and the crescent-shaped collection of Telepaque lying inferior to it (Fig. 1*B*). Indeed, I first thought that there was a large intraluminal tumor of the greater curvature side of the antrum above the opaque crescent. However, after some palpation I noted a few streaks of barium extending from the superior margin of the "mass" downward toward the crescent-shaped collection of Telepaque. As the examination progressed the opaque crescent also appeared to become slightly larger and more homogeneous as barium was apparently squeezed into it as a result of my palpation of the barium-filled lumen above it. When the spot films were studied in chronologic sequence it became clear that the crescent-shaped collection of Telepaque and the additional barium were located in the most redundant portion of a large gastric ulcer which was considerably undermined and had enormous overhanging edematous margins (Fig. 1, *B* and *C*).

I postulated that this unusual crater was benign, because these findings would hardly be expected in an ulcerating carcinoma or lymphosarcoma which would classically be manifested by an irregular somewhat

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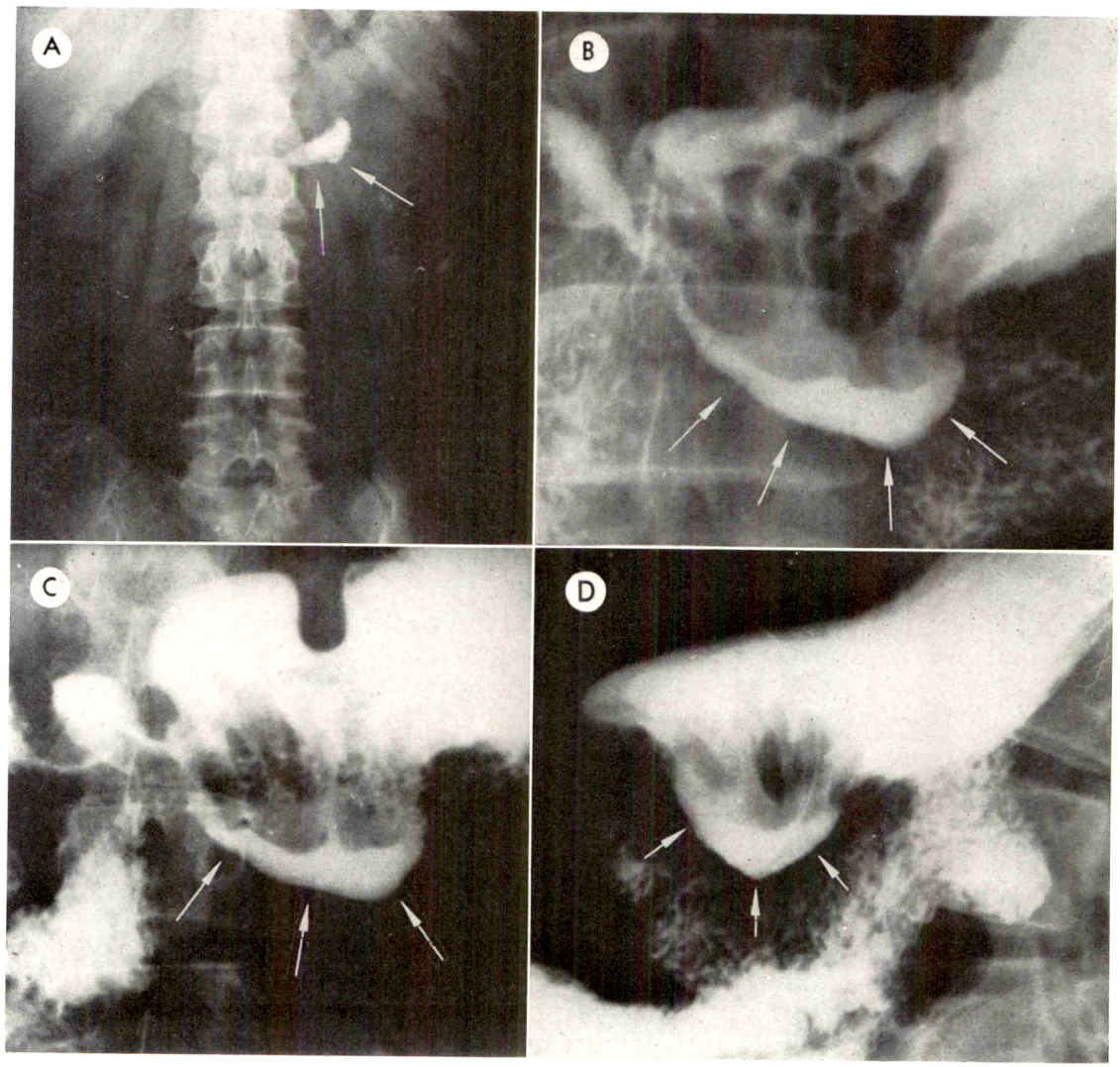


FIG. 1. The crescent sign was first observed in this middle-aged woman with epigastric pain.

(*A*) Cholecystogram shows nonvisualization of the gallbladder and a large crescent-shaped, somewhat obliquely-oriented collection of Telepaque (arrows) near the midline of the upper left quadrant of the abdomen. (*B–D*) Subsequent barium studies of the stomach on the same morning show that liquid barium passed from the antral lumen into the crescent-shaped collection (arrows) via a few faintly-seen streak-like channels which went through the mass.

Note that the tumor-like mass occupied the gastric lumen in both the frontal (*B* and *C*) and the lateral (*D*) projections. This mass of edematous tissue also helps to form the upper, somewhat lobulated margin of the crescent-shaped collection of opaque material in the crater. The crescent shape of the crater in both the frontal and lateral projections is due to the fact that the entire crater is filled with opaque material which is tangentially traversed by the roentgen-ray beam when the patient is recumbent (*A*) or erect (*B–D*).

saucer-shaped crater without evidence of undermining, overhanging edges, or such a high degree of occlusion of the orifice. Even in the more deeply ulcerating leiomyomas or leiomyosarcomas it is not likely that the orifice would be so obstructed or the edges so markedly undermined, these neoplastic ulcers usually being smaller (1 to 2 cm.), more sharply defined, and usually with a patent neck. Conversely, the previ-

ously mentioned huge crater which filled so slowly through the tiny parallel streak-like communications with the lumen indicated that I was dealing with an almost occluded orifice. I thought that this appearance was due to an enormous amount of edematous tissue projecting into the orifice and into the lumen, so as to cause the appearance of neoplasm.

DISCUSSION

Undermining of the edges of a gastric ulcer is considered to be strong evidence in favor of the presence of acid peptic juices which are able to digest the submucosa more rapidly than the more resistant mucosa. This type of submucosal digestion is reliable evidence of benign disease, in contrast to the lack of undermining in the craters which occurs when the central relatively avascular portion of the luminal surface of a carcinoma or sarcoma becomes necrotic. In such tumor tissue there is no submucosa to be destroyed and undermined by the acid peptic juices which, although sometimes present, are usually low or absent. For these reasons ulcerated gastric neoplasms rarely show undermining, although in some the nodular edges of the necrotic shallow crater may occasionally project into the orifice. This lack of undermining also precludes the presence of a crater "neck" which has a diameter smaller than that of the base, as is so frequently seen in the "collar button" shaped benign gastric ulcer. Conversely, the widest part of the crater in the luminal surface of a neoplasm is almost always the part closest to the lumen.

It has been known for many years that an occasional benign gastric ulcer may be surrounded by unusually edematous tissue which sometimes forms a tumor-like mass in which the small crater may not fill, and thus may not be recognized.[1] It is understandable that such a large inflammatory mass might lead the radiologist to diagnose neoplasm, unless he remembers that such an unusual inflammatory lesion can mimic neoplasm. If such an inflammatory mass almost, but not completely, occludes the orifice of a large gastric ulcer, it is understandable that small amounts of ingested cholecystographic contrast material can accumulate in the crater, so that a crescent-shaped collection eventually forms in the most redundant part beneath the overhanging tumor-like mass of edematous tissue. When the patient is in the erect position for long periods of time, gravity probably helps the contrast material enter those craters on the greater curvature of the gastric antrum, even though the orifice is almost occluded by the markedly edematous tissue.

Although the crescent sign is clearly seen when the roentgen-ray beam tangentially traverses the residual contrast material in the inferior portion of such a large benign ulcer located on the greater curvature of the antrum (Fig. 1; 2; and 3), the sign may also occur when there is retention of contrast material in the inferior recess of a large crater on the anterior or posterior wall of the gastric antrum (Fig. 4; 5; and 6). In the latter event any contrast material which enters the narrow orifice would assume a crescent-shaped appearance when the patient is erect, because it would gravitate into the most inferior recess of the undermined portion of such a crater, where it would be manifest as a crescent-shaped collection of contrast material when the crater is viewed *en face* (Fig. 4; 5; and 6). To understand this concept, one need only imagine a hollow lens-shaped container which is only partly filled with an opaque aqueous or viscid contrast material. If the convex surface of this lens-shaped container is directed inferiorly (Fig. 3), a horizontally-directed roentgen-ray beam would show the opaque material to have a convex inferior margin and a fairly horizontal or possibly slightly irregular superior margin quite similar to the appearance of the crescent-shaped collections seen in large ulcers of the greater curvature of the gastric antrum (Fig. 1; and 2). However, if this hollow lens-shaped structure is placed in a vertical position with its surfaces *en face* to

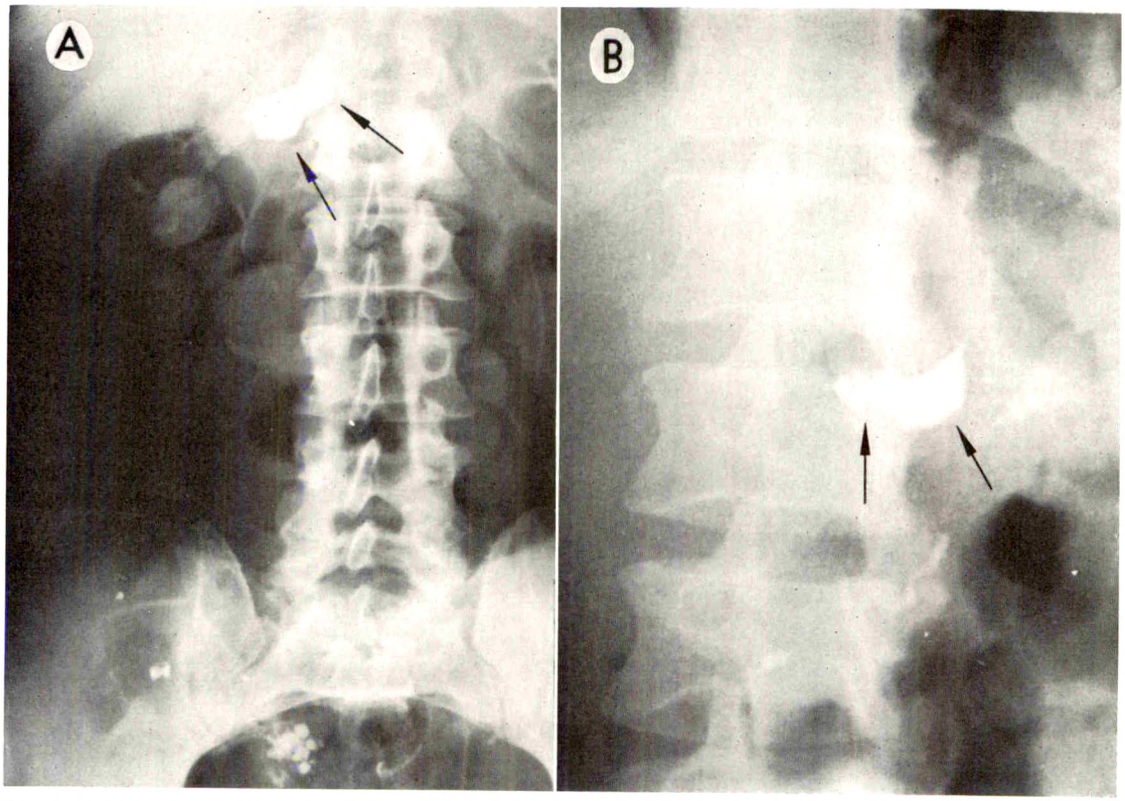


Fig. 2. Elderly man with peptic ulcer history and epigastric pain.

(*A* and *B*) Cholecystograms show faint opacification of the gallbladder and a crescent-shaped, somewhat bilobed collection of Telepaque (arrows) near the midline of the right upper quadrant of the abdomen. In the prone projection (*A*) the collection is obliquely oriented, whereas it is more horizontally oriented in the prone-oblique projection (*B*).

Barium examination of the stomach on the same morning showed a large crater on the greater curvature side of the gastric antrum similar in appearance to that seen in Figure 1.

a roentgen-ray beam (Fig. 6), it would be analogous to a similarly shaped crater located on the anterior or posterior wall of the stomach of an erect patient (Fig. 4; and 5). The opaque contrast material which partly fills such a lens-shaped cavity would gravitate to its most dependent portion, where it would have a convex inferior margin and a straight superior margin. Such a 3-dimensional schematic concept helps to explain the behavior and appearance of the opaque collections of residual contrast material in craters which have this shape (Fig. 6). These concepts were verified during subsequent barium studies of the 4 craters illustrated here. Unfortunately, it was not possible to study the intact fresh specimens immediately after operation in any of the cases.

Orientation of the Crescent-Shaped Collection of Residual Opaque Material

The crescent-shaped collections of cholecystographic contrast material were oriented horizontally with the convexity directed caudally or slightly obliquely with the convexity directed to the left and caudally, the orientation varying slightly with the patient's position during the filming. A horizontal orientation of the retained opaque material would be expected in a gastric ulcer of the greater curvature of the gastric antrum when the patient is in the

erect position and the ulcer is seen in profile (Fig. 1; and 2). However, the crescent-shaped collection of opaque material retained in the most dependent inferior recess of a deeply undermined crater located on the anterior or posterior wall would also be oriented approximately horizontally when the patient is erect (Fig. 4; 5; and 6), according to the previous description of how such collections of contrast material behave in a hollow lens-shaped container.

It is reasonable to ask why the crescent-shaped collections were oriented similarly on the prone and prone-oblique filming during the gallbladder examination and on the earliest erect roentgenograms made during the barium examination of the stomach. In the case of craters located on the anterior or posterior wall, I am certain that the relatively similar orientation is due to the fact that such a crater is usually partly occupied by a mixture of viscid or semisolid material consisting of secretions, fluid, food, and granules of contrast material. Since most of the patients are ambulatory or sitting in bed much of the time prior to the cholecystographic examination, the heavy contrast material which enters the crater would gradually settle to the most dependent portion according to the explanation previously given (Fig. 3; and 6), and the lighter secretions, food, fluid, etc. would occupy most of the available space in the crater above the contrast material. These semisolid materials would be unlikely to change position readily when the patient changes from erect to prone position. By the same token, if the space in a crater

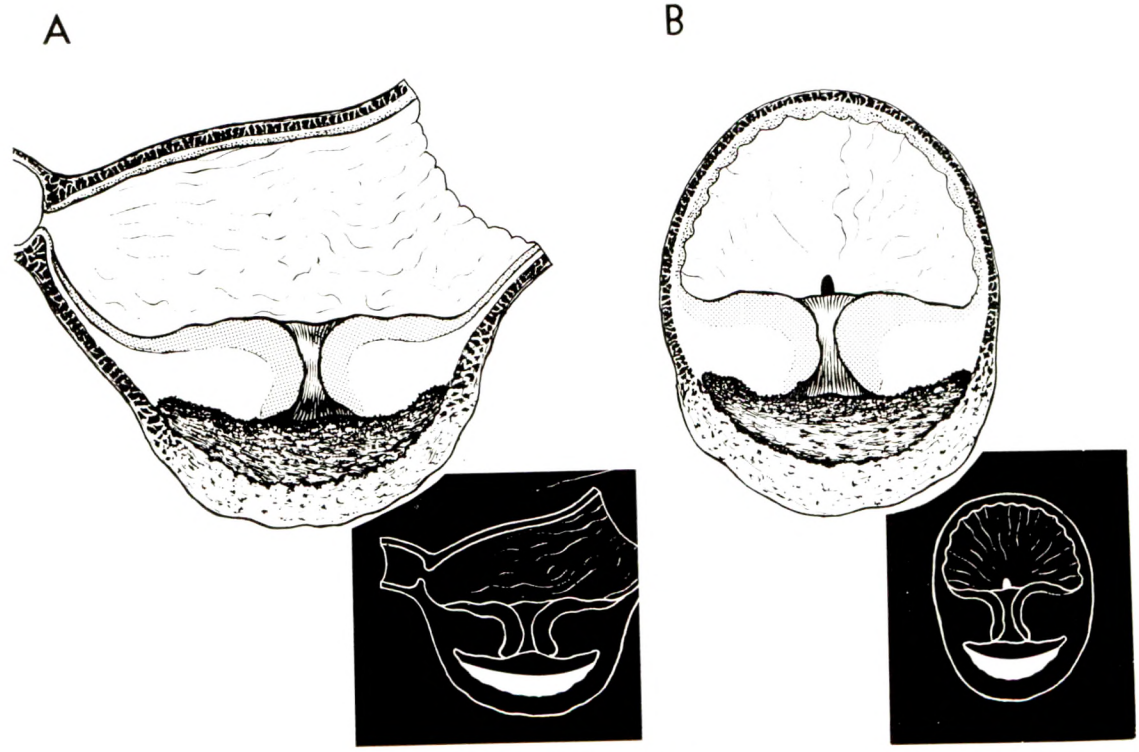


Fig. 3. Cross-sectional drawings of the type of crater illustrated in Figures 1 and 2.

(*A* and *B*) The markedly edematous mucosa (stippled) and submucosa (white) overhang the almost occluded orifice of the crater in the frontal (*A*) and lateral (*B*) projections.

Note the considerable undermining beneath the overhanging edematous submucosa and mucosa. Since the crater is on the greater curvature side, the trapped barium has the same crescent shape in both projections as depicted by the white crescents on the accompanying line drawings.

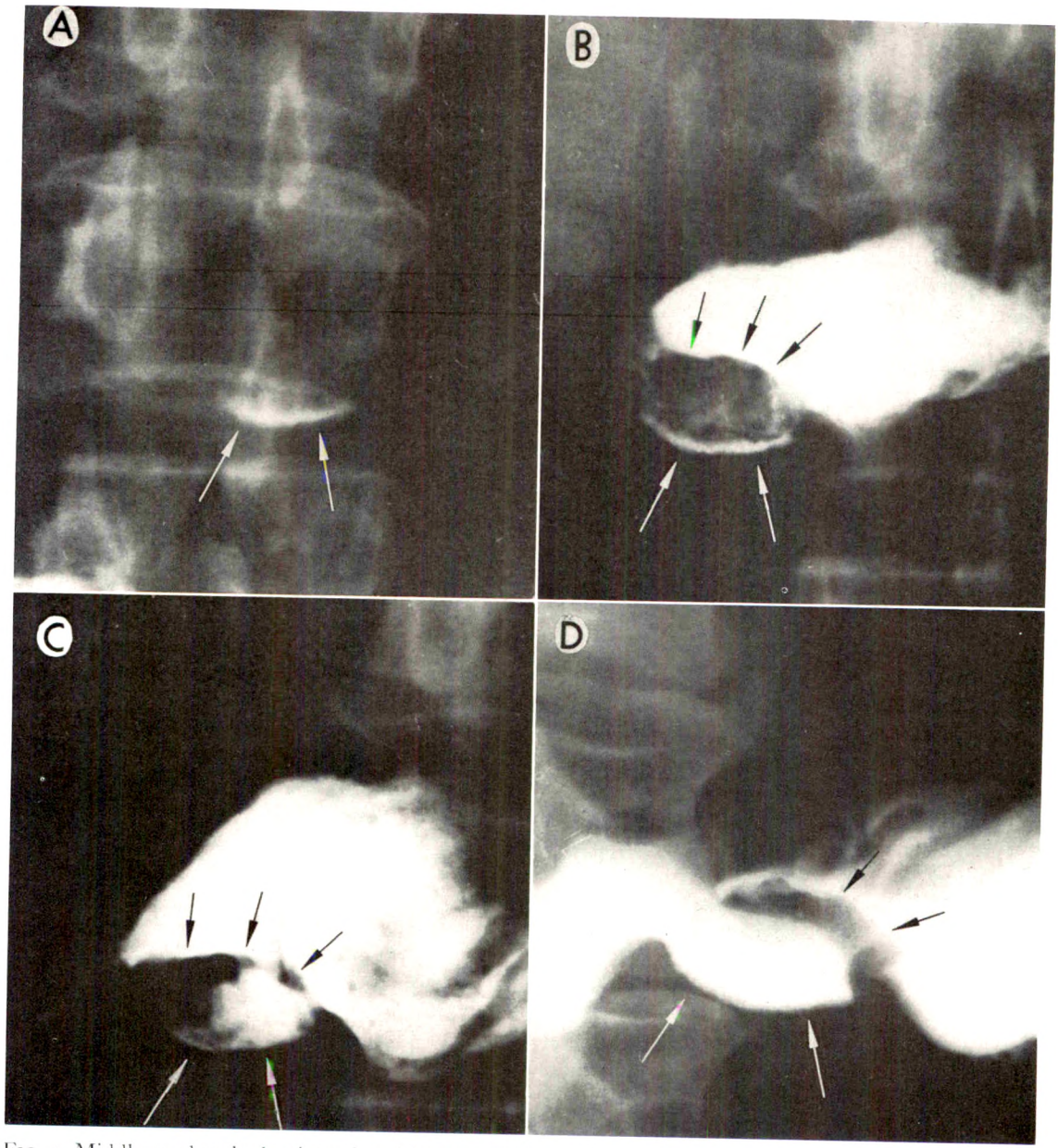


FIG. 4. Middle-aged male dentist referred because of diagnosis of "carcinoma of the gastric antrum."

(*A*) Cholecystogram shows faint visualization of the gallbladder and a thin, horizontally-oriented crescent-shaped collection of Telepaque (arrows) in the upper part of the abdomen near the midline. (*B–D*) Barium study of the stomach on the same morning shows what at first looked like an intraluminal mass (*B*, upper arrows) above the opaque crescent (*B*, lower arrows). The crater (*C* and *D*) slowly filled with barium during palpation. The crescent-shaped collection of Telepaque is in the most inferior recess of an undermined benign gastric ulcer located on the anterior wall of the stomach. This crater is not seen tangentially (as were those in Fig. 1 and 6), but is oriented more in the *en face* projection (see drawing in Fig. 6). The oblique or almost *en face* projection of the crater is best appreciated when the crater is completely filled with barium and Telepaque (*D*).

Note the obliquely-seen cuff of edematous tissue around the crater orifice (*D*, upper arrows) and the tendency of such tissue to simulate an appearance of neoplasm (*B* and *C*).

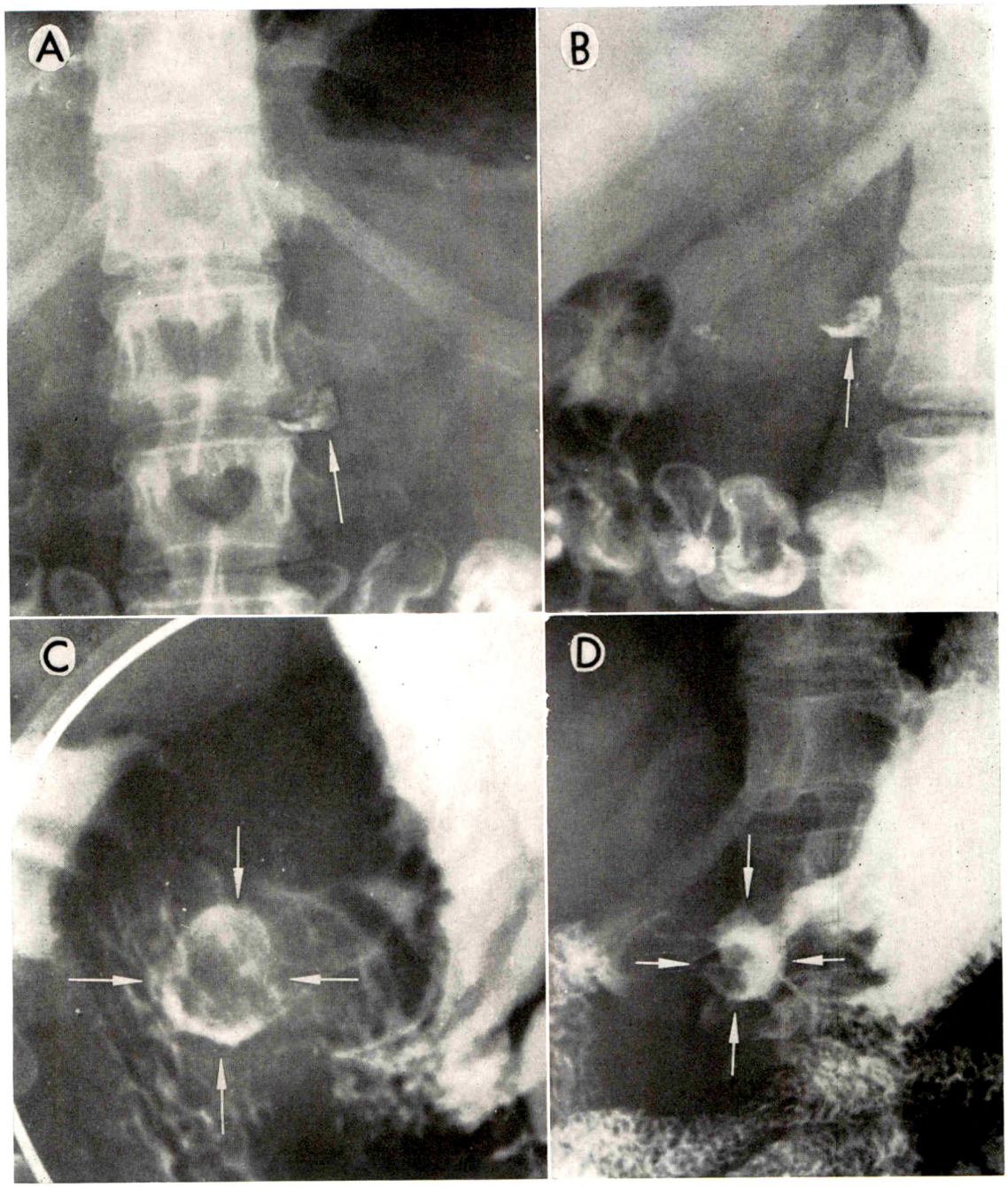


Fig. 5. A middle-aged female with nonspecific epigastric distress.

(*A*) Prone and (*B*) prone-oblique cholecystograms show nonvisualization of the gallbladder and a crescent-shaped, somewhat obliquely-oriented collection of residual Telepaque (arrows) in the left upper abdominal quadrant near the midline. (*C* and *D*) Subsequent barium study on the same morning shows incomplete filling of a crater (arrows) thought to be on the anterior or posterior wall of the gastric antrum. The residual Telepaque can clearly be seen in the most redundant inferior recess of the crater (*C*), before it is obscured by additional barium (*D*).

Note the centrally located radiolucent mass of blood, food, or secretions occupying some of the space in the central part of the crater (*C* and *D*). See Figure 6 for diagrammatic explanation of the crescent-shaped collection of residual Telepaque in the inferior recess of undermined craters which are seen *en face* (as in this case), or partially *en face* (as in Fig. 4), rather than in profile as in Figures 1, 2, and 3.

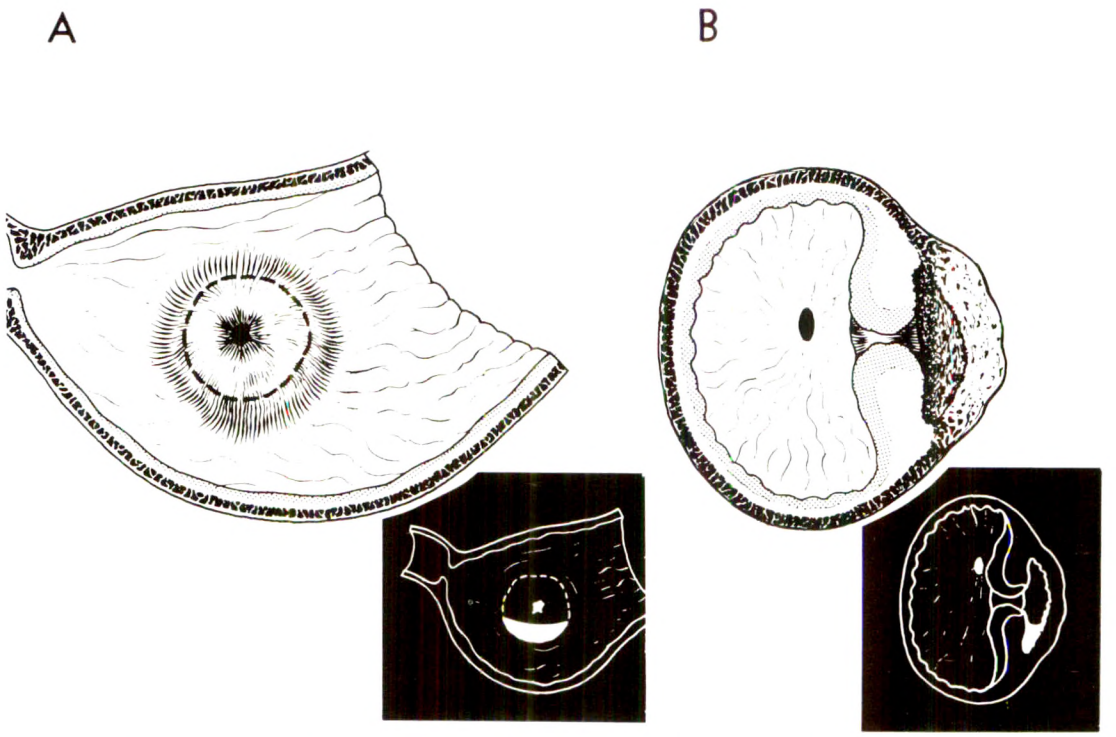


FIG. 6. (*A*) Artist's drawing of *en face* view and (*B*) cross-section in lateral projection of a large undermined ulcer on the posterior wall of the gastric antrum. In the lateral projection (*B*), note the edematous overhanging mucosa (stippled) and submucosa (white).

In the *en face* projection (*A*) the small crater-orifice in the center of the edematous tissue overhangs the crater (dotted circle). Opaque material such as Telepaque or barium which passes through the orifice gravitates to the most inferior recess of the crater, where it can accumulate as an opaque crescent (see white-on-black line drawing in *A*), thus explaining the appearance in Figure 5. It also helps to explain the appearance in Figure 4 in which the Telepaque has gravitated to the most inferior portion of an undermined crater viewed in the oblique projection.

It should be noted that opaque material in such craters viewed in profile on the greater curvature (Fig. 1, 2, and 3) will have a crescent shape in both frontal and lateral projections, whereas the opaque collection seen in the inferior recess of a crater on the anterior or posterior wall will also have a crescent shape when seen *en face*. However, in the lateral projection such an opaque collection in the inferior recess of a crater on the anterior or posterior wall would appear as seen on the white-on-black line drawing in *B*.

located on the greater curvature of the antrum is filled with contrast material and secretions, and if this space happens to be lens-shaped, and if it happens to be seen in profile as is a greater curvature antral crater (Fig. 1; and 2), then the tangentially-seen collection of trapped opaque material will be manifested as a "crescent" when filmed in either the recumbent or erect position. The semisolid or viscid state of the opaque and the nonopaque materials in such ulcers probably explains why the 4 cases illustrated here all showed the crescent sign on the recumbent gallbladder examination as well as on subsequent erect barium studies of the stomach.

THE DIAGNOSTIC SIGNIFICANCE OF THE CRESCENT SIGN

The fact that all of the craters in which this sign was present turned out to be benign was of great interest. The presence of normal amounts of free acid would certainly help explain the unusual degree of submucosal destruction with subsequent undermining of the mucosa so as to cause

such large craters. It is also of interest that the markedly edematous tissue which partly occludes the crater orifice helps to form the inner (luminal) margin of the crescent-shaped ulcer. These findings which are so indicative of benign disease are the distinguishing features of this sign. However, since the vast majority of gastric ulcers (usually over 95 per cent) *in a general* hospital turn out to be benign, the fact that the four illustrated crescent-shaped collections of cholecystographic contrast material happened to delineate large benign ulcers does not have any statistical validity. Admittedly, it is likely that patients with such large gastric craters would have symptoms (all of ours did), and most would, therefore, have barium studies of the stomach and duodenum. The ulcer would thus be diagnosed in most cases, even if the sign had not been noted previously during cholecystography. However, the sign is worth remembering because it is so highly suggestive of a large *benign* gastric ulcer, even though the subsequent barium study of the stomach may be highly suggestive of a neoplasm.

Since absorption of the cholecystographic contrast material would not be expected to occur from the base of such a crater, a poorly or nonvisualizing gallbladder in a patient with a large crescent-shaped collection of residual cholecystographic contrast material should suggest a lack of absorption due to the retention of much of the cholecystographic contrast material in a large gastric ulcer of the type mentioned in this article. As stated previously, these dense concentrated collections of cholecystographic contrast material are not to be confused with the more dispersed collections which are mixed with gastric secretions in patients with gastric outlet obstruction or gastric stasis due to other disease processes not associated with obstruction; *e.g.*, diabetic gastropathy. In the latter type of

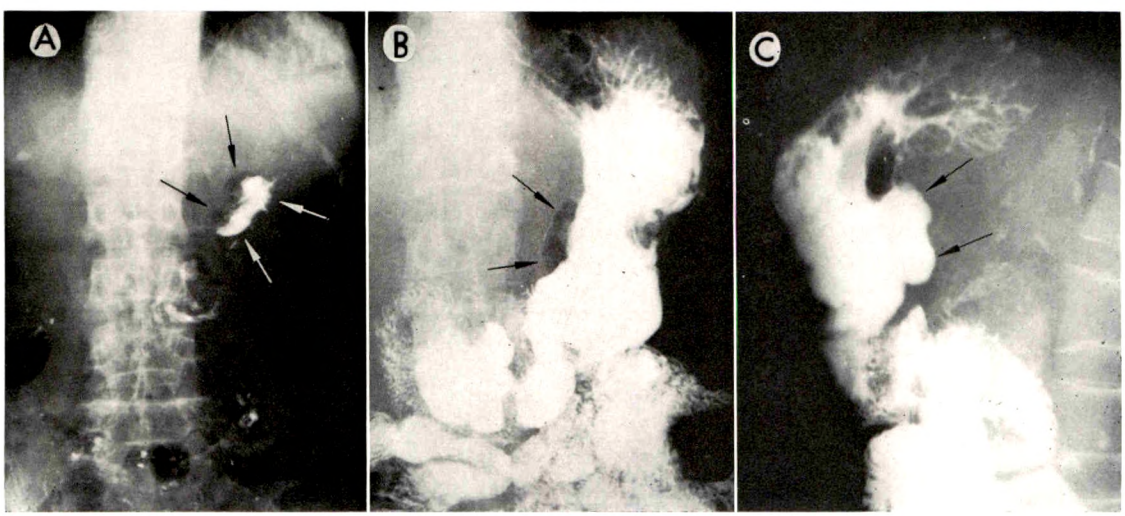


Fig. 7. An elderly lady with weight loss and epigastric distress.

(*A*) Cholecystogram shows nonvisualization of the gallbladder and an irregular and somewhat crescent-shaped, obliquely-oriented collection of Telepaque (arrows) in the left upper quadrant of the abdomen. (*B* and *C*) Subsequent barium examination shows a huge gastric ulcer on the posteromedial wall of the body of the stomach (arrows). In the prone position (*B*) the ulcer is partly filled with gas (arrows), and the lateral roentgenogram (*C*) shows the huge crater filled with barium (arrows).

The orifice of this particular crater was not occluded by edematous mucosa as was true of the previously described craters (Fig. 1–6), and the irregular collection of residual Telepaque is located in the most redundant part of the large crater which is seen primarily *en face* (*A*).

All films were exposed in the recumbent position because of the patient's condition.

retention the residual contrast material is intermixed and scattered heterogeneously throughout the intraluminal contents.

OTHER LESIONS CHARACTERIZED BY CURVILINEAR COLLECTIONS OF POSITIVE CONTRAST MATERIAL

A fifth patient with a large benign gastric ulcer located on the posteromedial wall of the distal portion of the body of the stomach (Fig. 7) had a somewhat irregular hemispheric collection of residual Telepaque on a cholecystogram made immediately prior to the barium study of the stomach. Unfortunately, it was necessary to examine the stomach with this patient in the recumbent position in which the horizontal beam was not used to profile the most dependent portion of the contrast collection tangentially. If the horizontal beam had been used, it is probable that the crescent sign may have been shown. The most redundant part of the crater showed a somewhat lobulated elongated collection of cholecystographic contrast material, whereas the higher recesses of the crater were outlined by gas, the composite outline being that of a somewhat lobulated oval (Fig. 7). The significance of this localized collection of gas and Telepaque was recognized only in retrospect, although the presence of a large gastric ulcer might well have been suspected, since the patient had no previous barium examinations. Under these circum-

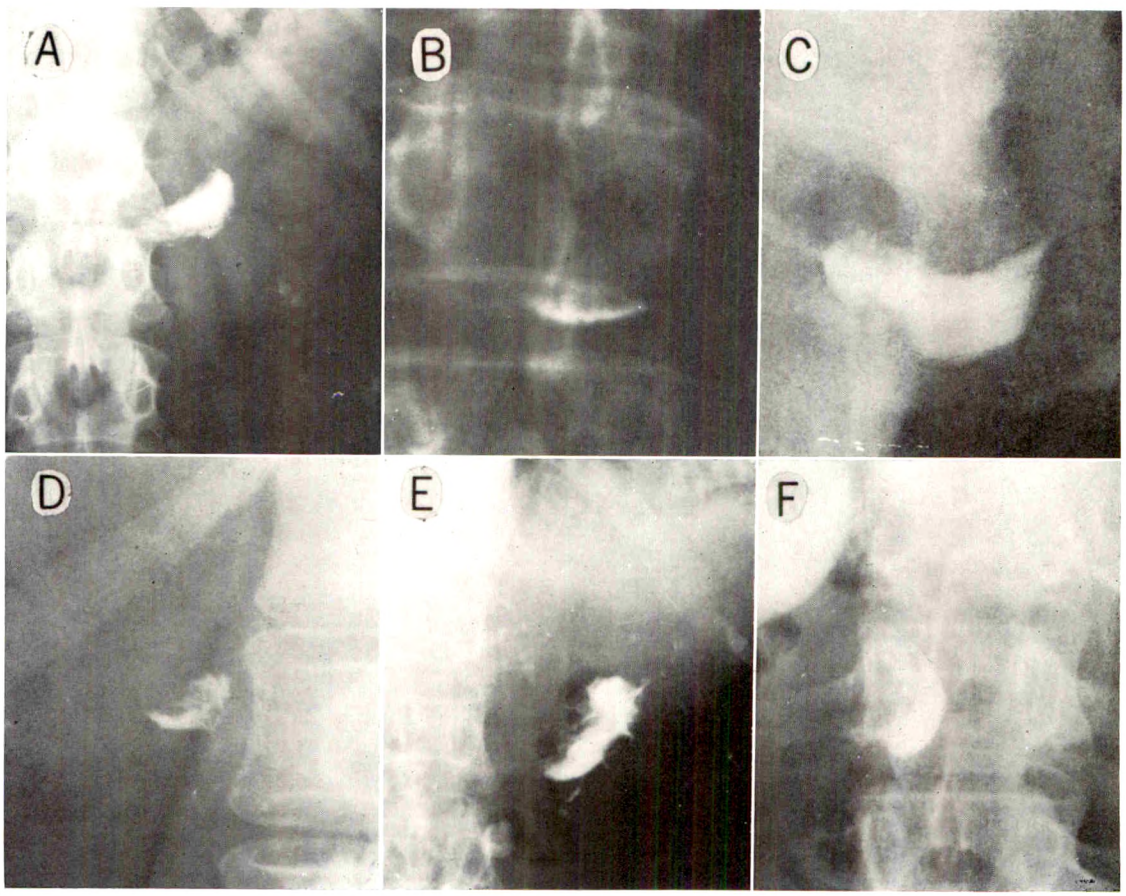


FIG. 8. (*A–D*) Composite illustration to show the appearance of the "crescent sign" in benign ulcers and (*E*) the appearance of residual Telepaque in the large gastric ulcer on the posterior wall. (*F*) The residual Telepaque in the diverticulum of the inner aspect of the second portion of the duodenum is oriented more vertically than the crescent-shaped collections in the benign gastric ulcers (*A–D*).

Barium studies will easily differentiate between a gastric ulcer and a duodenal diverticulum.

stances the presence of such a collection of residual cholecystographic contrast material and gas should have aroused suspicion of a large gastric ulcer.

Occasionally cholecystographic contrast material is retained within a duodenal diverticulum. We have seen 1 patient with a duodenal diverticulum which contained a somewhat crescent-shaped collection of cholecystographic contrast material (Fig. 8*F*). This opaque crescent was more *vertically* oriented than were any of the collections within the gastric ulcers illustrated here (Fig. 8, *A–D*) and was also clearly below the level of the antrum of the horizontally oriented stomach. Subsequent barium study easily differentiated the duodenal diverticulum from a gastric ulcer.

SUMMARY

A crescent-shaped collection of residual cholecystographic contrast material has been encountered in 8 patients, who were subsequently proven to have benign gastric ulcers characterized by marked undermining and overhanging edges of markedly edematous tissue which sometimes virtually occluded the communication between the crater and the gastric lumen.

It is believed that this sign is highly suggestive of a large benign gastric ulcer.

The presence of this sign during cholecystography can alert the radiologist to the presence of a benign gastric ulcer even if the subsequent barium studies show what looks like neoplasm.

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REFERENCES

1. Linsman, J. F. Gastric ulcers simulating intramural extramucosal tumors; report of three cases. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *101*, 421–424.
2. Nelson, S. W. Discovery of gastric ulcers and differential diagnosis between benignancy and malignancy. *Radiol. Clin. North America*, 1969, *7*, 5–25.

HYPOTONIC BARIUM ENEMA EXAMINATION*

By JOSEPH T. FERRUCCI, Jr., M.D.
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SMOOTH muscle spasm is a prominent feature of many disorders of the colon and accounts for a number of difficulties in the roentgenographic diagnosis of colonic disease. Colonic "valves" or "sphincters" have been misinterpreted as stenotic lesions;[3,5] apparent stricture formation in ulcerative colitis has been shown to be merely the result of smooth muscle spasm and hypertrophy.[2] During the performance of a colon examination, patients with inflammatory or functional disease may experience severe abdominal distress forcing premature termination of the enema study and hence an inconclusive result. This is a frequent problem in patients with diverticular disease in whom sigmoid spasm prevents adequate filling of the proximal colon.

Past experience with anticholinergic drugs for hypotonic duodenography has indicated their effectiveness in diminishing smooth muscle tone for more accurate anatomic definition during barium examination of the pancreas and duodenum. Although the use of anticholinergic agents for roentgenographic examination of the colon is well known in Europe, the technique is infrequently mentioned in this country.[1,4]

This report describes the indications and techniques for the use of anticholinergic drugs in roentgenographic examination of the colon.

MATERIAL

The clinical material forming the basis of this report consists of 54 patients examined by hypotonic barium enema in the Department of Radiology at the Massachusetts General Hospital since 1969.

The hypotonic technique was used primarily as a follow-up method in "problem cases" where the initial barium enema study was considered inconclusive or incomplete. A wide variety of clinical situations was encountered in which heightened smooth muscle tone or spasm appeared to contribute to the diagnostic difficulty. These included patients with functional and inflammatory bowel disorders as well as both local and diffuse colonic abnormality.

Table I

INDICATIONS FOR HYPOTONIC BARIUM ENEMA EXAMINATION

1. Diffuse painful spasm
   - Ulcerative or granulomatous colitis
   - Functional disorders
2. Localized narrowing ? spasm *vs* constricting lesion
   - Colonic valves or sphincters
   - Evaluation of strictures in colitis
3. Diverticular disease with sigmoid spasm
4. Reduction of smooth muscle tone for adequate distention during air contrast study
5. Functional inability to retain enema, especially aged patients

The indications for hypotonic barium enema examination are presented in Table 1. In 23 patients (42 per cent) the diagnosis was diverticulosis or diverticulitis of the sigmoid; 10 patients (19 per cent) had chronic ulcerative or granulomatous colitis; and 5 (9 per cent) had primary adenocarcinoma.

In this series all patients were examined using probanthine.* A detailed review of the pharmacology of probanthine, including side effects and contraindications has been presented elsewhere.[1] Major contraindications include glaucoma, prostatism, angina pectoris, and significant cardiac arrhythmias. Side effects are related to dose and route of administration. In this series, 30–45 mg. of probanthine by intramuscular injection was used. Transient side effects of mild tachycardia, blurring of vision, and

*Probanthine, propantheline bromide. G. D. Searle & Co.

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dry mouth may occur. Male patients are instructed to void prior to the procedure to avoid late urinary retention. Where possible, the attending physician should be consulted before administering probanthine. Probanthine is not given to patients who come to the radiology department unaccompanied. Similarly, patients are instructed not to drive after receiving probanthine because of the effects of the drug on visual accommodation of the eyes.

TECHNIQUE

The hypotonic enema examination is performed when the findings on a standard enema examination are inconclusive or when severe painful spasm and involuntary evacuation indicates that repeat of the standard study would be inadvisable.

In patients with severe spasm or localized narrowing, probanthine is administered as soon as the nature of the problem becomes apparent. With the patient remaining on the fluoroscopic table, perhaps partially filled with barium from the initial enema, probanthine is administered intramuscularly, assuming that no medical contraindications exist. Five minutes after administration of the drug the enema examination is resumed and the spastic or

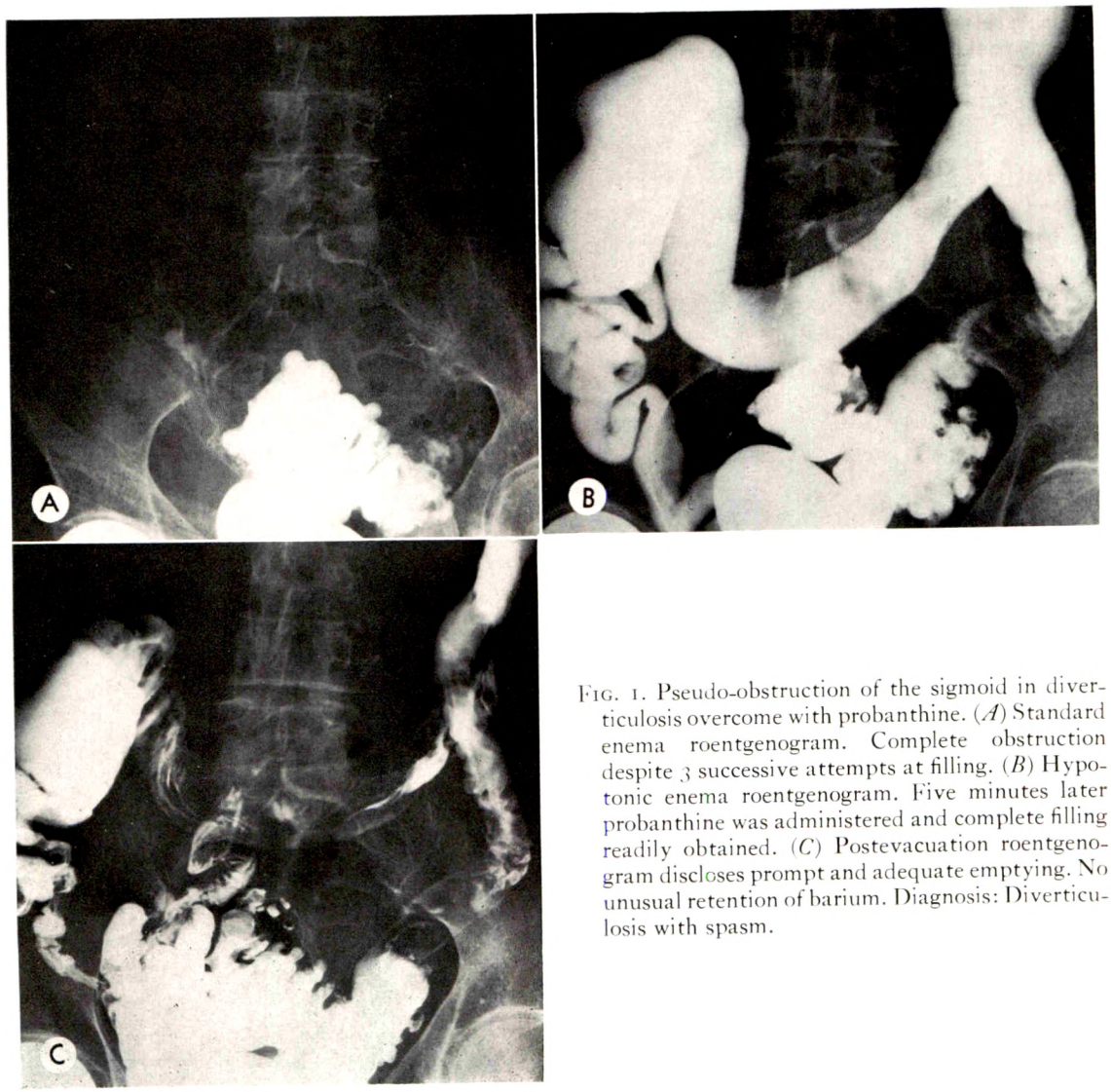


Fig. 1. Pseudo-obstruction of the sigmoid in diverticulosis overcome with probanthine. (*A*) Standard enema roentgenogram. Complete obstruction despite 3 successive attempts at filling. (*B*) Hypotonic enema roentgenogram. Five minutes later probanthine was administered and complete filling readily obtained. (*C*) Postevacuation roentgenogram discloses prompt and adequate emptying. No unusual retention of barium. Diagnosis: Diverticulosis with spasm.

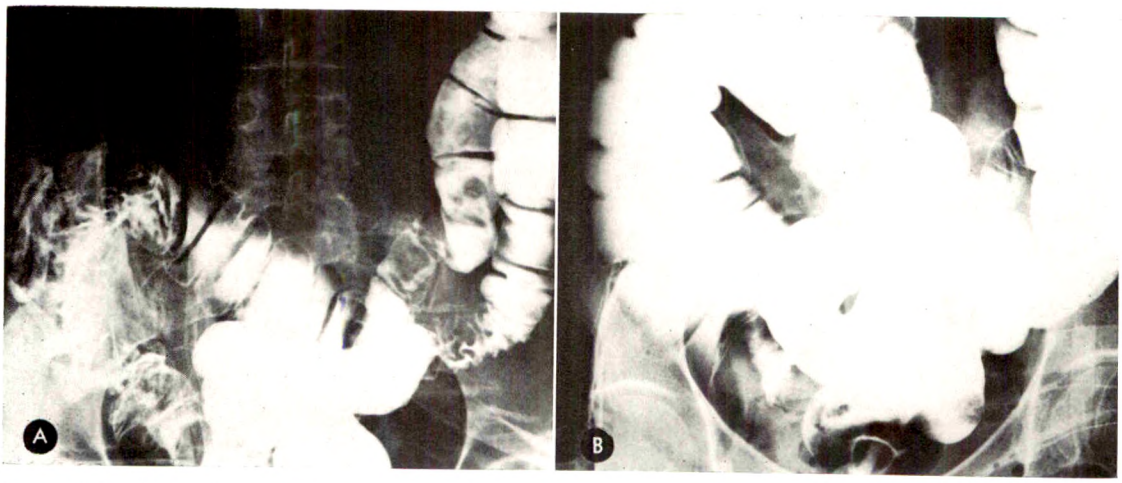


Fig. 2. Nondiagnostic standard enema examination followed by definite probanthine study. (*A*) Standard enema roentgenogram. Involuntary evacuation occurred before adequate fluoroscopic or roentgenographic study could be done. A cecal mass is suggested. (*B*) Hypotonic enema roentgenogram. One day later complete filling is accomplished after administration of probanthine, and a cecal lesion is confirmed. Diagnosis: Carcinoma of the cecum.

narrowed segment is observed fluoroscopically for the onset of relaxation. The enema examination is then completed in the usual manner. Recovery of colonic muscle tone occurs within 15–30 minutes and evacuation then proceeds in normal fashion.

Even patients with marked delay or apparent obstruction to retrograde filling associated with diverticular disease of the sigmoid generally demonstrate satisfactory colonic evacuation (Fig. 1, *A–C*). To date, no instance of prolonged barium retention or impaction has occurred. However, barium should not be allowed to overfill the hypotonic colon proximal to a rigid unyielding segment, since theoretically there would be a greater risk of inspissation of barium within the proximal portion of the colon.

When vigorous preparation of the colon is employed prior to an air contrast study, probanthine can be used to control resulting spasm. In this case, the drug is administered immediately prior to the procedure and filling is carried out in the usual manner.

Results and Discussion

In this selected group of "problem" examinations of the colon, the probanthine enema reduced patient discomfort or improved the quality of the study in the majority of cases. For the elderly patient with painful spasm or inability to retain the standard enema, the probanthine study affords a well-tolerated method to obtain a colonic study of diagnostic quality (Fig. 2, *A* and *B*).

In many instances an inconclusive study was salvaged and a specific diagnosis obtained. For example, when diverticulitis is suspected in an area of sigmoid spasm, the spasmolytic effect may permit sufficient relaxation so that microperforations fill, thus permitting a firm roentgenographic diagnosis (Fig. 3, *A* and *B*). Patients whose initial examination of the colon cannot be completed because of painful spasm may be saved a return visit on a later day. In view of the frequency with which diverticulosis of the sigmoid is associated with severe spasm and pseudo-obstruction, this is a significant consideration.

Most patients with diverticular disease do exhibit some response to probanthine. Nonetheless, interpretation of a hypotonic enema examination is determined by the anatomic findings rather than the response to the spasmolytic drug.

A distinct response to probanthine with

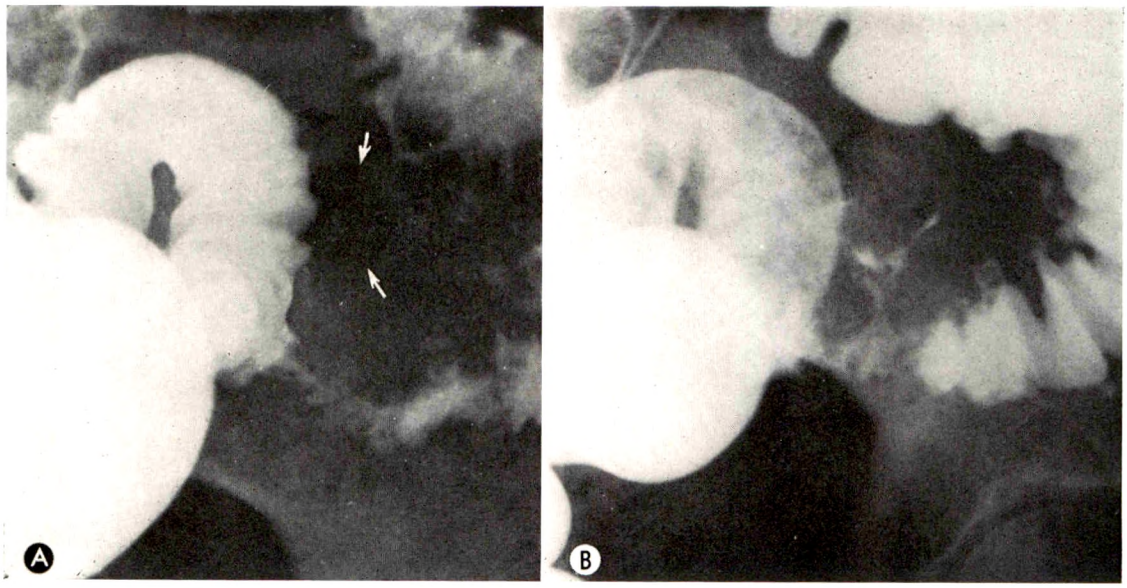


Fig. 3. Hypotonic enema may permit a firm diagnosis in suspected diverticulitis. (*A*) Standard enema roentgenogram. A stenosing lesion of the sigmoid with thickened mucosal folds and partial obstruction is demonstrated. A few diverticula are present and a small collection of gas is visible above the narrowed area (arrows). A definite diagnosis is not possible. (*B*) Hypotonic enema roentgenogram. Five minutes later probanthine study shows the segment to be distensible; barium fills a sinus tract and gas-containing abscess cavity. Diagnosis: Acute diverticulitis with abscess formation. (Reproduced with permission of *Radiol. Clin. North America*.[4])

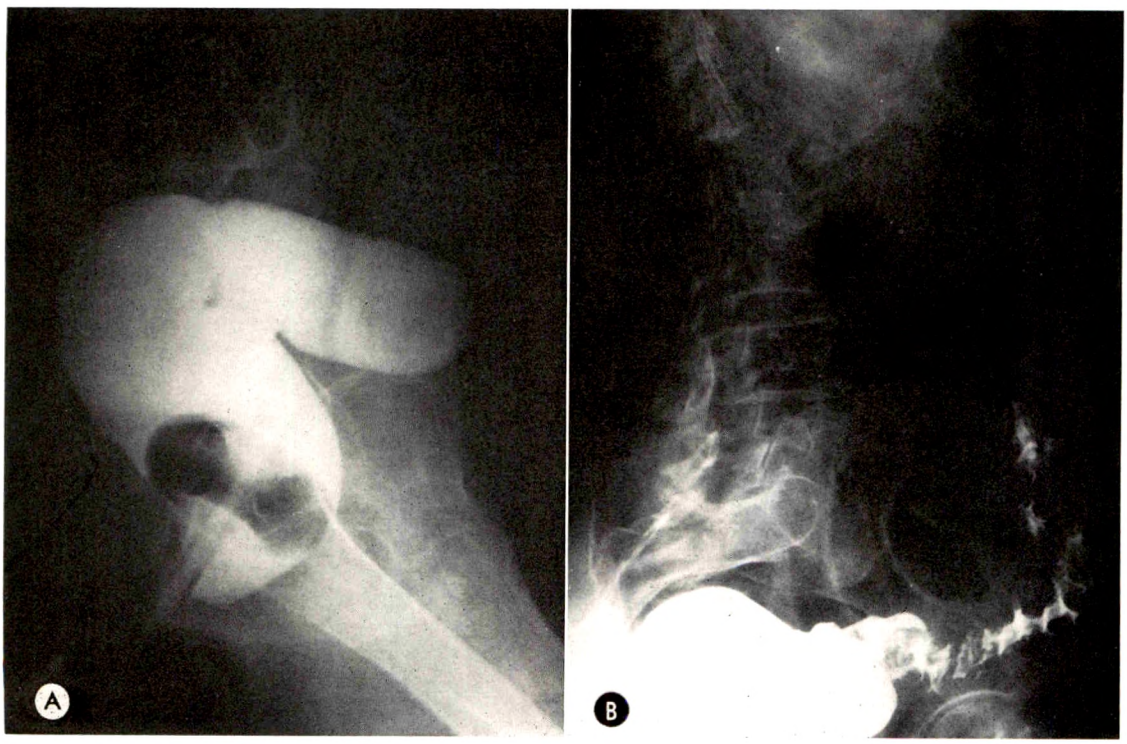


Fig. 4. Response to probanthine does not distinguish tumor from benign disease. (*A*) Standard enema roentgenogram. Complete obstruction of the sigmoid colon. (*B*) Hypotonic enema roentgenogram 1 day later. The stenotic segment distends; however, a distal shelf is suggested. Diagnosis: Carcinoma of the sigmoid.

distention of a narrowed segment is reassuring, but is of limited value in distinguishing carcinoma from benign disease. Of the 23 patients with diverticular disease, 4 (17 per cent) showed no significant response to the administration of probanthine. On the contrary, an obstructing carcinoma may show a definite response possibly related to associated smooth muscle spasm (Fig. 4, *A* and *B*). Consequently, residual anatomic abnormality persisting after probanthine must be evaluated according to conventional roentgenographic signs.

SUMMARY

Smooth muscle spasm accompanies a variety of colonic disorders, and during colonic examination may cause significant abdominal distress, as well as an incomplete or inconclusive study.

Probanthine-aided examination of the colon is of value whenever smooth muscle spasm is encountered. This technique was used in 54 patients with inflammatory and functional bowel disease, whose initial barium enema examination was inconclusive or incomplete. Using this method in "problem" studies, it was possible to reduce patient discomfort, salvage unsatisfactory studies, and increase diagnostic accuracy in a broad spectrum of colonic disorders.

The technique is consistently useful in diverticular disease of the sigmoid and is particularly applicable to the large number of patients with partial sigmoid obstruction secondary to diverticulosis with spasm.

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REFERENCES

1. Ferrucci, J. T., Jr, and Benedict, K. T., Jr. Anticholinergic-aided study of gastrointestinal tract. *Radiol. Clin. North America*, 1971, *9*, 23–29.
2. Goulston, S. J., and McGovern, V. J. Nature of benign strictures in ulcerative colitis. *New England J. Med.*, 1969, *281*, 290–295.
3. Janower, M. L. X-ray seminar No. 39. Vanishing lesion of colon. *J.A.M.A.*, 1964, *189*, 942–943.
4. Margulis, A. R., and Goldberg, H. I. Current state of radiologic technique in examination of colon: survey. *Radiol. Clin. North America*, 1969, *7*, 27–42.
5. Templeton, A. W. Colon sphincters simulating organic disease. *Radiology*, 1960, *75*, 237–241.

HYPOTONIC DUODENOGRAPHY IN THE EVALUATION OF CHOLEDOCHOLITHIASIS AND OBSTRUCTIVE JAUNDICE*

By ROBERT L. BREE, M.D.,† *and* ROBERT E. FLYNN, M.D.
PHOENIX, ARIZONA

HYPOTONIC duodenography in the evaluation of the head of the pancreas has been established as a useful method in diagnosis.[1,2,4] In recent years, evaluation of the duodenum itself has been described, especially in relation to postbulbar ulceration and stricture.[2] The normal anatomy of the perivaterian area of the duodenal loop has been very well outlined by Ferrucci *et al.*,[6] and with this normal anatomy as a guide, we have found it possible to detect impacted common bile duct stones by hypotonic duodenography. In several reports, patients with choledocholithiasis have been described with the typical findings of smooth enlargement of the papilla of Vater.[4,6,7,11–13]

It is the purpose of this report to present a series of patients with choledocholithiasis diagnosed or confirmed by hypotonic duodenography. Examples of carcinoma of the papilla of Vater and carcinoma of the head of the pancreas are also included as differential possibilities.

MATERIAL AND METHOD

During the past 6 months, selected patients with obstructive jaundice were studied with hypotonic duodenography.

All of the patients presented with signs of obstructive jaundice. In several patients elevation of several liver enzyme values made this conclusion equivocal.

Hypotonic duodenography was performed, using the tube method, with intramuscular injection of 30 to 60 mg. of propantheline bromide (Pro-Banthine).

In 8 patients, enlargement of the papilla of Vater was identified, based on the criteria of Ferrucci *et al.*[6] In these patients, the roentgenographic image of the papilla measured at least 2.0 cm. in greatest diameter. One case, interpreted as papillary edema, was found to have carcinoma of the head of the pancreas. A second case with a 5.0 cm.

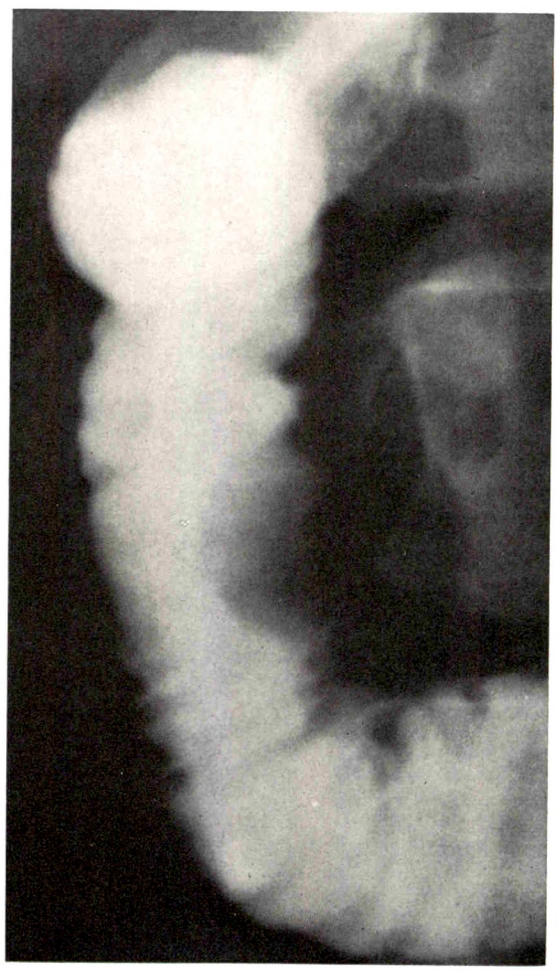


FIG. 1. Case 1. Hypotonic duodenogram with barium contrast demonstrates markedly enlarged papilla of Vater. The enlargement is smooth, and the size is 3.2×2.0 cm. A large stone is impacted in the distal common duct.

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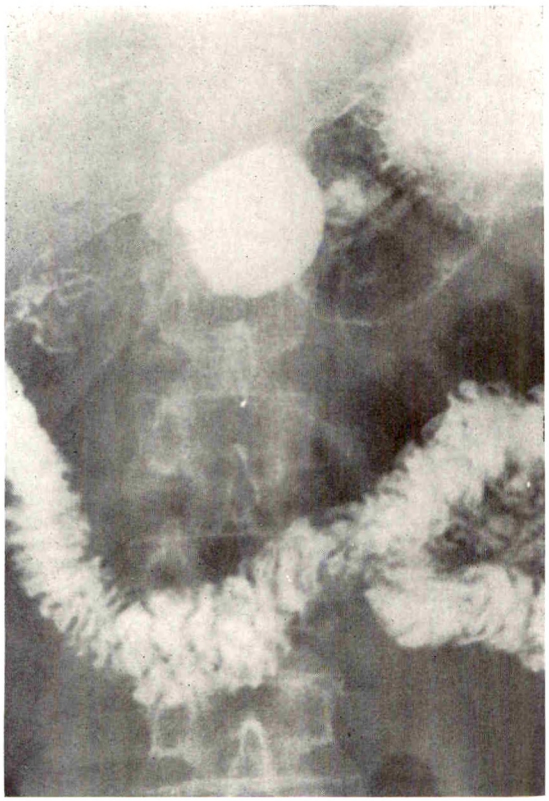


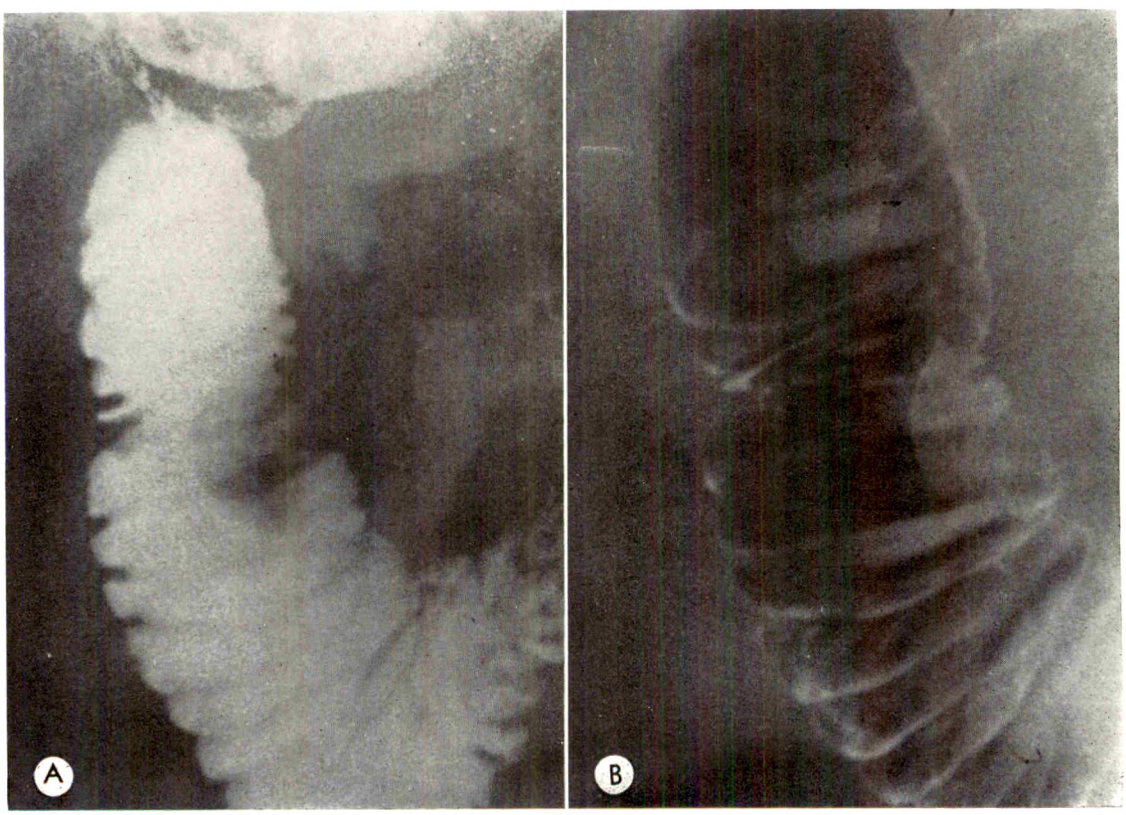
Fig. 2. Case II. Upper gastrointestinal roentgenogram showing no definite abnormality.

papilla was interpreted as representing either edema or neoplasm. This was a carcinoma of the papilla of Vater. There was 1 false negative: a patient with a stone impacted high in the common duct and a stricture of the ampulla.

REPORT OF CASES

Case 1. A 58 year old man was admitted to the hospital with complaints of dark urine, yellow skin and light stools for 3–4 weeks. Physical examination was unremarkable, except for scleral icterus. The alkaline phosphatase was 154 units. Bilirubin was 2.2 mg. per cent, and SGOT was 52 units.

Hypotonic duodenogram showed a very large papilla of Vater measuring 3.2 cm. × 2.0 cm. (Fig. 1). The enlargement was smooth.



At surgery, a large impacted stone was removed from the common bile duct, and because of the severe papillary edema, a sphincterotomy was performed.

Case II. A 60 year old man was admitted to the hospital with a history of jaundice for 1 month. He complained of dark urine, light stools, joint pain and itching. Bilirubin was 15.5 mg. per cent. Alkaline phosphatase was 250 units and SGOT was 385 units. A rose bengal liver scan showed no intestinal activity. A liver biopsy demonstrated cholestasis. Upper gastrointestinal series was interpreted as normal (Fig. 2).

Hypotonic duodenogram showed marked

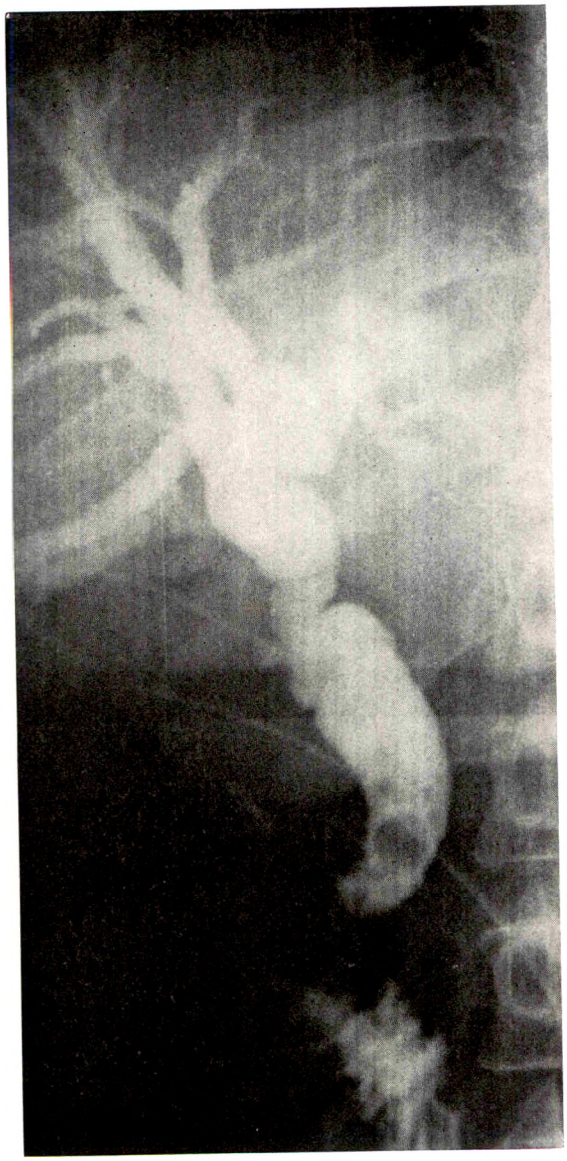


Fig. 4. Case II. Surgical cholangiogram shows choledocholithiasis with incomplete obstruction.



Fig. 5. Case II. Following removal of stones, there is patency of the ampulla with continued evidence of edema of the papilla.

Fig. 3. Case II. (*A*) Barium filled duodenum demonstrates enlarged papilla. (*B*) Air contrast roentgenogram further delineates the smooth mass measuring 2.4×1.5 cm.

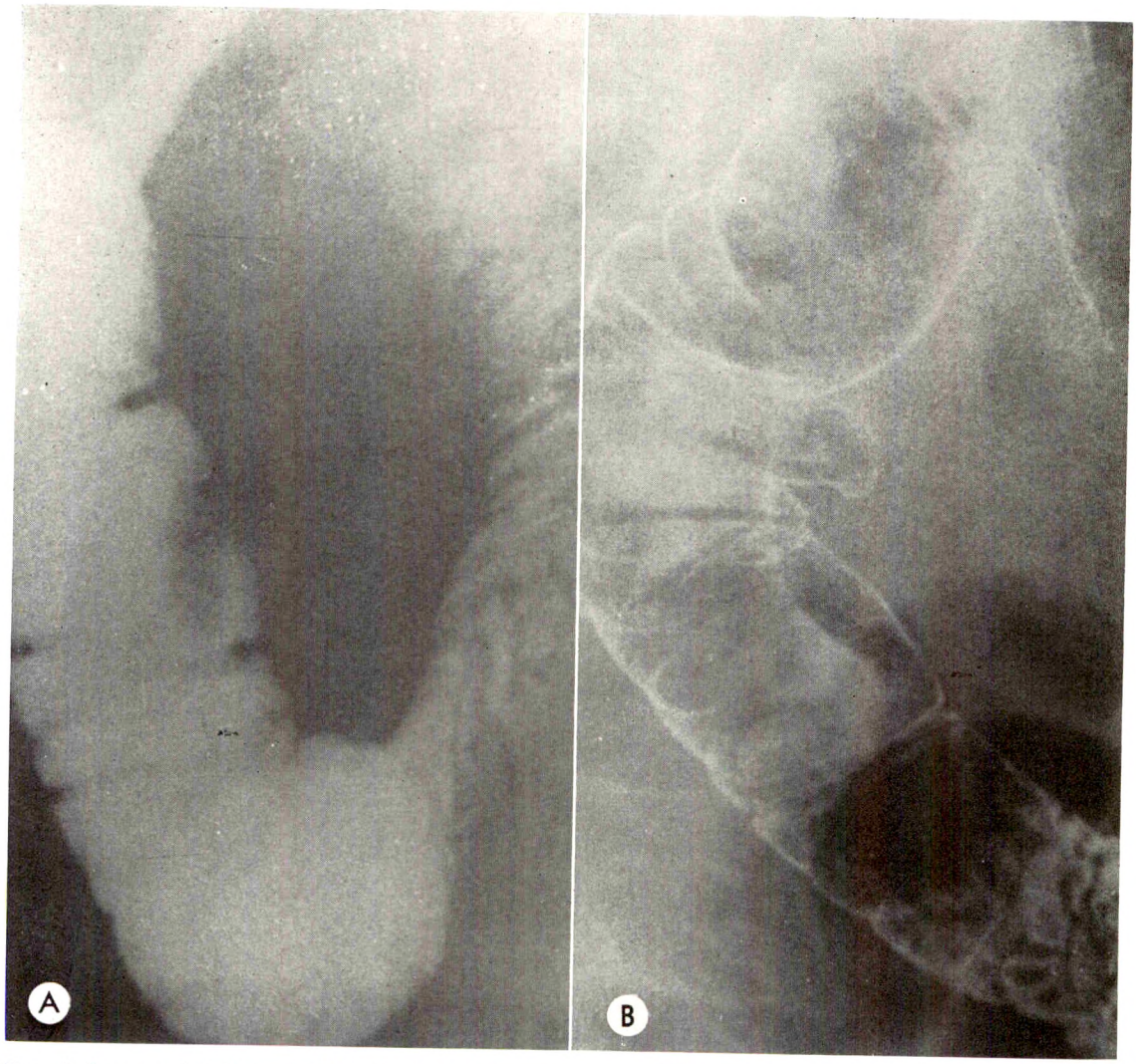


Fig. 6. Case III. (*A*) Barium filled duodenum demonstrates enlarged papilla and longitudinal fold. (*B*) Air contrast examination shows the smooth filling defect, which is elongated, indicating considerable edema of the longitudinal fold. A duodenal diverticulum is present.

smooth enlargement of the papilla of Vater. The dimensions of the papilla were 2.4×1.5 cm. (Fig. 3, *A* and *B*). An operative cholangiogram demonstrated multiple common duct stones and obstruction (Fig. 4).

Following removal of the stones, there was decompression of the duct and free flow of contrast material into the duodenum. The edematous papilla was again well seen (Fig. 5).

Case III. A 64 year old man was admitted with a history of jaundice for 2 months. He complained of indigestion and fatty food intolerance. He had dark urine and light stools. There was a recent 20 pound weight loss. Bilirubin was 19 mg. per cent, with 16.2 mg. per cent direct. SGOT was 130 units and alkaline phosphatase was 260 units. A working diagnosis of carcinoma of the pancreas was made.

Hypotonic duodenogram showed a moderately enlarged papilla measuring 2.4×1.0 cm. A large portion of this image was felt to be edema of the longitudinal fold (Fig. 6, *A* and *B*). Transhepatic cholangiogram showed a markedly dilated biliary tree and multiple large stones in the common bile duct with impaction at the distal end (Fig. 7, *A* and *B*).

At surgery, the stones were removed, but the patient expired following surgery from hepatorenal failure.

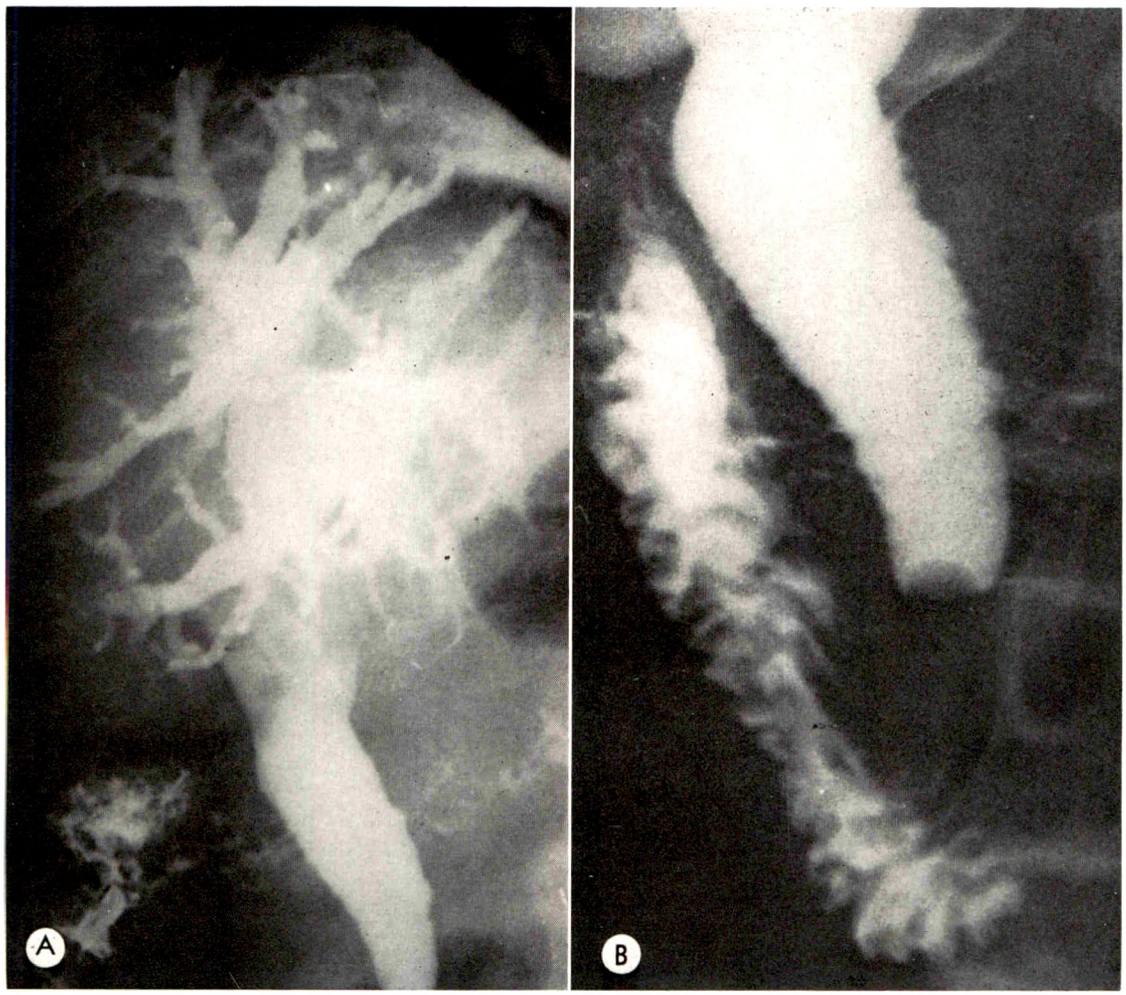


Fig. 7. Case III. (*A*) Transhepatic cholangiogram with markedly dilated biliary tree and multiple stones in the common duct. (*B*) View of the distal common bile duct with contrast material in the duodenum showing meniscus of impacted stone and relationship to the papilla.

Case IV. A 52 year old female was admitted for evaluation of progressive jaundice and abdominal pain. Bilirubin was 1.0 mg. per cent but there was history of recent jaundice. Alkaline phosphatase was 360 units and SGOT was 120 units. An intravenous cholangiogram demonstrated incomplete obstruction of the common bile duct with the point of obstruction not identified.

Hypotonic duodenogram demonstrated a large defect in the region of the papilla of Vater, which was interpreted as the papilla. The defect measured 3.5×1.0 cm. (Fig. 8, *A* and *B*).

At surgery, a large carcinoma of the head of the pancreas was found, which was partially obstructing the common bile duct.

Case V. A 63 year old female was admitted to the hospital with a chief complaint of epigastric pain. She noticed dark urine for 1 day. She had a cholecystectomy 5 years previously. Her bilirubin was 4.0 mg. per cent with 2.8 mg. per cent direct. Alkaline phosphatase measured 350 units. SGOT was 180 units.

Hypotonic duodenogram demonstrated an intramural mass on the medial wall of the duodenal loop at the junction of the second and third portions measuring 2.0×2.0 cm. (Fig. 9). Because of the history, this was felt to be the papilla of Vater in a lower position than normal.

At surgery, an impacted common duct stone was found.

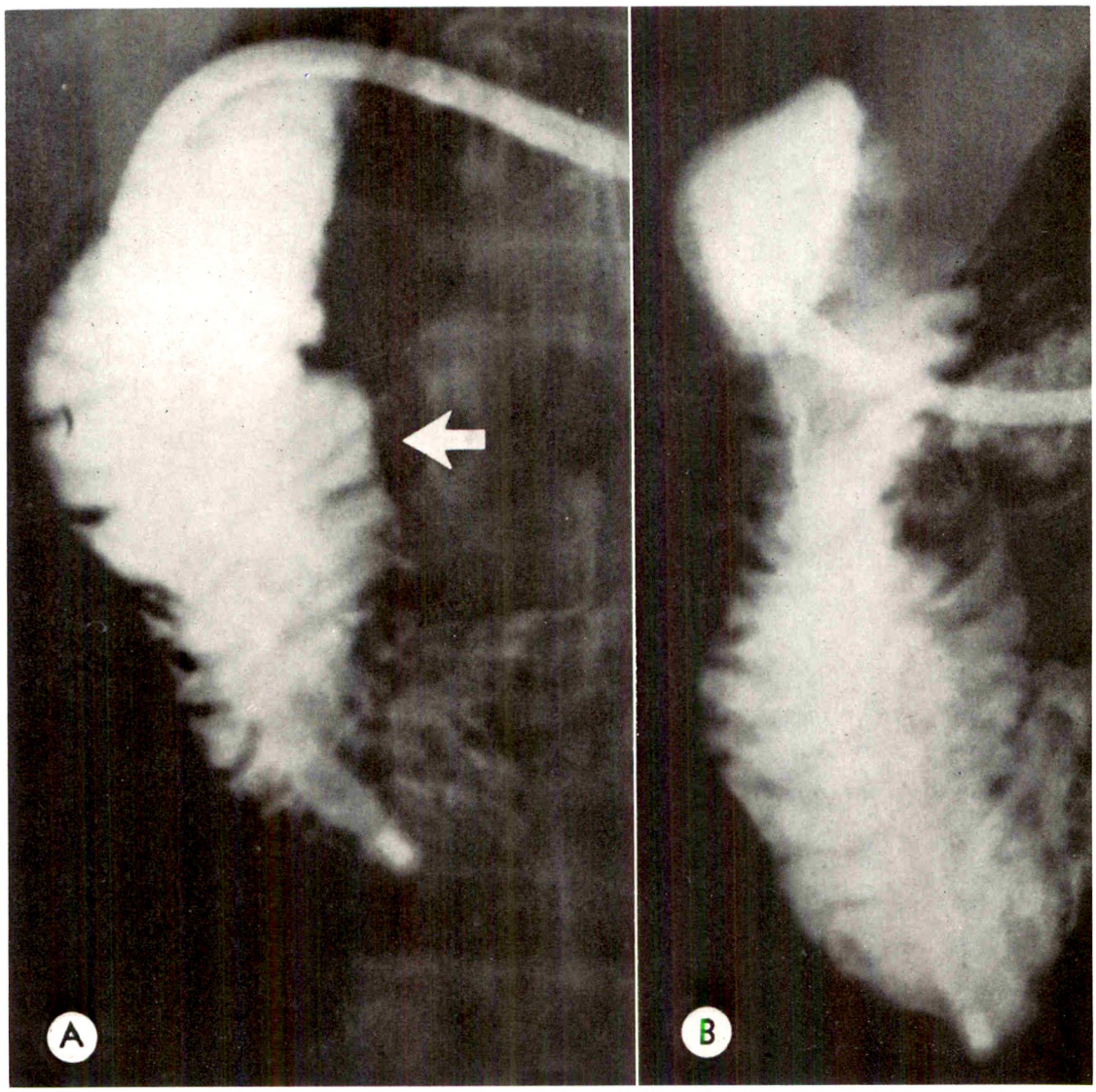


Fig. 8. Case iv. (*A* and *B*) Barium filled duodenum demonstrates filling defect below the promontory in the region of the papilla of Vater (arrow). The folds appear normal. At surgery, a carcinoma of the head of the pancreas was found.

A postoperative cholangiogram confirmed the low position of the ampulla of Vater (Fig. 10).

Case vi. A 68 year old Chinese man was admitted with a chief complaint of chronic epigastric pain. Upper gastrointestinal series was normal. The gallbladder did not visualize on oral cholecystography. Dark urine and light stools were noted. Initial bilirubin was 19 mg. per cent, but after about 6 to 7 days, it dropped to a level of 1.9 mg. per cent. SGOT measured 34 units and alkaline phosphatase 161 units. Intravenous cholangiogram demonstrated a large obstructing calculus high in the common duct (Fig. 11).

Hypotonic duodenogram demonstrated a moderately enlarged papilla measuring 3.0×1.0 cm. (Fig. 12). Because of the changing level of jaundice it was felt that the calculi were initially impacted, but the impaction was subsequently relieved.

At surgery, multiple calculi were removed from the common bile duct, the largest measuring 3.0 cm.

Case vii. A 72 year old man was admitted

to St. Joseph's Hospital with a history of having had a recent cholecystectomy, but because of associated pancreatitis, a T-tube was placed in the common bile duct without exploration. Following surgery, the patient remained jaundiced. A cholangiogram demonstrated a large calculus at the distal common bile duct (Fig. 13).

Hypotonic duodenograms were obtained to evaluate the size of the papilla. The papilla was markedly edematous, measuring 3.7×2.2 cm. (Fig. 14, *A* and *B*).

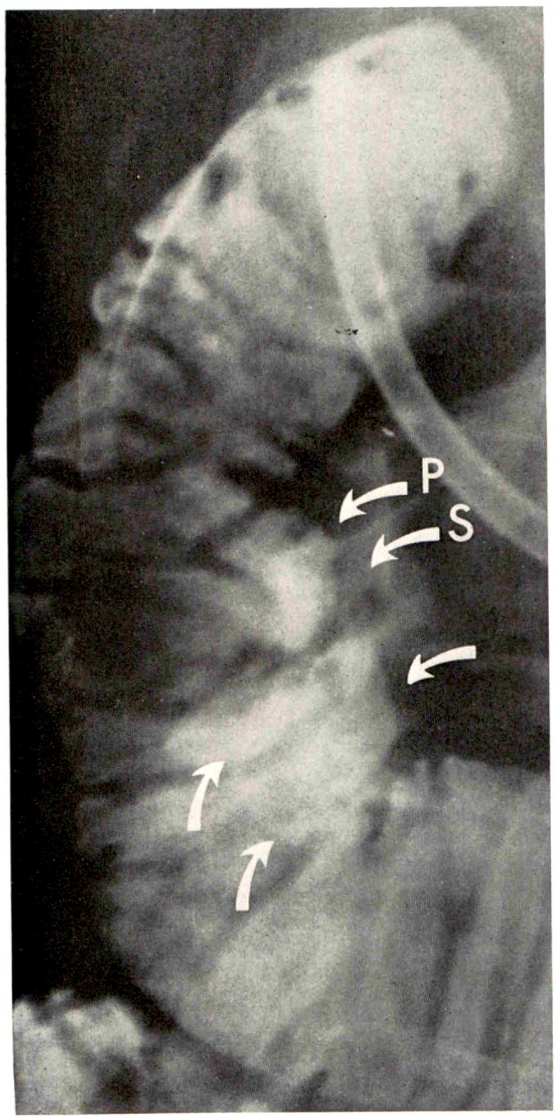


Fig. 9. Case v. Air contrast duodenogram shows promontory (P) and straight segment (S). A 2.0 ×2.0 cm. filling defect below the straight segment (arrows) was found to be the papilla. At surgery a common duct stone was found.

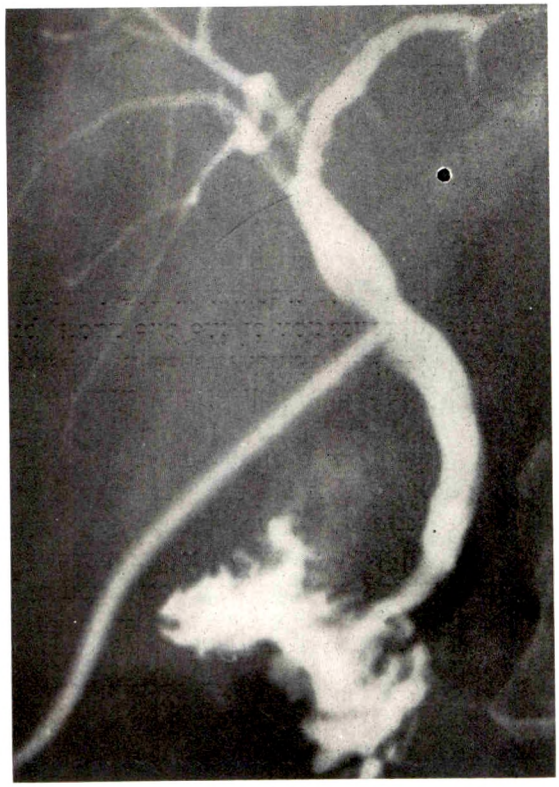


Fig. 10. Case v. Postoperative cholangiogram demonstrates low insertion of the common bile duct.

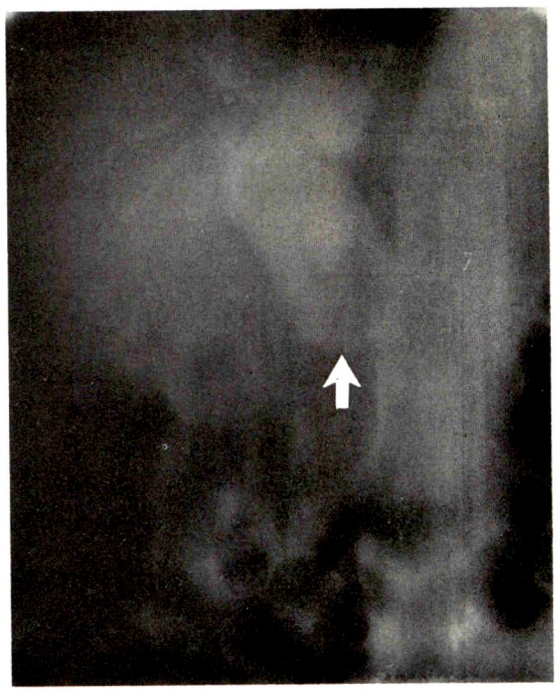


Fig. 11. Case vi. Intravenous cholangiogram shows dilated common bile duct with large obstructing calculus (arrow).

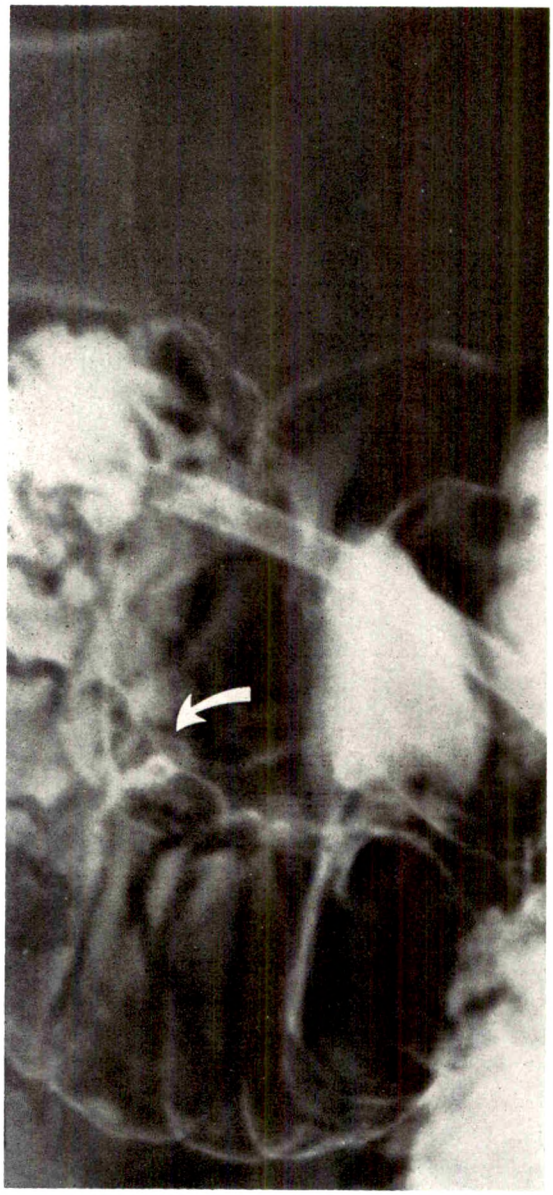


Fig. 12. Case vi. Air contrast duodenogram shows enlarged papilla measuring 3.0×1.0 cm. (arrow). Multiple stones were found in the duct.

Case viii. A 28 year old man presented to the hospital with severe jaundice and epigastric pain. Bilirubin was 12.0 mg. per cent, alkaline phosphatase was 270 units, and SGOT 450 units.

Hypotonic duodenogram was normal (Fig. 15). A transhepatic cholangiogram demonstrated a stone impacted high in the common bile duct.

At surgery, only 1 stone was found, and the ampulla was extremely stenotic. A choledochoduodenostomy was subsequently performed.

Case ix. A 50 year old woman presented to the hospital with a history of recent onset of jaundice. Bilirubin was 7.0 mg. per cent, and alkaline phosphatase was 65 units. No other abnormal laboratory studies were apparent.

Hypotonic duodenogram demonstrated smooth enlargement of the papilla of Vater, which measured 5.5×2.5 cm. (Fig. 16). Papillary edema was suspected, but neoplasm was also considered because of the size.

At operation, carcinoma of the papilla of Vater was found.

Discussion

The problem of jaundice of unknown etiology has been difficult to assess by

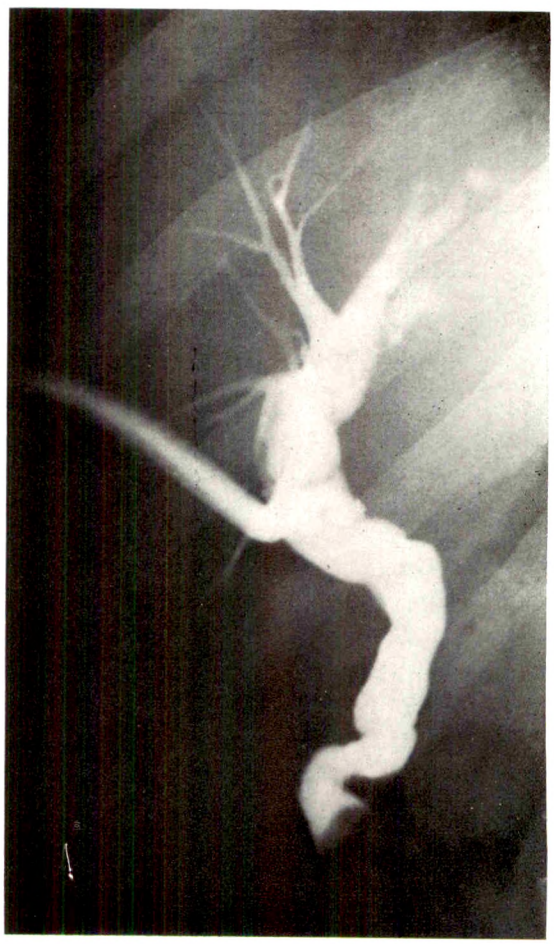


Fig. 13. Case vii. Direct cholangiogram shows mild ductal dilatation and obstructing calculus.

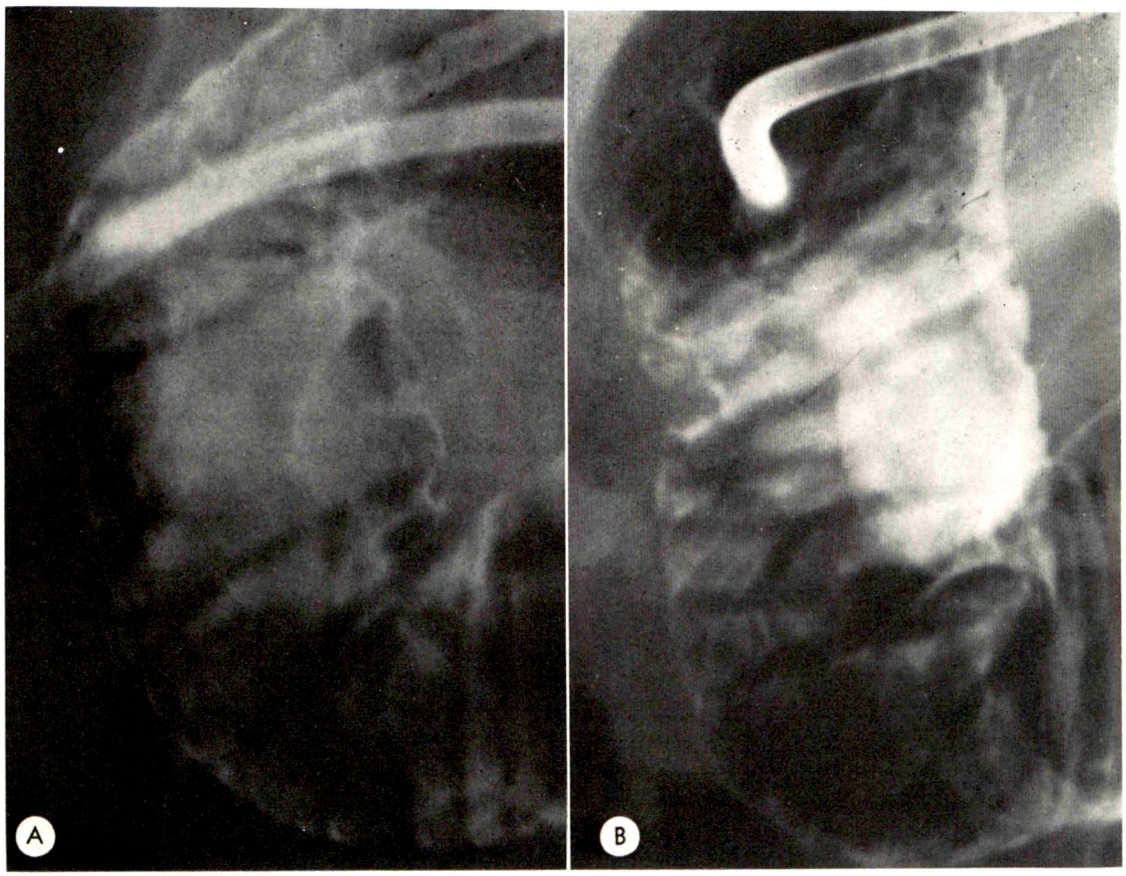


Fig. 14. Case VII. (*A*) Air contrast duodenogram shows markedly edematous papilla measuring 3.7×2.2 cm. (*B*) Different obliquity shows similar findings.

roentgenographic means. In most cases, the bilirubin is elevated, and intravenous cholangiography is impossible. Enzyme elevations can be misleading with SGOT and alkaline phosphatase elevated in most of the cases presented.

The rationale for the performance of hypotonic duodenography for jaundice centers around the determination of whether obstruction is present. With hypotonic duodenography, one is able to contribute to the decision if hepatic disease or obstruction is the cause of the jaundice. If the hypotonic duodenogram is normal, this helps the clinician determine if surgery should be performed or postponed. In the case of an abnormal duodenogram, further studies may be performed, including transhepatic cholangiography and angiography, followed by surgery.[7]

The procedure is relatively simple to perform using the method advocated by Ferrucci and Benedict.[5] Using 30 mg. of Pro-Banthine intramuscularly and using combinations of barium and air, adequate analysis of the duodenum is possible in nearly all cases. Complications have been nonexistent in our experience. Occasionally, patients have complained of difficulty in urinating for 12 hours following the procedure, but we have not had to insert a urethral catheter in any patient. The procedure is contraindicated in glaucoma or cardiac arrhythmia.[5] We routinely empty the stomach of all air and barium before withdrawing the tube, to prevent gastric distention.

The average greatest diameter of the papilla of Vater in 6 cases of impacted common duct stones was 2.8 cm. Considering



Fig. 15. Case VIII. Normal duodenogram. The patient had a stone impacted high in the common bile duct.

some magnification in fluoroscopic spot filming, an average size of about 2.0 cm. would seem realistic. Poppel and associates[10] demonstrated an average size of the papilla as 1.5 cm. in anatomic specimens. In later publications, however, a maximum size of 1.5 cm. is generally recognized as the upper limit of normal.[1,6,7,11]

The longitudinal fold may also be involved in the edematous process and contribute to the size of the papillary image.[6] In Case III, the papillary size is relatively normal, but the longitudinal fold is widened and elongated, indicating edema[6] (Fig. 6, *A* and *B*).

The problem of differentiating edema of the papilla of Vater from neoplasm of the papilla or of the head of the pancreas may be difficult. Carcinoma of the head of the pancreas was found in Case IV (Fig. 8, *A* and *B*). At duodenography, a defect was identified, but without the usual mucosal alterations.[9] The interpretation was that of an intrinsic defect, rather than extrinsic. The probability exists, however, that the papilla may have been enlarged from the carcinoma. Further evaluation with angiography may have established the correct diagnosis.

Carcinoma of the papilla of Vater in its

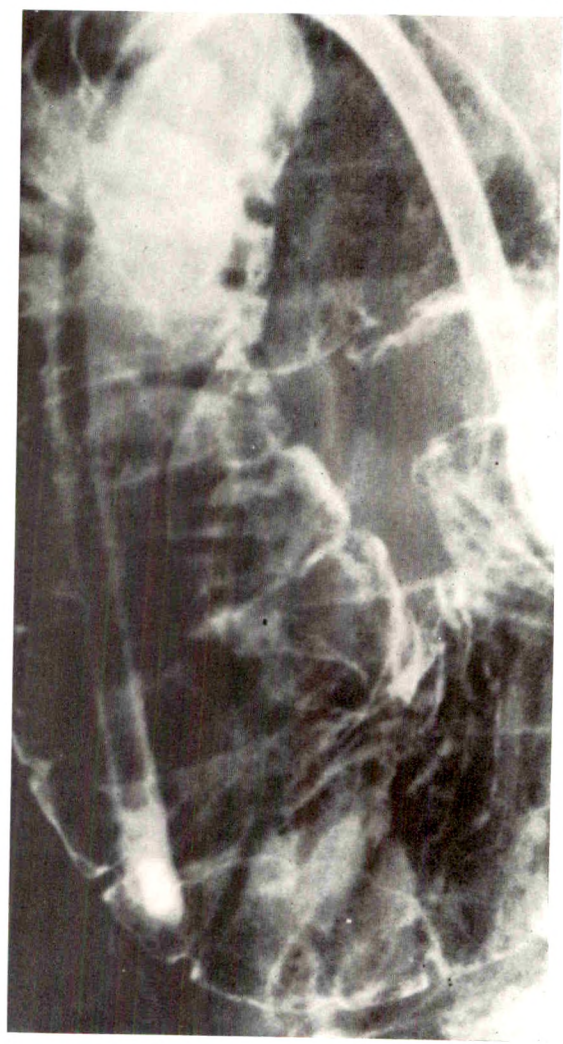


Fig. 16. Case IX. Hypotonic duodenogram with air contrast showing markedly enlarged papilla measuring 5.5×2.5 cm. No ulceration or mucosal irregularity was identified. At surgery, a carcinoma of the papilla of Vater was found.

early stage may be impossible to differentiate from papillary edema. This was the differential problem in Case IX. The size of the papilla may be 2.0 to 3.0 cm., and the image may be smooth. As this neoplasm grows, however, mucosal irregularities, vegetative growths and ulcerations can be identified.[7,8,11] None of these findings were appreciated in Case IX (Fig. 16). The value of the duodenogram is still great, however, because of the abnormalities found.

When calculi are impacted high in the common bile duct, as in Case VIII, the diagnosis cannot be made by hypotonic duodenography, and further diagnostic procedures may be attempted (Fig. 15). This would be a major drawback of total reliance upon the duodenogram for a decision to delay operation.

The differentiation of enlargement of the papilla and longitudinal fold secondary to pancreatitis, from edema of a common duct stone, is also a consideration. The clinical findings, however, should be helpful in this regard.

Aberrant positions of the papilla of Vater should be searched for in the interpretation of the duodenogram. In Case V, a typical appearance of the promontory is seen in a normal position. However, the papilla is located far below the promontory (Fig. 9). This finding, as discussed by Ferrucci *et al.*,[6] is unusual and must be recognized.

SUMMARY

Hypotonic duodenography has been found to be of definite value in the preoperative diagnosis of choledocholithiasis.

By evaluating the size of the papilla of Vater, as visualized on the duodenogram, edema of the papilla can be detected. A papilla larger than 1.5 cm. is considered abnormal.

Nine cases are reported, including 1 false negative because of high impaction of a stone; 1 false positive was carcinoma of the pancreas. One case of carcinoma of the papilla of Vater, showing smooth enlargement of the papilla which we associate with edema, is also presented.

The value of the duodenogram in differentiating obstructive from nonobstructive jaundice is stressed.

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REFERENCES

1. Bilbao, M. K., Frische, L. H., and Dotter, C. T. Hypotonic duodenography. *Radiology*, 1967, *89*, 438–443.
2. Bilbao, M. K., Frische, L. H., and Rösch, J. Post-bulbar duodenal ulcer and ring stricture. *Radiology*, 1971, *100*, 27–35.
3. Bilbao, M. K., Rösch, J., and Frische, L. H. Hypotonic duodenography in diagnosis of pancreatic disease. *Seminars in Roentgenol.*, 1968, *3*, 280–287.
4. Eaton, S. B., Benedict, K. T., Jr., and Ferrucci, J. T., Jr. Hypotonic duodenography. *Radiol. Clin. North America*, 1970, *8*, 125–137.
5. Ferrucci, J. T., Jr., and Benedict, K. T., Jr. Anticholinergic-aided study of gastrointestinal tract. *Radiol. Clin. North America*, 1971, *9*, 23–39.
6. Ferrucci, J. T., Jr., Benedict, K. T., Jr., and Page, D. L. Radiographic features of normal hypotonic duodenogram. *Radiology*, 1970, *96*, 401–408.
7. Jacquement, P., Liotta, D., and Mallet-Guy, P. The Early Radiological Diagnosis of Disease of the Pancreas and Ampulla of Vater: Elective Exploration of the Ampulla of Vater and the Head of the Pancreas by Hypotonic Duodenography. Charles C Thomas, Publisher, Springfield, Ill., 1965.
8. Mallet-Guy, P., Jacquement, P., and Cavazutti, P. 500 cas de duodenographie hypotonique. *J. de radiol., d'électrol. et de méd. nucléaire*, 1963, *44*, 249–258.
9. Martel, W., Scholtens, P. A., and Lim, L. W. Tubeless hypotonic duodenography: technique, value, and limitations. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *107*, 119–130.
10. Poppel, M. H., Jacobson, H. G., and Smith, R. W. The Roentgen Aspects of the Papilla and Ampulla of Vater. Charles C Thomas, Publisher, Springfield, Ill., 1953.
11. Rösch, J. Roentgenology of the Spleen and Pancreas. Charles C Thomas, Publisher, Springfield, Ill., 1967.
12. Rösch, J., and Salamoun, V. Die Duodenographie in Hypotonie in der Diagnostik der Pankreaserkrankungen. *Radiologe*, 1965, *5*, 289–298.
13. Stage, P., and Banke, L. Hypotonic duodenography. *Gut*, 1969, *10*, 428–432.

USE OF THE C-TERMINAL OCTAPEPTIDE OF CHOLECYSTOKININ FOR GALLBLADDER EVACUATION IN CHOLECYSTOGRAPHY*

By PAVO HEDNER *and* ANDERS LUNDERQUIST
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PREPARATIONS of cholecystokinin (CCK) extracted from swine intestine have proved to be a valuable aid in roentgen diagnostics, principally for evacuation of the gallbladder in connection with cholecystography. More than 200,000 intravenous injections of refined CCK have been given, up to 1970, without serious side reactions.[9]

However, due to limited supply of raw material and increasing cost, extracted CCK of good purity is no longer commercially available everywhere for routine clinical use.

After development of purification procedures of increasing efficiency, Mutt and Jorpes[8] were able to perform a structural analysis of porcine CCK. They found the hormone to be a linear polypeptide containing 33 amino acids. They observed that among the products resulting from tryptic degradation of the molecule, the C-terminal octapeptide was highly active. This C-terminal octapeptide of CCK was synthesized by Ondetti *et al.*[10] and shown in animal experiments to affect the gallbladder, sphincter of Oddi, small intestine and exocrine pancreas in the same way as extracted CCK, but it had a higher potency.[7,8,12]

As a molecule of this size and structure can be synthesized at a reasonable cost, the C-terminal octapeptide of CCK may be expected to be an alternative to extracted preparations in routine clinical work. Furthermore, the fact that the synthetic preparation is highly active and free from heterologous material may be regarded as an advantage.

Therefore, it was considered to be of interest to investigate the efficiency of the synthetic C-terminal octapeptide of CCK in emptying the gallbladder and visualizing the bile ducts in cholecystography in man.

MATERIAL AND METHOD

The investigation was carried out in 50 patients, 22 men and 28 women, aged 18–65 years. They were referred for cholecystography because of symptoms more or less suggestive of cholecystopathy.

All of the patients were prepared according to our usual procedure for cholecystography; *i.e.*, partaking 4 gm. iodopanoic acid orally and then fasting for 12 hours before the investigation. Only patients without roentgenologic signs of cholecystopathy were accepted for investigation with C8-CCK. In 15 patients, the C-terminal octapeptide of cholecystokinin (C8-CCK) was injected slowly intravenously, during 1–2 minutes, in a dose of 0.033 μg. per kg. body weight, corresponding to 1 Ivy dog unit per kg. body weight. Roentgenograms were made 5, 8, 12, 15, and, in a few cases, 30 minutes after the beginning of the injection.

In 36 patients, 0.033 μg. C8-CCK per kg. body weight was given intravenously during 1–2 minutes as a priming dose, followed by infusion of 0.3–0.6 μg. C8-CCK per minute until a total dose of 6 μg. was reached after 6–10 minutes. Roentgenograms were made before and 3, 6, 10 and 15 minutes after the beginning of the administration of C8-CCK. In 14 of these patients, laboratory investigation of blood and liver parameters was performed before the injection of C8-CCK and 4–5 days afterwards.

Five patients who did not respond to 6 μg. C8-CCK received 1 Ivy dog unit per kg. body weight of extracted cholecystokinin (obtained from Prof. J. E. Jorpes,

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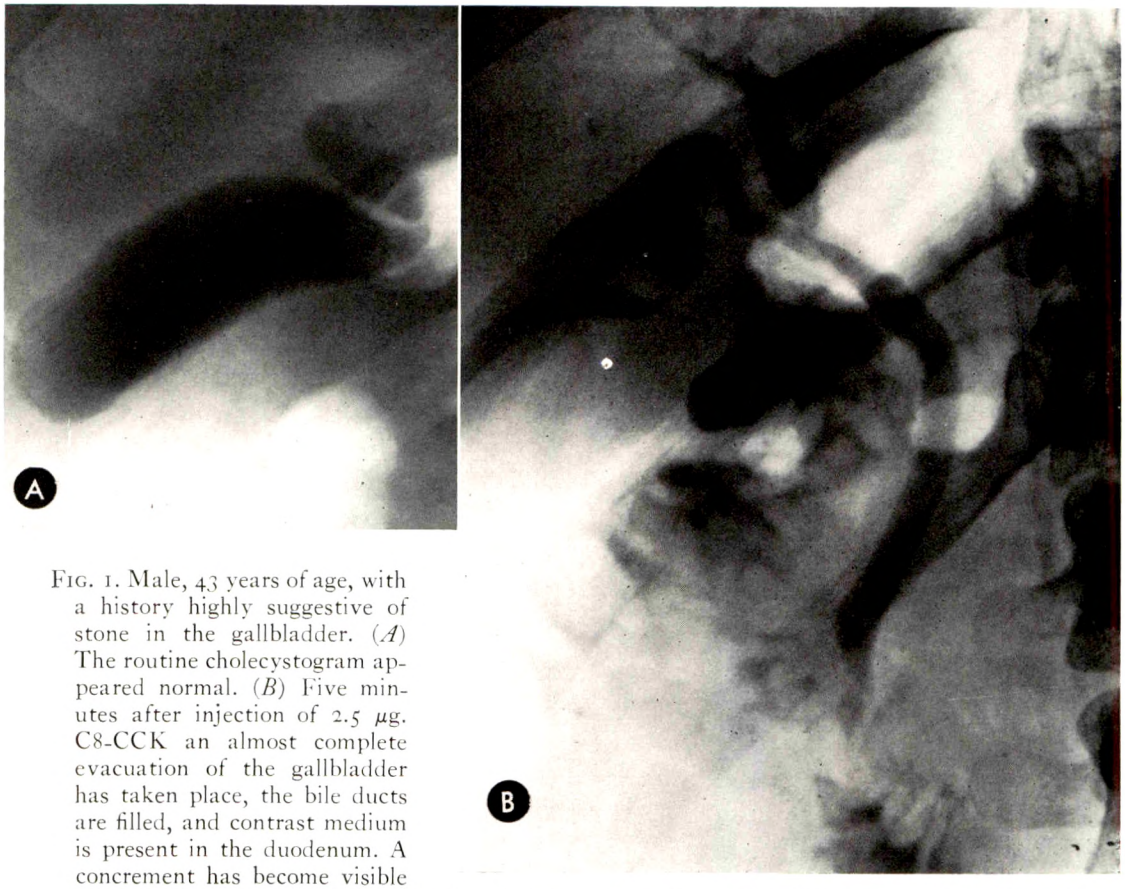


Fig. 1. Male, 43 years of age, with a history highly suggestive of stone in the gallbladder. (*A*) The routine cholecystogram appeared normal. (*B*) Five minutes after injection of 2.5 μg. C8-CCK an almost complete evacuation of the gallbladder has taken place, the bile ducts are filled, and contrast medium is present in the duodenum. A concrement has become visible in the collum part of the gallbladder. It was confirmed on a later intravenous cholangiography.

G. I. H. Laboratory, Karolinska Institutet, Stockholm, for investigational use).

The synthetic C-terminal octapeptide of cholecystokinin (SQ 19 844) was obtained as a gift from Dr. M. A. Ondetti, The Squibb Institute for Medical Research, New Brunswick, N. J., U. S. A. The cholecystokinetic potency of this substance has been assayed to 30,000 Ivy dog units per mg.[12] The substance was dissolved in 0.9 per cent NaCl to a concentration of 0.33 μg. per ml., corresponding to 10 Ivy dog units per ml., and sterile filtered.

All films were exposed with a vertical beam and the patient supine. Care was taken to have the position of the patient and the direction of the beam unchanged during the investigation.

The size of the gallbladder was estimated as the product of the largest diameter measured on the film and the largest diameter perpendicular to the first one.

RESULTS

A. Single injection of 0.033 μg. C8-CCK per kg. body weight

The first sign of a response to C8-CCK was the visualization of the cystic duct and the common bile duct (Fig. 1, *A* and *B*). This response was always seen on the first film, exposed 5 minutes after the injection, but in 1 case where the cholecystograms were taken earlier these changes were seen after 30 seconds. Then, as a rule also visible on the 5 minute film, the presence of contrast medium in the duodenum could be observed. In a few cases (Fig. 1*B*) an obvious reduction of the size of the gallbladder was seen 5 minutes after the injection, in other cases a measurable reduction

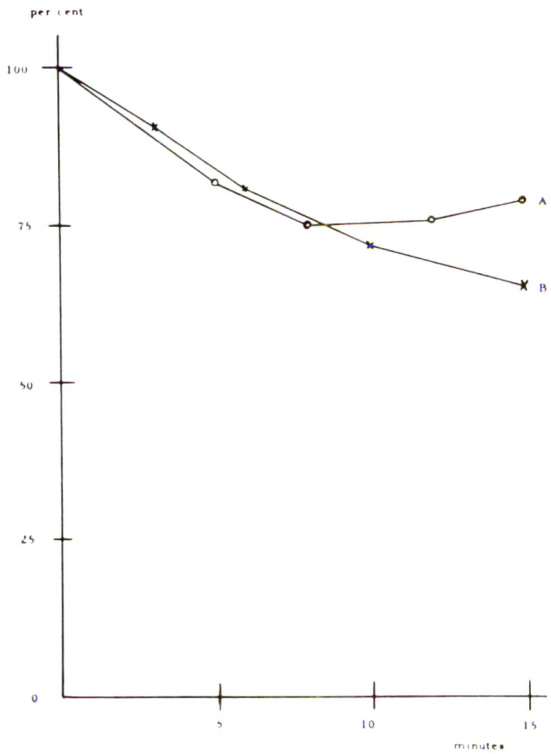


FIG. 2. Changes of the mean product of gallbladder diameters in per cent of preinjection values, following C8-CCK given as a single intravenous injection (A) and as an infusion (B) between 0 and 10 minutes.

was not obtained until the 8 minute film. In no case was any further reduction of the gallbladder size obtained 15 or 30 minutes after the injection compared with the 12 minute film, and in most cases practically no difference of the gallbladder size was found between the 8 and 12 minute films. Thus, with this technique of injection, we obtained a rapid onset of the effect of C8-CCK, giving a continuous reduction of the mean gallbladder size up to 8 minutes after the injection, but not later. This is also clear from Figure 2, which graphically illustrates the change of the mean gallbladder size with time.

The short duration of the effect of C8-CCK was also demonstrated in 1 patient, where visualization of the bile ducts, presence of contrast medium in the duodenum and a slight reduction of the size of the gallbladder were found on the films exposed 5 and 8 minutes after the injection but not later. However, after 15 minutes, a second injection of the same dose of C8-CCK was given, and the same response as after the first injection was again obtained for a corresponding period after the injection.

In the 15 patients forming this group, visualization of the bile ducts was obtained in all, and reduction of the product of the gallbladder diameters by more than 10 per cent in 13 cases.

B. PRIMING DOSE OF 0.033 μg. C8-CCK PER KG. BODY WEIGHT FOLLOWED BY INFUSION, 0.6 μg. PER MINUTE

Depending on the body weight of the patient, this mode of administration meant infusion for 6–8 minutes until a total dose of 6 μg. was reached as an arbitrary upper limit. This way of administration was tried to neutralize the short duration of the preparation and enable a greater degree of evacuation of the gallbladder.

Figure 2 depicts the change of the mean gallbladder size with time during and after the infusion. A more durable effect was obtained with this technique compared to the single injection. It was often observed that soon after the end of the infusion, no further decrease of the size of the gallbladder was obtained. A representative series of roentgenograms during the infusion of C8-CCK is shown in Figure 3, *A–D*.

Of the 35 patients infused, 30 responded with visualization of the bile ducts and contrast medium in the duodenum. Of these 30, 8 patients presented a reduction of the product of the gallbladder diameters by more than 50 per cent.

In 5 of the 35 patients in this group, no reduction at all of the gallbladder size was obtained during an observation time of 15 minutes. At this time, these patients were injected with extracted cholecystokinin, 1 Ivy dog unit per kg. body weight, and then observed with repeated cholecystograms for another 15 minutes. However, all of these 5 patients, who did not respond to C8-CCK, failed to respond also to extracted cholecystokinin.

In some cases, contraction of the neck of

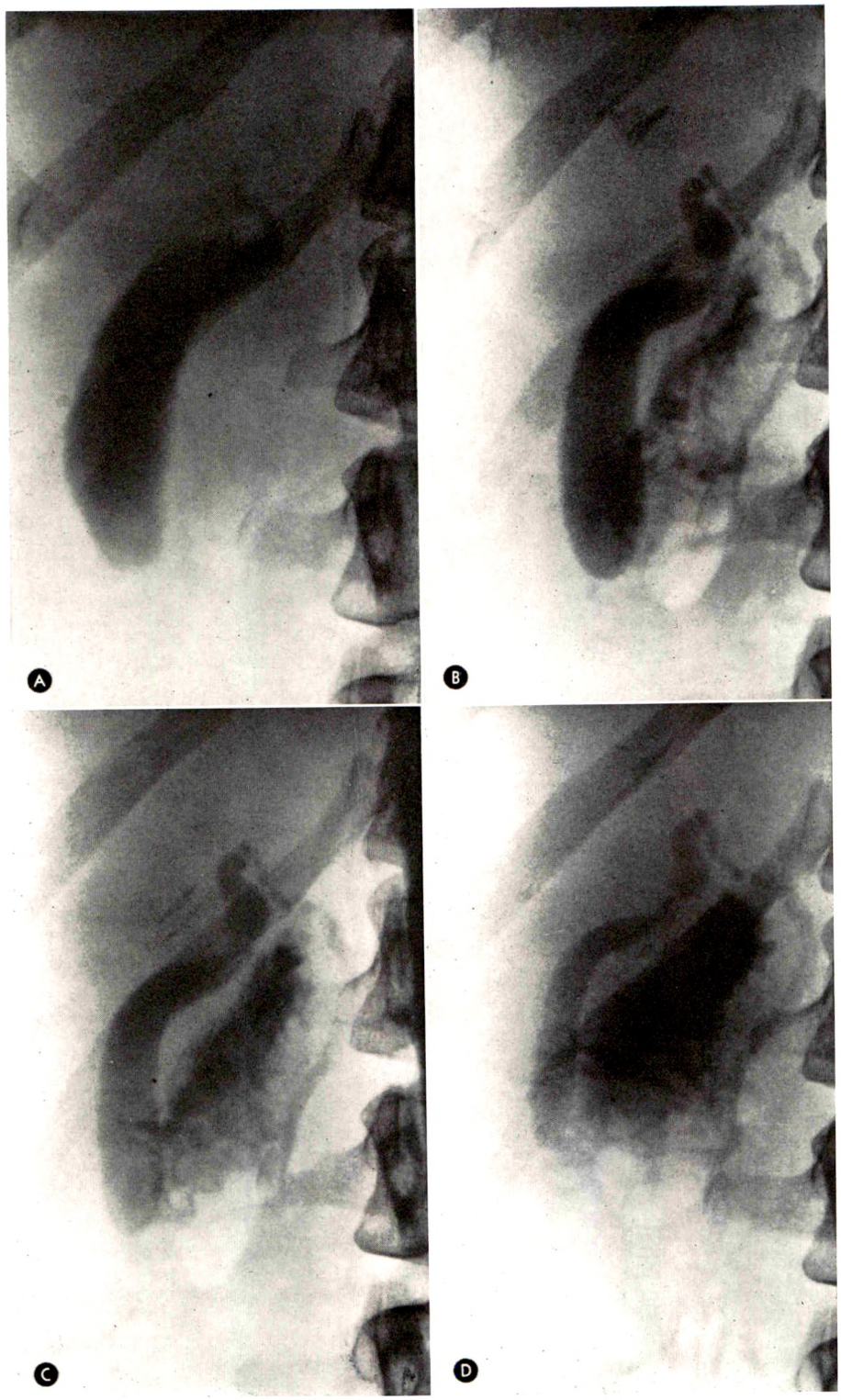


Fig. 3. Female, 42 years of age, with a representative emptying pattern of the gallbladder during a 10 minute infusion of 6 μg. of C8-CCK. (*A*) Preinjection roentgenogram; (*B*) 3 minutes after the beginning of the infusion; (*C*) 6 minutes after the beginning of the infusion; and (*D*) 10 minutes after the beginning of the infusion.

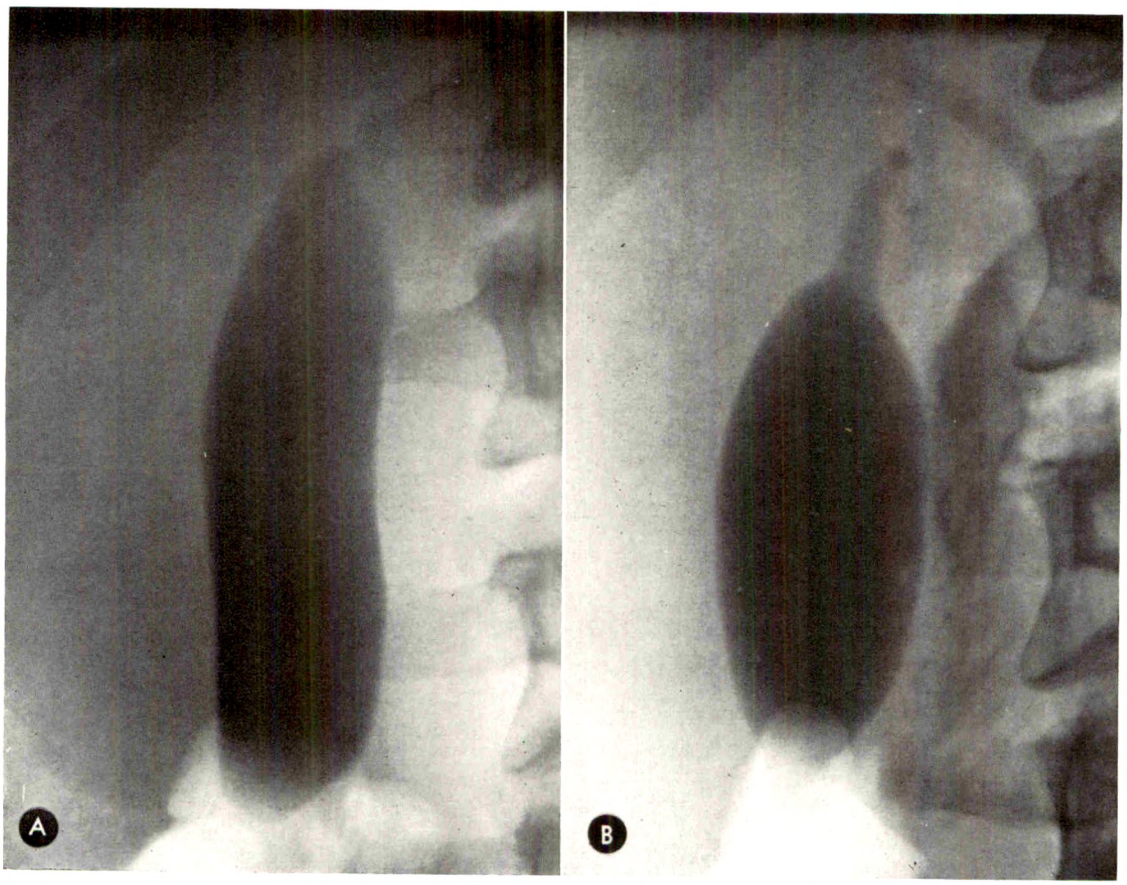


Fig. 4. (*A* and *B*) Male, 19 years of age, with a characteristic contraction of the gallbladder neck after too rapid an injection of C8-CCK.

the gallbladder was observed after too rapid injection of C8-CCK (Fig. 4, *A* and *B*). This seemed to delay the reduction of the size of the gallbladder. Four women among the patients were taking oral contraceptives. Three of them showed a reduction of the product of the gallbladder diameters by more than 25 per cent following C8-CCK.

SIDE REACTIONS

The only side reactions experienced by the patients were a moderate nausea that usually disappeared within 1–2 minutes, and in some cases abdominal discomfort for 2–5 minutes.

The mean systolic blood pressure decreased with 2.2 ± 1.5 mm. Hg during the 15 minutes consumed by the investigation. Correspondingly, a decrease of the mean diastolic blood pressure with 0.3 ± 0.5 mm. Hg was registered. The changes were not statistically significant and in no cases did they exceed 10 mm. Hg.

The following laboratory tests were performed before the injection of C8-CCK and repeated 4–5 days later in 14 patients receiving a total dose of 6 μg.: sedimentation rate; red blood cell count; hemoglobin concentration; hematocrit; MCV; MCHC; MCH; white blood cell count and differential count; thrombocyte count; SGOT; SGPT; LDH and bilirubin concentration; and alkaline phosphatase activity in serum. No statistically significant changes of any of these parameters were found after the administration of C8-CCK.

DISCUSSION

It should be emphasized that the aim of this investigation was not to directly compare the effects of extracted CCK and C8-CCK, but to study whether C8-CCK could

be used clinically and fill the place of extracted CCK.

It was apparent from most cholecystograms that following C8-CCK the gallbladder emptied part of its content, filling the bile ducts and eventually also the duodenum. However, as pointed out by Brodén[4] and Edholm[5] the numerical assessment of the changes of the gallbladder volume obtained by measurement on the film is severely biased by many factors. Therefore, we have renounced a statistical treatment of our measurements on the films, and the mean values of the products of the gallbladder diameters given in Figure 2 should be regarded as a very rough estimate.

It was, however, apparent from this investigation that C8-CCK could produce the evacuation of the gallbladder that is sometimes wanted for routine roentgen diagnostic purposes with a minimum of discomfort to the patient and without affecting laboratory blood and liver parameters. After C8-CCK, visualization of the cystic and the common bile duct was obtained within 5 minutes in most cases. Thus, if the state of these structures is questioned, administration of C8-CCK may provide an answer at the cost of prolonging the cholecystographic procedure by 5 minutes and eventually eliminating the need for an intravenous cholangiography. If the ability to empty the gallbladder is to be investigated, we suggest the administration of C8-CCK as an infusion which, however, requires a longer time of observation —at least 15 minutes.

Without making any direct comparison, we feel that the effect of C8-CCK is less durable than that of extracted CCK which has been reported to last for 15 minutes[6] to 2 hours.[2] Also, in animal experiments the effect of C8-CCK has been reported to be less durable than that of extracted CCK.[12] For visualization of the bile ducts the short duration of C8-CCK is usually no serious drawback, but it necessitates the more laborious infusion if a more complete evacuation of the gallbladder is wanted.

In 7 of our 50 patients no reduction of the gallbladder size was obtained, and there was no visualization of the bile ducts in 5 of them. This was probably not due to the preparation of C8-CCK, as 5 of these patients failed to respond also to extracted CCK. In fact, about 15 per cent of patients selected in a similar manner as ours failed to respond to extracted CCK according to Backlund.[1] Upon reinvestigation 1–2 years later he found that about half of these "nonresponders" had developed roentgenologic signs of cholecystopathy or calculus formation. Backlund[1] suggests that inability to respond to CCK may be an early sign of cholecystopathy. However, it should also be borne in mind that too rapid injection or too large doses of CCK have been reported to contract the gallbladder neck so completely that evacuation of bile is prevented, at least temporarily.[13] Therefore, we administered the first portion of C8-CCK (single and priming injection, respectively) slowly, during 1–2 minutes, but still we observed some cases of neck contraction in our material lasting for about 5 minutes. Nathan *et al.*[9] consider neck spasm lasting for more than 5 minutes after injection of CCK as a sign of cholecystopathy.

Plessier *et al.*[11] reported impaired or abolished contracting effect of CCK on the gallbladder during pregnancy in humans. Oral contraceptives mean hormonal changes in part similar to those occurring during pregnancy. However, 3 of the 4 patients in the present study who were on oral contraceptives responded normally to C8-CCK. Thus oral contraceptives do not seem to invalidate an investigation performed with the aid of C8-CCK.

Practically no side reactions were encountered after C8-CCK in the present study. The slight and insignificant decrease of systolic and diastolic blood pressure may be ascribed to the horizontal position of the patient during 15 minutes as well as to the preparation.

In spite of being contaminated with heterologous material, extracted CCK has not been found to exert any harmful side effects during an extensive practical use.

However, the limited supply of extracted CCK makes alternative preparations desirable. Admittedly, a fatty meal may meet the requirements, but it is time consuming and makes a subsequent investigation of, *e.g.*, the stomach, impossible. As alternatives remain synthetic peptides with high cholecystokinetic activity. Such criteria are fulfilled by caerulein and C8-CCK. Caerulein, a decapeptide with a structure slightly different from that of C8-CCK, was originally isolated from the skin of the frog *Hyla caerulea* and has been shown to have a cholecystokinetic effect in man.[3] The present investigation demonstrated that C8-CCK, with a structure of mammalian origin, also can be used in man for emptying the gallbladder and visualizing the bile ducts with a minimum of side effects.

SUMMARY

Cholecystokinin is a linear polypeptide containing 33 amino acids. Among the tryptic degradation products of the molecule, the C-terminal octapeptide is known to be highly active.

In the present study, the synthetic C-terminal octapeptide of cholecystokinin was administered to 50 patients referred for cholecystography. The preparation was found to have a shorter duration of action compared to extracted cholecystokinin, but produced evacuation of the gallbladder and visualization of the bile ducts without any harmful side effects.

The synthetic C-terminal octapeptide of cholecystokinin may be expected to be a valuable alternative to extracted preparations that are not available at present in amounts sufficient for routine clinical use.

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REFERENCES

1. Backlund, V. Kolecystokinin vid röntgenundersökningar. *Läkartidningen*, 1967, *64*, 2473–2476.
2. Backlund, V. Die Verwendung des Cholecystokinins in der Röntgendiagnostik. *Der Radiologe*, 1970, *10*, 36–39.
3. Bertaccini, G., Braibanti, T., and Uva, F. Cholecystokinetic activity of new peptide caerulein in man. *Gastroenterology*, 1969, *56*, 862–867.
4. Brodén, B. Experiences with cholecystokinin in cholecystography. *Acta radiol.*, 1958, *49*, 25–30.
5. Edholm, P. Emptying of human gall bladder under stimulus of cholecystokinin. *Acta radiol.*, 1958, *50*, 521–532.
6. Edholm, P. Gall bladder evacuation in normal male induced by cholecystokinin. *Acta radiol.*, 1960, *53*, 257–265.
7. Hedner, P. Effect of C-terminal octapeptide of cholecystokinin on guinea pig ileum and gallbladder in vitro. *Acta physiol. scandinav.*, 1970, *78*, 232–235.
8. Mutt, V., and Jorpes, J. E. Structure of porcine cholecystokinin—pancreozymin. I. Cleavage with thrombin and with trypsin. *European J. Biochem.*, 1968, *6*, 156–162.
9. Nathan, M. H., Newman, A., Murray, D. J., and Camponovo, R. Cholecystokinin cholecystography. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *110*, 240–251.
10. Ondetti, M. A., Pluscec, J., Sabo, E. F., Sheehan, J. T., and Williams, N. Synthesis of cholecystokinin—pancreozymin. I. C-terminal dodecapeptide. *J. Am. Chem. Soc.*, 1970, *92*, 195–199.
11. Plessier, J., Wettendorff, P., Plessier, B., and Cohen, J. L'atonie vésiculaire chez la femme et le cobaye gravides. Essai d'explication par le dosage biologique des hormones digestives. *Ann. Biol. Clin.*, 1961, *19*, 843–850.
12. Rubin, B., Engel, S. L., Drungis, A. M., Dzelzkalns, M., Grigas, E. O., Waugh, M. H., and Yiacas, E. Cholecystokinin-like activities in guinea pigs and dogs of C-terminal octapeptide (SQ 19 844) of cholecystokinin. *J. Pharmacol. Sc.*, 1969, *58*, 955–959.
13. Torsoli, A., Ramorino, M. L., Colagrande, C., and Demaio, G. Experiments with cholecystokinin. *Acta radiol.*, 1961, *55*, 193–206.

PERCUTANEOUS TRANSHEPATIC CHOLANGIOGRAPHY: THE DIFFERENTIAL DIAGNOSIS OF BILE DUCT PATHOLOGY*

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SINCE its beginnings in the 1920s[1] and 1930s,[4] its rejuvenation in the 1950s,[2,6] and its consolidation in the 1960s,[8–10] percutaneous transhepatic cholangiography has become a well-recognized but perhaps underutilized aid in the diagnosis and management of the jaundiced patient. The advent of image intensification fluoroscopy and television monitoring has made it a rapid and safe procedure when performed by an experienced radiologist.

This report presents the criteria for the interpretation of the percutaneous transhepatic cholangiogram based on our experience at the Mayo Clinic.

MATERIAL AND METHOD

We studied the results of 88 attempts at percutaneous cholangiography undertaken on 87 patients (Table I). Sixty cholangiograms were obtained. Fifty-four of these showed obstruction of the extrahepatic ducts, which varied from slight to complete and produced different degrees of dilatation of the intrahepatic ducts. Three showed irregular narrowing of the intrahepatic ducts with slight dilatation proximal to the narrowed areas. Two patients had normal duct systems. One had a fibrotic, old, intrahepatic abscess cavity with normal common hepatic and common bile ducts.

TABLE I

PERCUTANEOUS CHOLANGIOGRAPHY: 88 ATTEMPTS ON 87 PATIENTS

Successful		Unsuccessful	
Ducts obstructed and dilated	54	Parenchymal disease	21
Multiple intrahepatic strictures	3	Obstructed at laparotomy	7
Normal	2		
Intrahepatic abscess cavity	1		
Total	60	Total	28

TABLE II

GROUPS STUDIED FOR SIGNIFICANCE OF BILE DUCT DILATATION

Group	No.
Benign stricture	24
Bile duct carcinoma	13
Carcinoma of head of pancreas	6
Hilar metastasis	5
Impacted gallstones	4
Total	52

Of the 3 patients with narrowed ducts, one was thought to have a chronic fibrosing inflammatory duct disease secondary to previous recurrent cholelithiasis and stricture. Another had a downhill clinical course which suggested a sclerosing carcinoma of the ducts, but autopsy was not permitted and no proof was available. The third had chronic ulcerative colitis complicated by carcinoma of the colon and had had a previous cholecystectomy and common duct exploration; however, since the bile ducts were not obstructed, biliary tract surgery was not done and no proof was available of the etiology of the sclerosing duct disease.

Of the 54 cholangiograms showing obstruction, 52 were divided into 5 groups according to the cause of obstruction as determined by laparotomy and biopsy (Table II). Of the remaining two, one study was

* Presented at the Seventy-second Annual Meeting of the American Roentgen Ray Society, Boston, Massachusetts, September 28–October 1, 1971.
From the Mayo Clinic and Mayo Foundation, Rochester, Minnesota.

considered incomplete and the other was an isolated case of chronic pancreatitis. The widest intrahepatic duct was measured on each of the 52 cholangiograms. In those with a diagnosis of malignancy, histologic proof was obtained by biopsy or autopsy studies.

The 60 successful cholangiograms were analyzed with regard to the following criteria: (1) history of previous biliary tract surgery; (2) statistical significance of the degree of dilatation of the intrahepatic ducts in different groups separated according to the ultimate diagnosis; (3) shape of the obstructing lesion when completely outlined; (4) shape of the lower end of the opaque column when obstruction was complete; (5) course of the extrahepatic ducts; (6) consistency or inconsistency in the caliber of the intrahepatic ducts; (7) intraductal filling defects; (8) cavities within the liver; and (9) biliary-intestinal fistulae.

Twenty-eight attempts at percutaneous cholangiography were unsuccessful. These patients were studied with a view to determining the reason for failure and whether it had any inferential value. Operative or T-tube cholangiograms were available for 10 of these, and biopsy results for 9.

INDICATIONS

This examination is performed for 2 reasons:

1. To differentiate between intrahepatic cholestasis and extrahepatic obstruction in those jaundiced patients in whom clinical and laboratory examinations have been inconclusive.

2. To delineate the anatomy of a known obstructing lesion to facilitate operative management.

This preoperative knowledge is even more valuable if the nature of the obstruction can be assessed from the cholangiogram.

CONTRAINDICATIONS

The procedure is contraindicated in the presence of a bleeding diathesis, sensitivity to the contrast medium, suppuration in the proposed puncture area, or known echinococcal disease of the liver. It would be unwise to perform the examination on a patient whose general condition precluded surgery in the event of this becoming necessary as a result of either the findings at cholangiography or any complications therefrom.

TECHNIQUE

The patient's prothrombin time should be checked and corrected if necessary prior to scheduling percutaneous cholangiography. Adequate premedication is essential. Dorsal and lateral approaches to the intrahepatic bile ducts have been advocated[8,10] in order to avoid the possible complications of bile peritonitis or hemorrhage due to puncture of the gallbladder, extrahepatic bile ducts, or blood vessels in the porta hepatis. However, in agreement with most authors, we prefer to use a frontal approach.

After assessing the size and position of the liver under fluoroscopy, we use an aseptic technique to produce local anesthesia of the skin, abdominal wall, and liver capsule just below the right costal margin in the midclavicular line. Then, a small skin incision is made and a 23 gauge 8 inch needle sheathed in a 20 gauge Teflon catheter is plunged into the peripheral parenchyma of the liver during controlled apnea. The needle is inclined 45° to the horizontal and directed cranially and slightly medially. When its tip is well within the liver, the needle is withdrawn leaving the catheter in place, and the patient is allowed to breathe normally. The soft catheter is not likely to tear the liver during respiration, whereas a rigid needle might do so.

A syringe containing a suitable water-soluble contrast medium is then attached to the catheter and small amounts of medium are injected as the catheter is slowly withdrawn under fluoroscopic observation. If the catheter tip is in a blood vessel—which is common due to the abundance of veins in the liver—the contrast medium is quickly washed away, whereas if it is in the parenchyma pooling of the medium occurs.

Medium injected into the bile duct system does not wash away and flows slowly toward the liver hilum. When identified, the bile ducts are injected with as much contrast medium as is necessary to fill the entire ductal system.

If cross-filling of the other half of the ductal system does not occur, it sometimes can be accomplished by rolling the patient over onto the opposite side.[5] If this is not successful, puncture of the other lobe (through the same skin incision) with further attempts to outline these ducts should be made. If peripheral liver punctures are unsuccessful in locating a duct, subsequent punctures are made nearer the hilum where the ducts are larger. Lack of success after 5 attempts is usually considered to indicate that the ducts are not dilated, but we have had to make as many as 8 attempts to complete the examination.

When the entire duct system has been satisfactorily opacified, spot roentgenograms are taken in various positions and developed for immediate evaluation. If the ducts are obstructed, roentgenograms taken with the patient upright may provide better filling of the lower duct and better delineation of the obstruction. Further injections and further roentgenograms are made if indicated by the fluoroscopic and spot roentgenographic assessment.

Laparotomy is usually carried out on the same day unless the roentgenologic findings obviate the need for surgery, in which case the patient is carefully observed for possible complications over a period of 3 days.

COMPLICATIONS

The reported complications are bile peritonitis, hemorrhage, and gram-negative septicemia. A complication rate of 2 to 5 per cent, including 4 fatalities, has been reported in 1,740 examinations.[7,8] Our only complication in 88 studies was a gram-negative septicemia in 1 patient who had a history of intermittent biliary colic and obstructive jaundice prior to being referred to this clinic, but who had a normal percutaneous cholangiogram shortly after admission. A gram-negative septicemia developed 6 hours after cholangiography, which responded quickly to tetracycline therapy. It was inferred that he may have passed an obstructing calculus prior to cholangiography since there is a high incidence of gram-negative organisms in the bile associated with cholelithiasis and benign strictures, but rarely in those patients with complete obstruction due to carcinoma of the head of the pancreas.[3]

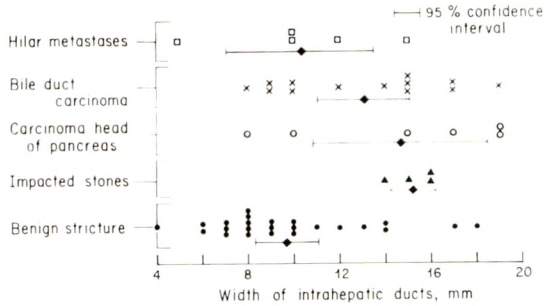


FIG. 1. Dilatation of intrahepatic ducts in 52 patients. Symbol in middle of 95 per cent confidence limit denotes mean value.

Some of our patients experienced transient right upper quadrant pain at the time of cholangiography or shortly afterward. An injection too close to the diaphragm may produce right shoulder pain. We do not regard these transient and easily relieved symptoms as complications but rather as concomitants to the procedure. However, a patient who complains of any symptom should be carefully observed for complications.

INTERPRETATION OF RESULTS

SIGNIFICANCE OF PREVIOUS BILIARY TRACT SURGERY

All of our patients with benign stricture had had previous biliary tract operations and 6 had documented accidental trauma to the extrahepatic ducts at operation. In 2 of these, sutures had been inadvertently tied around a duct. If an obstruction is noted on cholangiography in a patient who has not had previous biliary tract surgery, tumor and impacted stone must be the major considerations.

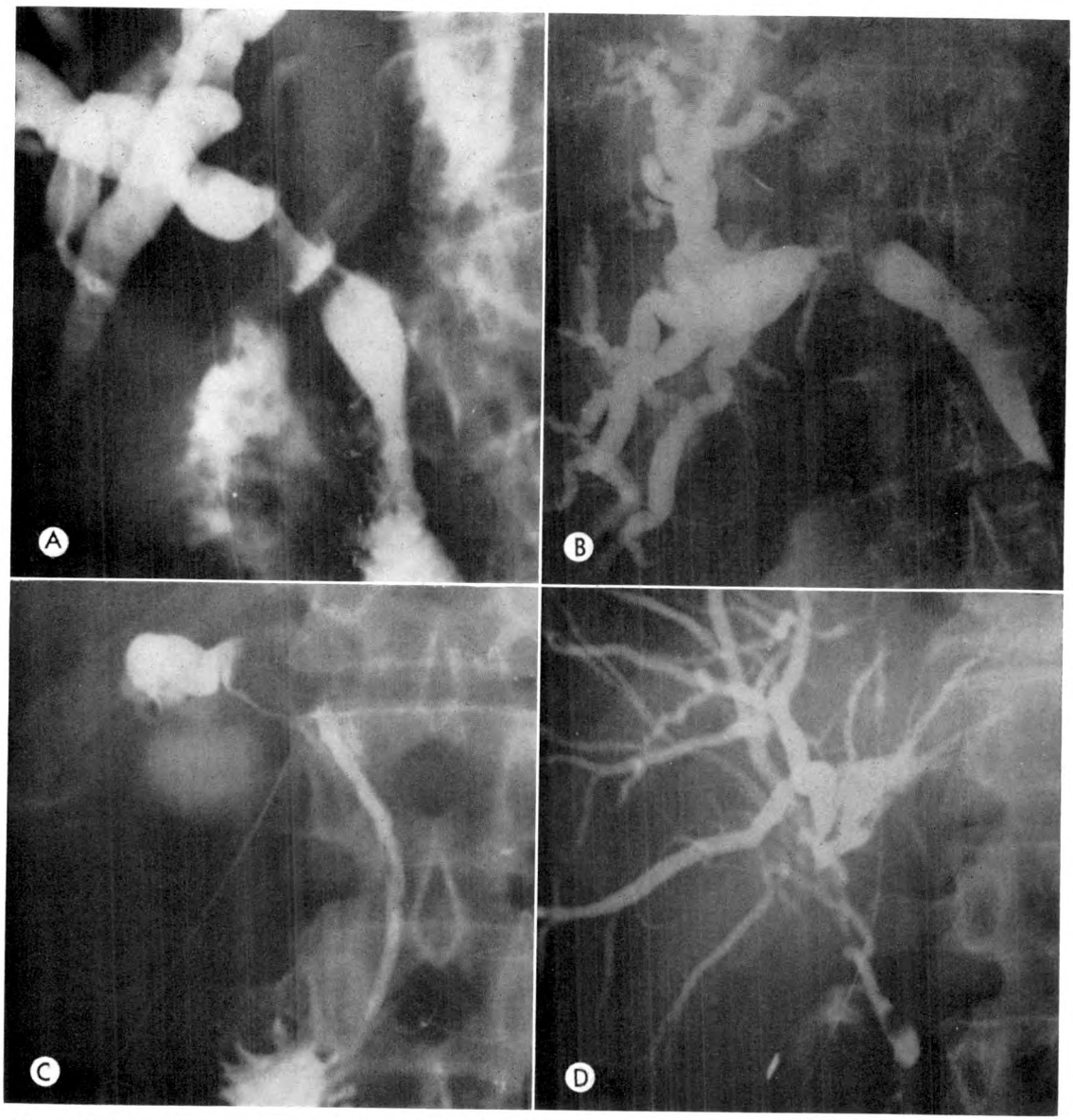


FIG. 2. (*A*) Annular carcinoma producing "apple core" lesion in common hepatic duct. Multiple rounded filling defects in intrahepatic ducts resemble stones, but extent of tumor precluded investigation at operation. (*B*) Similar lesion in another patient. (*C*) Large, irregular, polypoid carcinoma in common hepatic duct. Duct proximal to lesion is dilated, whereas common duct distal to it is normal. (*D*) Small benign "polyp" in lower common duct. It was composed of fibrous tissue.

DEGREE OF DILATATION OF INTRAHEPATIC DUCTS

The mean maximal dilatation was 15 mm. in patients with impacted stones causing obstruction, 15 mm. in patients with carcinoma of the head of the pancreas, and 13 mm. in patients with primary bile duct carcinoma (Fig. 1). These values were significantly greater than the mean, 10 mm., in patients with obstruction due to benign stricture.* Although there was considerable overlap, the evidence suggests that a patient with greatly dilated ducts is more

* Analysis of variance showed a significant difference between groups ($P < 0.025$). Individual comparisons of the means of each of the primary cancer groups (pancreas and duct) with the mean of the benign stricture group gave $P < 0.02$ and $P < 0.04$, respectively.

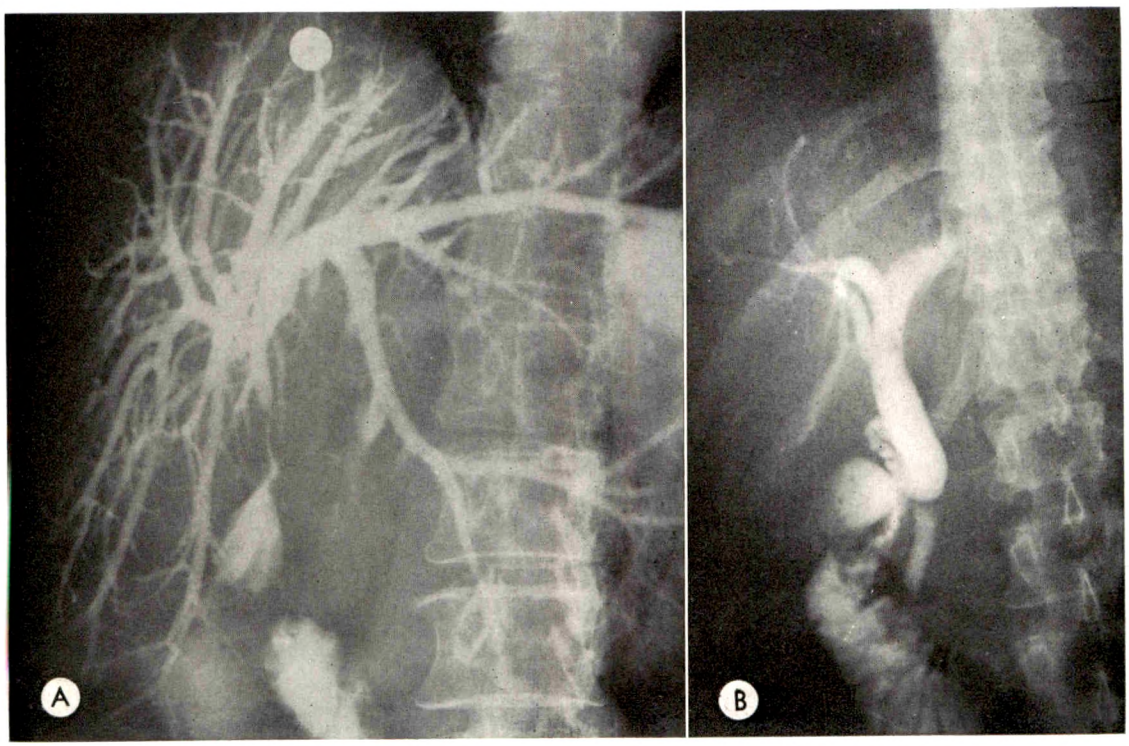


Fig. 3. Strictures. (*A*) At site of previous hepaticoduodenostomy. (*B*) Diaphragm-like stricture at site of previous stabbing injury to common duct just below its junction with cystic duct. Note stones in gallbladder and common hepatic duct.

likely to have stone obstruction, carcinoma of the head of the pancreas, or primary ductal carcinoma rather than a benign stricture. A roentgenogram taken with the patient erect may enhance the outline of an obstructing stone, and carcinoma of the pancreas and primary carcinoma of the ducts can sometimes be distinguished by other means, described below.

SHAPE OF OBSTRUCTING LESION

In 2 cases of carcinoma of the common hepatic duct, there were "apple core" lesions such as seen in carcinoma of the colon. This appearance in a contrast-filled tubular structure is almost pathognomonic of carcinoma (Fig. 2, *A* and *B*). A third case of bile duct carcinoma presented as a large, irregular, polypoid lesion projecting from one wall of the duct and incompletely surrounded by medium (Fig. 2*C*). This also is similar to polypoid carcinoma of the colon and, because benign tumors of the bile ducts are rare, this lesion would be very unlikely to be other than carcinoma. However, in 1 case a small, smooth, benign-appearing, polypoid lesion in the common duct turned out to be a mass of fibrous scar tissue (Fig. 2*D*).

The irregular stricture of a previous anastomosis between a bile duct and the bowel is also characteristic (Fig. 3*A*) as is the thin, diaphragm-like, filling defect seen in 3 of our cases of benign stricture (Fig. 3*B*).

SHAPE OF LOWER END OF OPAQUE COLUMN

An impacted stone produces a typical downwardly concave smooth meniscus (Fig. 4*A*); tumor, such as that of the ampulla of Vater (Fig. 4*B*), may produce a similar appearance. All 6 cases of carcinoma of the pancreas showed a downwardly convex meniscus (Fig. 4*C*); there was complete obstruction in 4 and almost complete obstruction in 2. This appearance has been substantiated by others.[7,8] A similar ap-

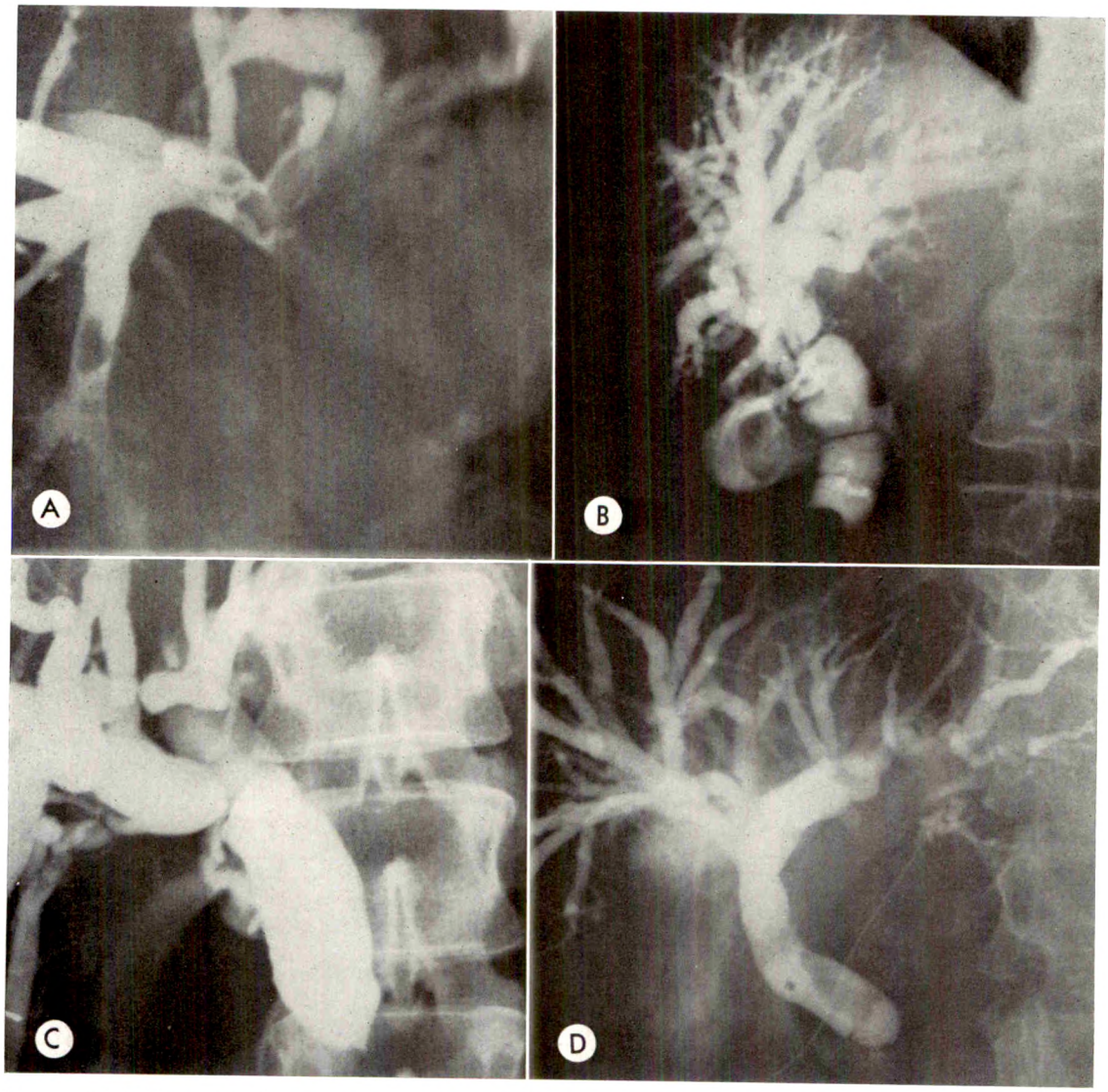


FIG. 4. (*A*) Biliary calculi, one of which is impacted below confluence of right and left hepatic ducts and is producing smooth, downwardly concave meniscus in contrast medium. (*B*) Carcinoma of the ampulla of Vater. (*C*) This downwardly convex meniscus was seen in 6 cases of carcinoma of the head of pancreas. Note "nipple" in center of meniscus. This patient had not had previous biliary tract surgery. (*D*) Benign stricture of common duct in patient who had had previous cholecystectomy and common duct exploration. Note elongated filling defect caused by a mucus floccule. Small, round, filling defect adjacent to this is also mucus.

pearance may be seen with benign strictures, but the degree of ductal dilatation is usually less and there usually is a history of previous biliary tract surgery (Fig. 4*D*).

COURSE OF EXTRAHEPATIC DUCTS

These were distorted by extrinsic tumor masses compressing and partly obstructing them in 5 cases of metastatic malignancy involving the hilar lymph nodes (Fig. 5). In another case in which there were hilar and liver metastases, injection of the bile ducts at autopsy showed the intrahepatic ducts to be distorted and stretched around the parenchymal tumor nodules (Fig. 6, *A* and *B*). In the case of chronic pancreatitis,

the extrahepatic ducts were dilated proximally with a gradual, smooth tapering of the intrapancreatic portion of the common duct which was compressed medially by a pseudocyst just above the ampulla (Fig. 7). The smooth outlines of the narrowed duct suggest a benign process but carcinoma of the head of the pancreas could conceivably produce a similar appearance in the early stages.

CONSISTENCY OR INCONSISTENCY IN CALIBER OF INTRAHEPATIC DUCTS

Multiple short strictures of the intrahepatic ducts with slight dilatation proximal to them were seen in 3 patients. One of these had small free stones in the left hepatic duct; this patient had a history of recurrent stones and a left hepatic duct stricture. The stones were removed at operation, and biopsy revealed periductal fibrosis. This was thought to be chronic fibrosing inflammatory duct disease secondary to the recurrent stones and previous stricture. A histologic diagnosis was not available in the 2 other cases since operation would not have been of benefit in either of them. The clinical course in 1 suggested the possibility of a sclerosing bile duct carcinoma but autopsy was refused (Fig. 8).



Fig. 5. Eccentric extrinsic compression of extrahepatic ducts (arrows) by metastatic nodules of gastric carcinoma in hilar lymph nodes.

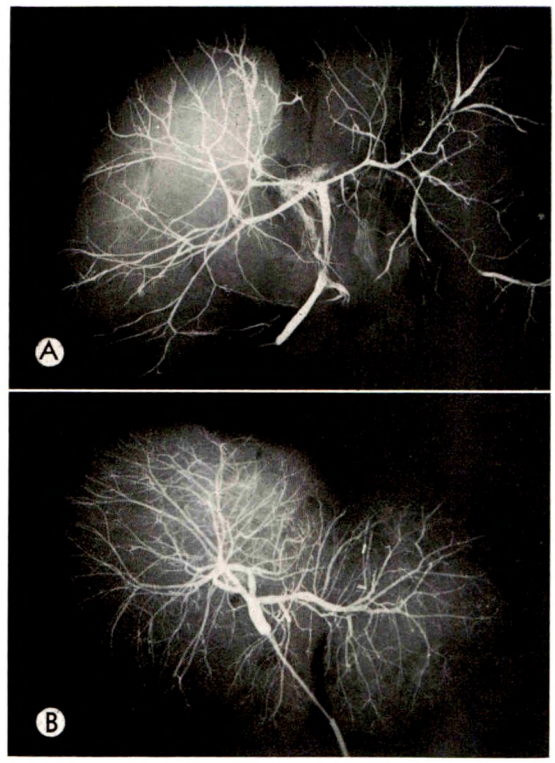


Fig. 6. (*A*) Biliary tree injected with barium at autopsy through cannula in ampulla of Vater, showing extrinsic compression of common duct and distortion of intrahepatic ducts by metastatic gastric carcinoma. Note stretching of intrahepatic ducts around tumor nodules with slight dilatation of ducts distal to compressed areas. (*B*) Autopsy injection of normal biliary tree for comparison. Catheter is in common hepatic duct.



Fig. 7. Smooth, circumferential narrowing of intrapancreatic portion of common bile duct due to chronic fibrosing pancreatitis. Compression on medial side of duct above ampulla was due to a pseudocyst.

INTRADUCTAL FILLING DEFECTS

These vary in number, shape, and size and, apart from polypoid tumors, are due to stones, debris, or mucus. Unless impacted, stones may move with a change in the patient's position but do not change

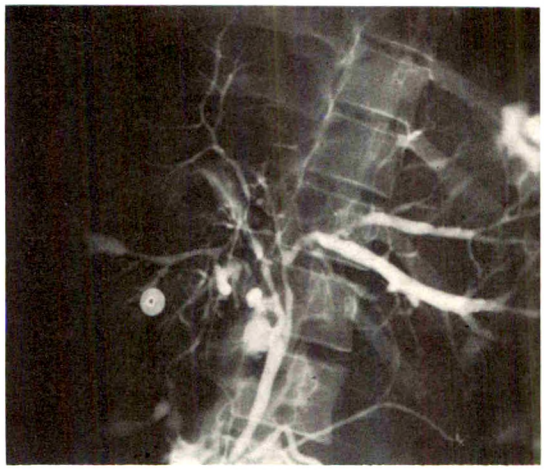


FIG. 8. Diffuse sclerosing duct disease thought clinically to be sclerosing carcinoma. No histologic proof was available.

shape, whereas mucus may change shape. Mucus floccules also tend to be elongated,[5] whereas stones more commonly are rounded. Debris tend to be amorphous and sometimes diffuse, giving a double contrast appearance to the cholangiogram (Fig. 9), or, rarely, may form casts of the ducts.

CAVITIES WITHIN LIVER

Cavities within the liver can be distinguished from intraparenchymal injections by their sharp outlines[5] and occasional communication with the bile ducts (Fig. 10*A*). Multiple tiny contrast-filled cavities, resembling bunches of grapes, were seen surrounding the smaller ducts in 1 patient (Fig. 10*B*) who had a stricture complicated by *Escherichia coli* infection of the bile. We were not able to prove their etiology, but a similar picture, obtained by other authors,[5] was found to be due to multiple small abscesses.

FISTULAE

Fistulae between the biliary tract and intestine present no diagnostic difficulty.

UNSUCCESSFUL STUDIES

Twenty-one of the 28 patients on whom attempted cholangiography was unsuccessful had parenchymal liver disease, but the 7 others proved to have obstruction at operation. This substantiates the experience of others[8] and modifies earlier assumptions that failure to puncture a duct is almost certain evidence of lack of obstruction and dilatation.[10]

Therefore, if cholangiography is unsuccessful, the probability of parenchymal disease outweighs that of obstruction by 3 to 1, but the latter is not ruled out and the clinical picture should be carefully studied in this light before proceeding with or cancelling laparotomy.

DISCUSSION

A successful percutaneous cholangiographic study enables the radiologist to distinguish extrahepatic obstruction from parenchymal liver disease and to arrive at a preoperative diagnosis of the nature of the lesion in the majority of cases. This information is of immense value to the surgeon because it facilitates preoperative



FIG. 9. Debris impacted 3 cm. above stricture of common hepatic duct. Note "double contrast" appearance of cholangiogram. Debris has partially solidified to form cast of portion of duct in lower right hand corner.

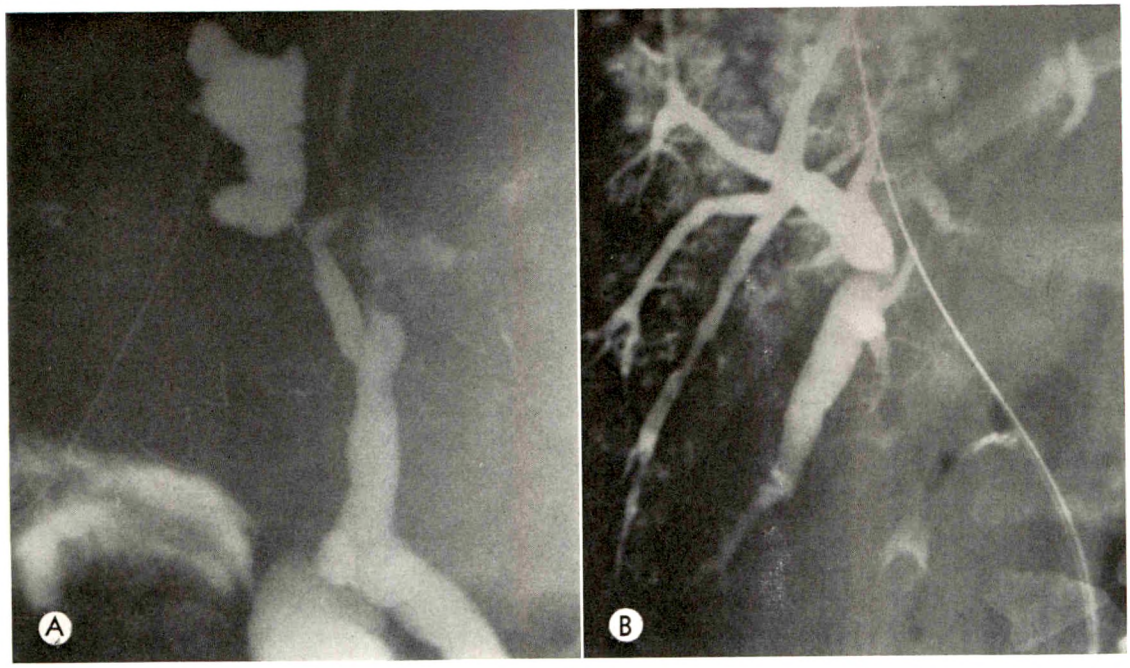


FIG. 10. (*A*) Old fibrotic abscess cavity which communicates with the right hepatic duct. Note distinct outline compared with fuzzy margin of intraparenchymal injection in the lower right lobe. Large collection of contrast medium is in the duodenum. (*B*) Multiple small acinar collections of contrast medium on either side of larger biliary radicles in patient who had stricture of right hepatic duct complicated by *Escherichia coli* infection of bile. Attempts to puncture the left hepatic duct failed and it could not be located at operation. The extrahepatic ducts were necrotic. This appearance may be due to multiple tiny abscess cavities.

planning and interoperative identification of hilar or intrahepatic strictures. It also justifies avoidance of operation and its coincident increased morbidity in patients with parenchymal disease. If good quality cholangiograms are obtained, the radiologist should be able to make a differential diagnosis in the light of the clinical history. An unsuccessful study must be interpreted with caution in the light of the clinical findings. The procedure is seldom used when clinical evidence strongly supports a diagnosis of neoplastic obstruction; it is most often used to resolve problems in differential diagnosis and to localize sites of benign stricture.

Annular and polypoid carcinomas of the bile ducts have an appearance similar to that seen in the colon. Benign polypoid lesions of the bile ducts are rare, but we had 1 "polyp" due to scar tissue. Complete obstruction due to an impacted gallstone produces a downwardly concave meniscus, whereas advanced carcinoma of the head of the pancreas and some benign strictures produce a downwardly convex meniscus. Carcinoma of the head of the pancreas is likely to result in a greater dilatation of the intrahepatic ducts than that due to benign stricture. Benign stricture is unlikely to occur without a history of previous biliary surgery. The common duct is extrinsically compressed in an eccentric fashion by hilar metastases, whereas it has a smooth tapered appearance when circumferentially narrowed in its intrapancreatic portion by fibrosing pancreatitis. Multiple short intrahepatic strictures appear to be due to either chronic fibrosing inflammatory duct disease or the rare diffuse sclerosing type of bile duct carcinoma. However, we did not have a histologic diagnosis in 2 of the 3 patients with this appearance and cannot arrive at any definite conclusion. Free stones, mucus, or debris cause intraductal filling defects. The margins of intrahepatic cavities are

more sharply defined than the fuzzy outline of an intraparenchymal injection.

SUMMARY

We studied 88 attempts at cholangiography on 87 patients.

Sixty were successful and we analyzed these in an effort to establish criteria for the differential diagnosis of bile duct pathology.

Twenty-eight were unsuccessful; operative or T-tube cholangiograms were studied in 10 of these cases and biopsy results in 9, in order to determine the reason for failure and its inferential value. Seven of the 28 patients in whom cholangiography was unsuccessful were found to have obstructed bile ducts at operation.

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REFERENCES

1. Burckhardt, H., and Müller, W. Versuche über die Punktion der Gallenblase und ihre Röntgendarstellung. *Deutsche Ztschr. Chir.*, 1921, *162*, 168–197.
2. Carter, R. F., and Saypol, G. M. Transabdominal cholangiography. *J.A.M.A.*, 1952, *148*, 253–255.
3. Flemma, R. J., Schauble, J. F., Gardner, C. E., Jr., Anlyan, W. G., and Capp, M. P. Percutaneous transhepatic cholangiography in differential diagnosis of jaundice. *Surg., Gynec. & Obst.*, 1963, *116*, 559–568.
4. Huard, P., and Do-Xuan-Hop. La ponction transhépatique des canaux biliaires. *Bull. Soc. méd. chir. l'Indochine*, 1937, *15*, 1090–1100.
5. Isley, J. K., Jr., and Schauble, J. F. Interpretation of percutaneous transhepatic cholangiogram. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1962, *88*, 772–777.
6. Kidd, H. A. Percutaneous transhepatic cholangiography. *A.M.A. Arch. Surg.*, 1956, *72*, 262–268.
7. Mujahed, Z., and Evans, J. A. Percutaneous transhepatic cholangiography. *Radiol. Clin. North America*, 1966, *4*, 535–546.
8. Seldinger, S. I. Percutaneous transhepatic cholangiography. *Acta radiol.*, 1966, Suppl. 253, 7–133.
9. Shaldon, S., Barber, K. M., and Young, W. B. Percutaneous transhepatic cholangiography: modified technique. *Gastroenterology*, 1962, *42*, 371–379.
10. Wiechel, K. L. Percutaneous transhepatic cholangiography: technique and application. *Acta chir. scandinav.*, 1964, Suppl. 330, 5–99.

VOL. 116, NO. 2

PSEUDOCALCULUS DEFECT IN CHOLANGIOGRAPHY*

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THE roentgenographic demonstration of a defect at the distal end of the common bile duct on operative, postoperative or percutaneous cholangiography is usually due to one of the following conditions: (1) a calculus (Fig. 1); (2) a tumor of the common bile duct, ampulla of Vater, duodenum, or the pancreas (Fig. 2); (3) edema of the ampulla of Vater; and (4) spasm of the sphincter of Oddi. The last 2 conditions may be caused by instrumentation during the operative procedure.

Another less commonly encountered defect, probably due to prominence of the musculature at the distal end of the common bile duct, has been referred to by Beneventano and Schein[1] as the pseudocalculus sign, and by Caroli *et al.*[4] as the pseudocalculus formation. Its roentgenographic appearance, as seen in 3 patients we recently examined, is illustrated in Figures 3, *A* and *B*; 4, *A* and *B*; and 5, *A* and *B*. One of our 3 patients (Fig. 3, *A* and *B*) presented a characteristic clinical and roentgenographic picture. The pertinent history and the roentgenographic studies in this patient are given below.

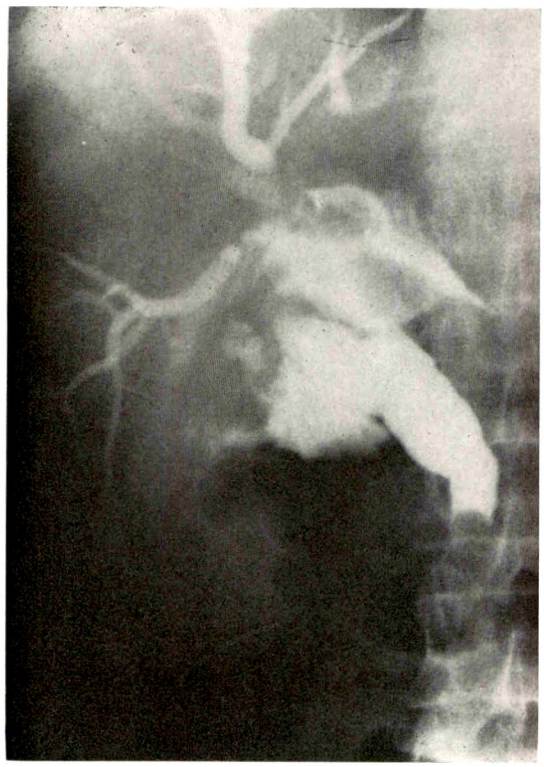


FIG. 1. Calculus at the distal end of the common bile duct seen on percutaneous cholangiography.



FIG. 2. Tumor of the ampulla of Vater seen on T-tube cholangiography.

REPORT OF A CASE

A 57 year old white male was admitted to The New York Hospital complaining of pain in the right upper quadrant of 3 days' duration. Following a diagnostic work-up which pointed to a diseased gallbladder, the patient was taken to

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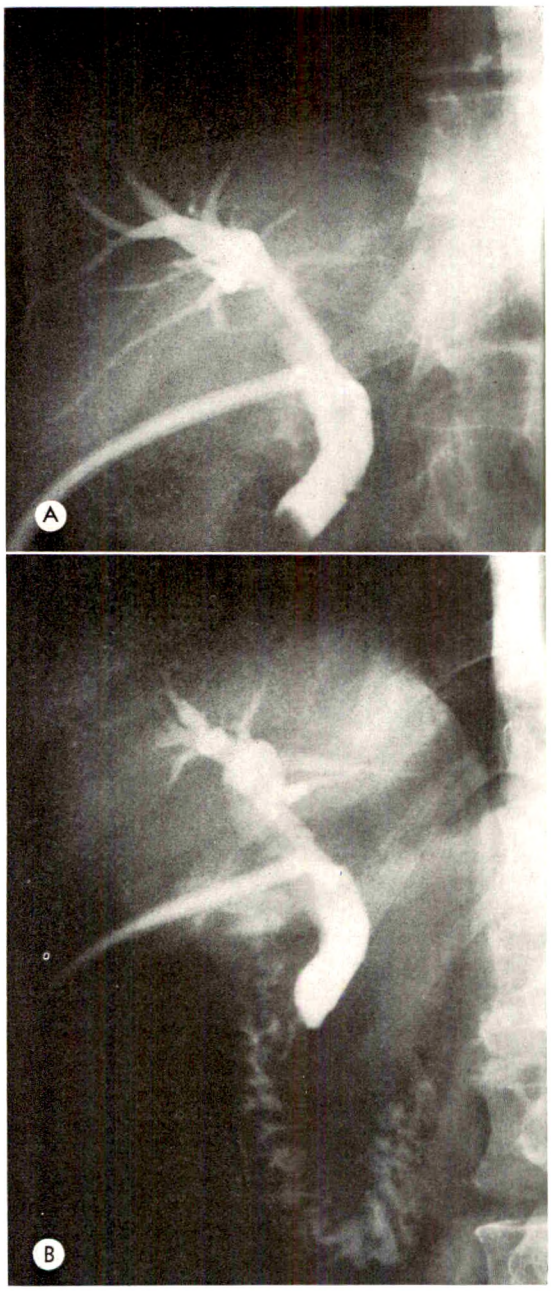


FIG. 3. (*A*) Defect at the distal end of the common bile duct extending throughout the entire lumen of the duct. The border of the defect is irregular. (*B*) This exposure was made a few minutes after *A*. The defect is no longer present. The intramural portion of the duct is well demonstrated and is normal.

the operating room for cholecystectomy. At operation the gallbladder was found to be moderately to markedly distended but not gangrenous. It contained no calculi. Inspection and palpation of the adjoining viscera showed no tumor. A diagnosis of hydrops of the gallbladder and chronic cholecystitis was made and a cholecystectomy was performed. An op-

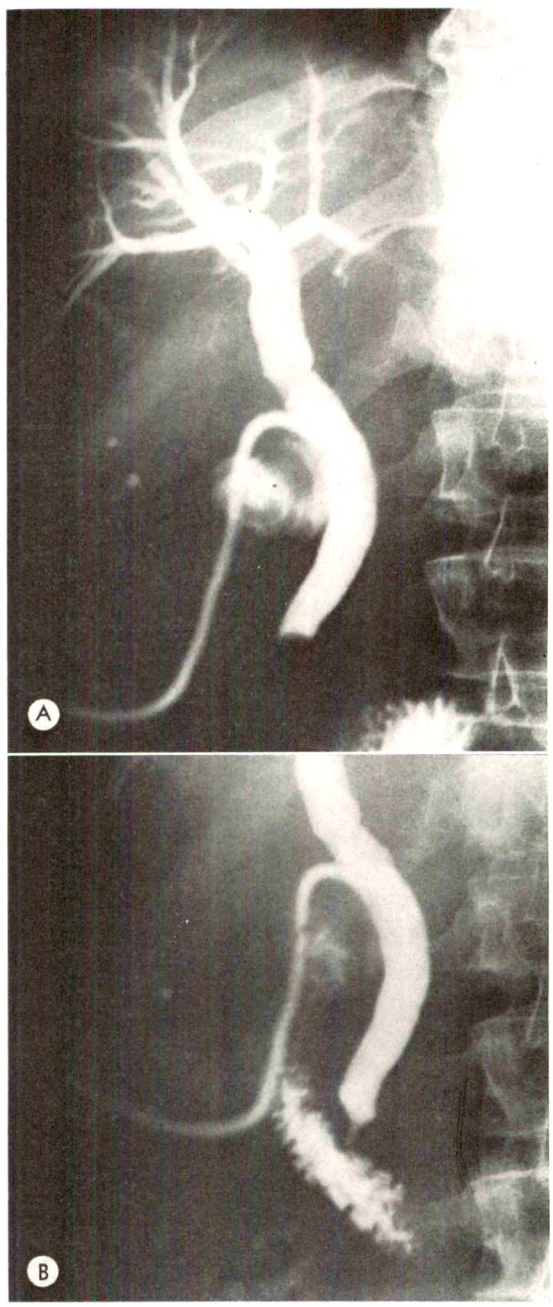


FIG. 4. (*A*) Defect at the distal end of the common bile duct. The border of the defect is smooth. (*B*) Exposure made a few minutes before *A*. The distal end of the common bile duct and intramural portion are normal.

erative cholangiogram showed a meniscus like defect at the distal end of the common bile duct. Careful palpation of the common bile duct following the cholangiographic finding did not reveal any calculi or tumefaction. Edema of the ampulla of Vater and spasm of the sphincter of Oddi were ruled out by the easy passage of a variety of silk woven bougies and stone forceps through the common bile duct and into the duodenum. A T-tube was left in the common bile duct, the abdomen was closed and the patient sent to the recovery room.

On the 10th postoperative day a T-tube cholangiography again showed the defect at the distal end of the common bile duct which, however, was inconstant, showing up on some films but not on others (Fig. 3, *A* and *B*). The passage of the contrast agent into the duodenum through the ampulla of Vater was free and the examination was otherwise normal. The patient's recovery was proceeding satisfactorily. His bile drainage was normal. The clinical picture was inconsistent with the presence of a calculus or other obstructive disease at the end of the bile duct.

On the 21st postoperative day a repeat T-tube cholangiogram was obtained, duplicating the findings of the previous 2 examinations. It was then decided to examine the common bile duct with cineradiography to study the formation of this pseudocalculus defect, its relation to the rhythmic contractions of the common bile duct and duodenum, as well as the effect, if any, of antispasmodic agents on the formation of the defect. This was done 5 weeks after cholecystectomy. The patient was still showing satisfactory recovery and had no signs or symptoms of choledocholithiasis. Cineradiography confirmed the cyclical nature of the pseudodefect at the distal end of the common bile duct, appearing during the contraction phase and disappearing with relaxation. Its formation was independent of the duodenal peristaltic activity. Spasm was thought to play no part in the causation of the defect, as the rhythmic contractions of the distal end of the common duct were unaltered. Furthermore, the demonstration of the defect on 4 cholangiographic examinations over a 5 week period suggested a permanent rather than a transient condition.

Three cc. of a 2 per cent solution of Xylocaine was nevertheless introduced into the T-tube as a topical anesthetic to counteract any spasm

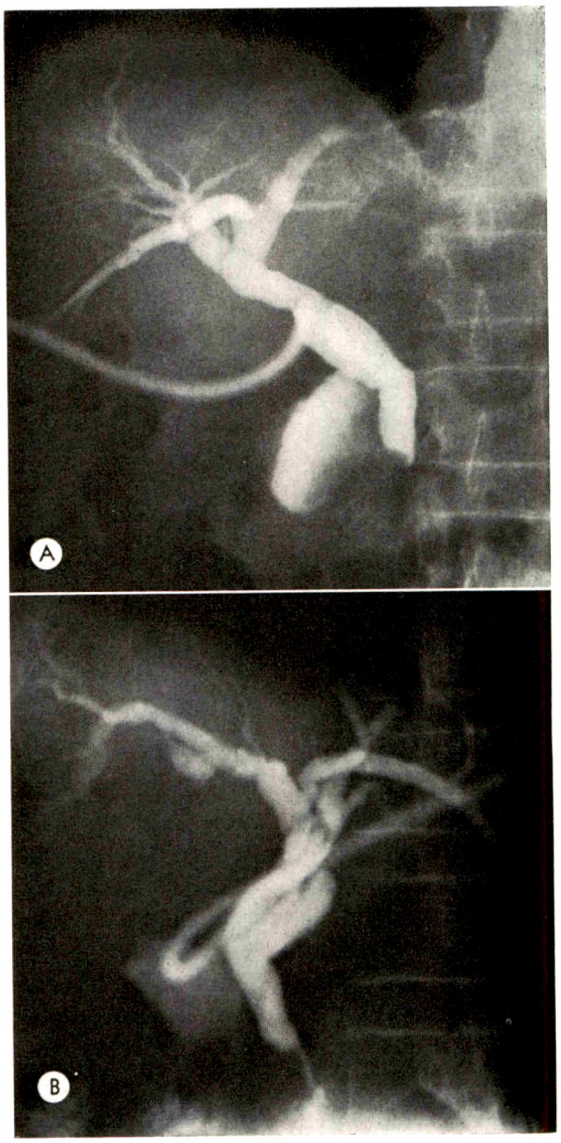


Fig. 5. (*A* and *B*) Two exposures made a few minutes apart. The defect is seen on one cholangiogram but not on the other.

that may be present. The formation of the defect as well as the cyclical contractions at the distal end of the common bile duct were not affected by the local anesthetic. About 30 minutes later the patient was given 75 Ivy units of cholecystokinin intravenously and additional cineradiographic observations recorded. Cholecystokinin has been shown to cause relaxation of the sphincter of Oddi.[3,5,6] This again failed to obliterate the pseudocalculus defect indicating that its formation was not due to any spastic condition.

The patient's T-tube was removed 1 week after his last cholangiography and 4 months after operation the patient was asymptomatic and in a satisfactory condition.

ROENTGENOGRAPHIC AND PHYSIOLOGIC CHARACTERISTICS

From a review of our cases as well as those reported in the literature[1,4] certain characteristics of the pseudocalculus defect became apparent.

Location. The defect occurs only at the distal end of the common bile duct.

Borders. Only the upper border can be seen; it is convex upward and may be smooth (Fig. 4*A*) or irregular (Fig. 3*A*); it usually covers the entire lumen of the duct without stretching it. The lower border of the pseudodefect cannot be outlined. It merges with the wall of the intramural portion of the common bile duct.

Effect on the function of the sphincter of Oddi. The defect does not cause any obstruction to the flow of bile through the sphincter of Oddi.

Cyclical nature. It is cyclic in nature, appearing during the contraction phase at the distal end of the common bile duct and disappearing during relaxation. This is easily seen at fluoroscopy and cineradiography and helps to explain its presence on some films and its absence on others during the same examination. The contraction of the distal end of the common bile duct as well as the appearance of the pseudocalculus defect is independent of the contraction of the duodenum.

Permanency. The pseudocalculus defect is probably of a permanent rather than a transient nature. As mentioned above it was seen on 4 cholangiograms over a 5 week period in 1 patient. It was seen on 2 cholangiograms at a 2 week interval in another.

Absence of pain. Distention of the common bile duct by contrast agent in the presence of obstructive or spastic disease causes crampy abdominal pain. No such pain occurs in the pseudocalculus defect condition. The contrast agent passes into the duodenum before a pain-producing pressure can build up.

ETIOLOGY

The pseudocalculus defect is seen during the rhythmic contraction at the distal end of the common bile duct. It probably is due to the prominence of the muscle fibers at the distal end of the common bile duct. The prominence of these muscle fibers may be due to their particular arrangement which makes them conspicuous roentgenographically during the contraction phase. Hypertrophy of these muscles similar to the hypertrophy in achalasia or hypertrophic pyloric stenosis as a causative agent is unlikely as these are obstructive conditions and the pseudocalculus defect is not.

Histologically the common bile duct is composed mainly of fibrous and elastic tissue. Smooth muscle fibers are present in a minimal amount except at the distal end, where there is an abundance of smooth muscles as demonstrated by Boyden.[2] This author also demonstrated that the muscle fibers of the common bile duct first appear in the human embryo about 5 weeks later than the intestinal musculature, and that it arises from separate mesenchymal tissue. The independent contraction of the distal end of the common bile duct and the duodenum, easily observed at fluoroscopy and cineradiography, is thus explained.

DIFFERENTIAL DIAGNOSIS

The pseudocalculus defect has an appearance strongly suggestive of a calculus or a tumor at the end of the common bile duct, in the ampulla of Vater or at the head of the pancreas. If unrecognized, it leads to a misdiagnosis and unnecessary surgical intervention. The main feature in the differential diagnosis is the cyclical nature observed at fluoroscopy and on the roentgenograms. The absence of obstruction to the flow of the contrast agent into the duodenum is another helpful characteristic. Pain caused by distention of the

common bile duct due to obstructive or spastic disease is absent in this condition.

SUMMARY

A defect occasionally seen at the distal end of the common bile duct is described. It is probably due to prominence or particular arrangement of the musculature at the choledochoduodenal junction.

The defect is cyclical in nature, always located at the distal end of the common bile duct and is not obliterated by the administration of antispasmodic or anesthetic agents.

If unrecognized, this defect can lead to misdiagnosis and sometimes unnecessary exploration.

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REFERENCES

1. Beneventano, T. C., and Schein, C. Y. Pseudocalculus sign in cholangiography. *A. M. A. Arch. Surg.*, 1969, *98*, 731–733.
2. Boyden, E. A. Anatomy of choledochoduodenal junction in man. *Surg., Gynec. & Obst.*, 1957, *104*, 641–652.
3. Broden, B. Experiments with cholecystokinin in cholecystography. *Acta radiol.*, 1958, *49*, 25–30.
4. Caroli, Y., Porcher, P., Pequiguot, G., and Delattre, M. Contribution of cineradiography to study of function of human biliary tract. *Am. J. Digest. Dis.*, 1960, *5*, 677–696.
5. Stassa, G., and Grafe, W. Cineradiographic evaluation of biliary tract after drug therapy following cholecystectomy, sphincterectomy and vagotomy. *Radiology*, 1967, *91*, 297–301.
6. Torsoli, A., Ramorino, M. L., Colaragrande, C., and Demais, G. Experiments with cholecystokinin. *Acta radiol.*, 1961, *55*, 193–206.

IATROGENIC HEPATIC DUCT RUPTURE*

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CALCULI are found within the hepatic ducts in 24 per cent of patients with choledocholithiasis at surgery.[9,10] Because they frequently are responsible for retained stones, better methods for their removal are necessary. Several authors have described the use of a Fogarty balloon-tipped arterial embolectomy catheter for the removal of biliary calculi.[1,4,6] This catheter has been replaced by a specially designed Fogarty biliary catheter. Shore and Berci[10] recently described their experience with operative management of hepatic duct calculi using the Fogarty biliary catheter, fiberoptic choledochoscopy, suction tips, scoops, forceps and a new directable balloon catheter.

Two patients in our hospital with iatrogenic hepatic duct rupture discovered by operative cholangiography, and the recent papers by Henzel,[5] and Eaton[2] have prompted this report.



FIG. 1. Case 1. Calculus in distal common bile duct discovered on postoperative T-tube cholangiogram. Note normal appearing right hepatic ducts.

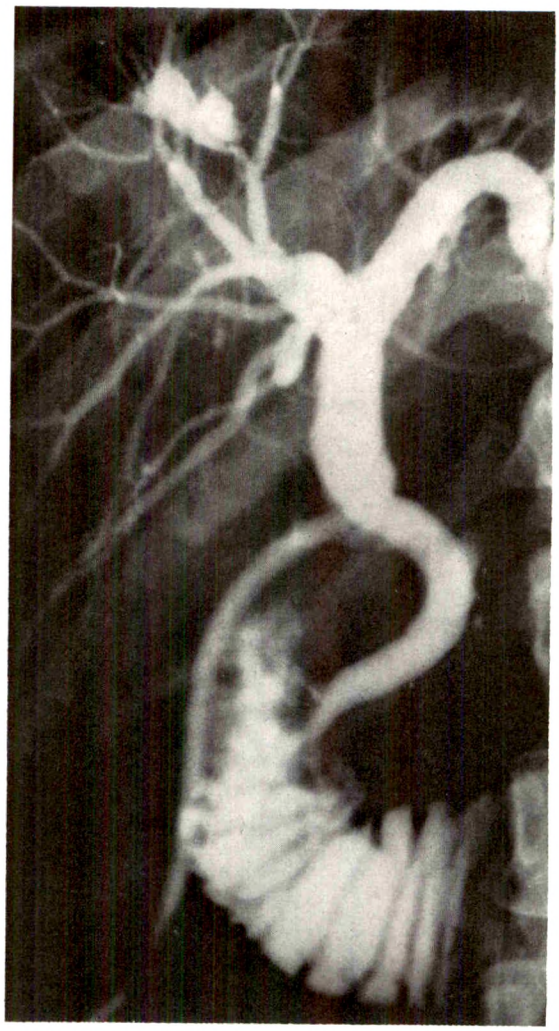


FIG. 2. Case 1. Intraoperative cholangiogram shows iatrogenic biliary disruptions of a ventrocranial branch of the right hepatic duct.

REPORT OF CASES

CASE 1. M.L.B., a 37 year old female, was admitted for retained common bile duct cal-

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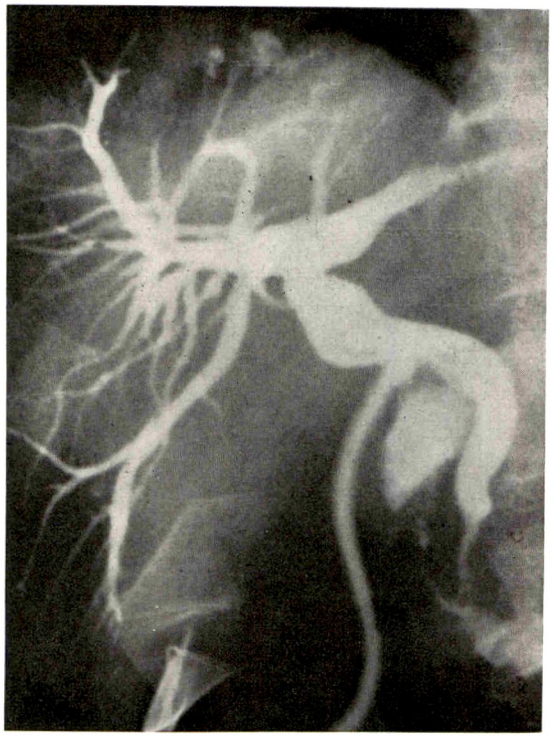


Fig. 3. Case II. Postoperative T-tube cholangiogram shows retained common bile duct calculi (not well demonstrated) and normal right hepatic ducts. The density just below the diaphragm represents a calcified lung granuloma.

culus following cholecystectomy and choledocholithotomy 5 months earlier (Fig. 1).

During the interval between hospitalizations, the patient had been asymptomatic and without clinical evidence of biliary tract obstruction.

Re-exploration of the common bile duct with passage of the Fogarty catheter proximally into both major hepatic radicals produced no stone. It had presumably passed into the duodenum.

Operative cholangiogram revealed biliary disruption of a ventrocranial branch of the right hepatic duct (Fig. 2).

The patient's recovery was uneventful without pain, temperature elevation or hematobilia.

Case II. This 63 year old male admitted with acute pancreatitis, was treated successfully with nasogastric suction, intravenous fluids and antibiotics. During this hospitalization, intravenous cholangiography showed nonfilling of the gallbladder and choledocholithiasis.

Cholecystectomy and common bile duct exploration with removal of calculi were performed.

A postoperative T-tube cholangiogram revealed retained stones in the common bile duct presumably arising from small hepatic ducts (Fig. 3).

This patient was re-admitted 2 months later for re-exploration of his common bile duct. Multiple calculi were then removed from his common bile duct. An intraoperative T-tube cholangiogram showed no evidence of retained stone and intrahepatic biliary rupture (Fig. 4).

A postoperative T-tube cholangiogram 10 days later showed no evidence of stones or extravasated contrast material (Fig. 5).

This patient had intermittent temperature elevation postoperatively with 3 positive blood cultures of *E. coli* and *Enterobacilli*. The infection was treated with antibiotics and the subsequent course was uneventful.

DISCUSSION

With the more aggressive surgical management of biliary calculi, an increase in the frequency of iatrogenic biliary rupture can be expected. Biliary duct perforation, duct stricture, hematobilia, and abscess formation may result even with the gentle passage of a Fogarty balloon catheter. The

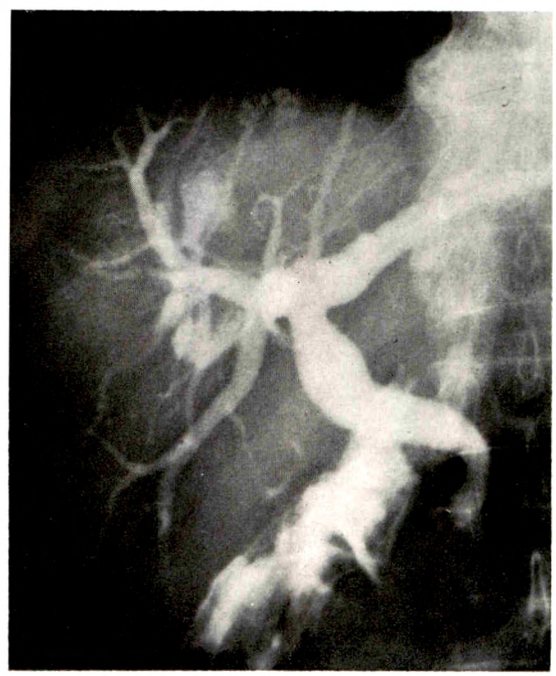


Fig. 4. Case II. Intraoperative cholangiogram shows iatrogenic hepatic duct rupture.

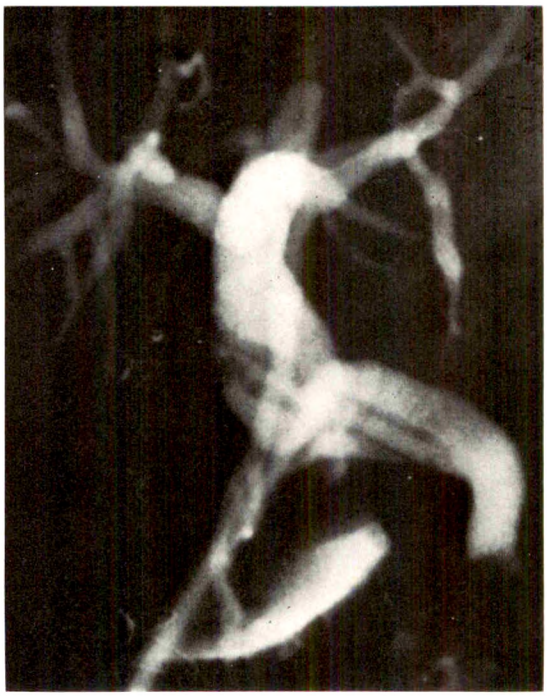


FIG. 5. Case II. Postoperative spot cholangiogram 10 days after Figure 4 shows no extravasated contrast material from the site of the rupture.

roentgenographic appearance of extravasated contrast material adjacent to the intrahepatic biliary duct is characteristic. There should be no confusion with hepatic duct diverticula or other congenital anomalies,[3,7,8] if an operative cholangiogram obtained before the use of Fogarty catheters or other instrumentation is available for comparison.

Henzel[5] recommends minimal and gentle inflation of the balloon with careful insertion and withdrawal of the biliary catheter. If hepatic duct rupture occurs, the bile should be cultured, with appropriate antibiotics given, if found positive. Continued external biliary drainage should be considered until resolution of the cavity has occurred, to avoid hepatic abscess formation.[5]

SUMMARY

Two examples of iatrogenic hepatic duct rupture following duct instrumentation are reported.

Extreme care must be exercised to avoid the consequences of duct perforation, structure, abscess formation or hematobilia.

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REFERENCES

1. Berman, I. R., and Pfeffer, R. B. Technique of extraction of hepatic duct calculi with modified Foley catheter. *Am. J. Surg.*, 1967, *114*, 969–971.
2. Eaton, S. B. Iatrogenic liver injury resulting from ductal instrumentation with Fogarty-biliary balloon catheter. *Radiology*, 1971, *100*, 581–584.
3. Esguerra-Gómez, G., and Riveros-Gamboa, E. Case of multi-diverticular cystic dilatation of common and hepatic ducts. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1965, *94*, 477–479.
4. Fogarty, T. J. Evaluation of improved operative technique in common duct surgery. *Am. J. Surg.*, 1968, *116*, 117–183.
5. Henzel, J. H. Intrahepatic biliary disruption. *A. M. A. Arch. Surg.*, 1971, *102*, 218–220.
6. Knight, C. D. Use of balloon-tipped catheter in exploration of common duct. *Am. J. Surg.*, 1967, *113*, 717–718.
7. Maxwell, J. W. Hepatodochal diverticulum: new entity. *Am. J. Surg.*, 1967, *33*, 153–158.
8. Mujahed, Z., Glenn, F., and Evans, J. A. Communicating cavernous ectasia of intrahepatic ducts (Caroli's disease). Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *113*, 21–26.
9. Norman, O. Studies on hepatic ducts in cholangiography. *Acta radiol.*, 1951, Suppl. 84.
10. Shore, J. M., and Berci, G. Operative management of calculi in hepatic ducts. *Am. J. Surg.*, 1970, *119*, 625–631.

PLAIN FILM ROENTGENOGRAPHIC FINDINGS IN ALVEOLAR HYDATID DISEASE— *ECHINOCOCCUS MULTILOCULARIS**

By WILLIAM M. THOMPSON, M.D.,†§ DAN P. CHISHOLM, M.D.,† *and* ROBERT TANK, M.D.‡
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ALVEOLAR hydatid disease is caused by the larval stage of a tape worm, *Echinococcus multilocularis*.[32] It differs from the more common and better known cystic hydatid disease (*E. granulosus*) in host response, clinical course, and roentgenographic appearance.[7,24,31–33,44,45] The cestodes are distinguished by morphologic and biologic characteristics in both the larval and strobilar stages. The differences in the 2 diseases are not well known in North America, since much of the information on alveolar hydatid disease is in the foreign literature.

Alveolar hydatid disease has long been known to occur in man in central Europe and Siberia.[32] Until recently, the causative organism in North America was known to occur only in the arctic zone of northern and western Alaska and Canada. However, Leiby *et al.*,[23] and Rausch[32] have reported *E. multilocularis* in carnivores and rodents in 5 of the north-central states. Since mammals of the same species have extensive distributions in the United States, gradual spread of the parasite is to be expected. In urban areas, the possibility exists that a domestic cycle, involving house mice and cats, might become established. Foxes are the most common definitive host of *E. multilocularis*, and arvicoline and cricetine rodents (voles, lemmings, and wild mice) serve as intermediate hosts (Fig. 1).

Two forms of cystic hydatid disease caused by the larval *E. granulosus* are recognized: sylvatic, which is endemic in northern regions (including Alaska and Canada), and the better known pastoral, which is world-wide in distribution.[13,32,46] There is some evidence that 2 biologically distinct strains of *E. granulosus* are responsible for the 2 types of cystic hydatid disease.[46] The wolf is the main definitive host for the sylvatic strain of *E. granulosus*, and wild ruminants (moose and reindeer in North America) serve as intermediate host. The dog is the important definitive host in the pastoral cycle, and domestic ungulates (cattle, sheep, and swine) serve as intermediate host. Man accidentally becomes an intermediate host when he ingests the infective embryophores (eggs) excreted in the feces of infected carnivores (Fig. 1).

The larval stage of these cestodes is responsible for disease in man. After being ingested, the embryophores hatch in the duodenum, and the embryos (oncospheres) penetrate the intestinal wall and enter the portal circulation.[7,31–33] In the case of *E. multilocularis*, the oncospheres localize in the liver; those of *E. granulosus* most commonly localize in the liver and lungs, but may also be distributed to other sites. The larvae of *E. multilocularis* bud outward, exogenously, into the host tissue. The tissue reaction evoked does not result in formation of a limiting capsule, but, similarly to cancer, the larval cestode invades and destroys the affected organ. Infection may spread by extension to adjacent organs. A chronic, indolent, granulomatous reaction can occur, with central necrosis, cavitation,

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The work in this paper reflects the views of the authors and not the Federal Government of the United States.
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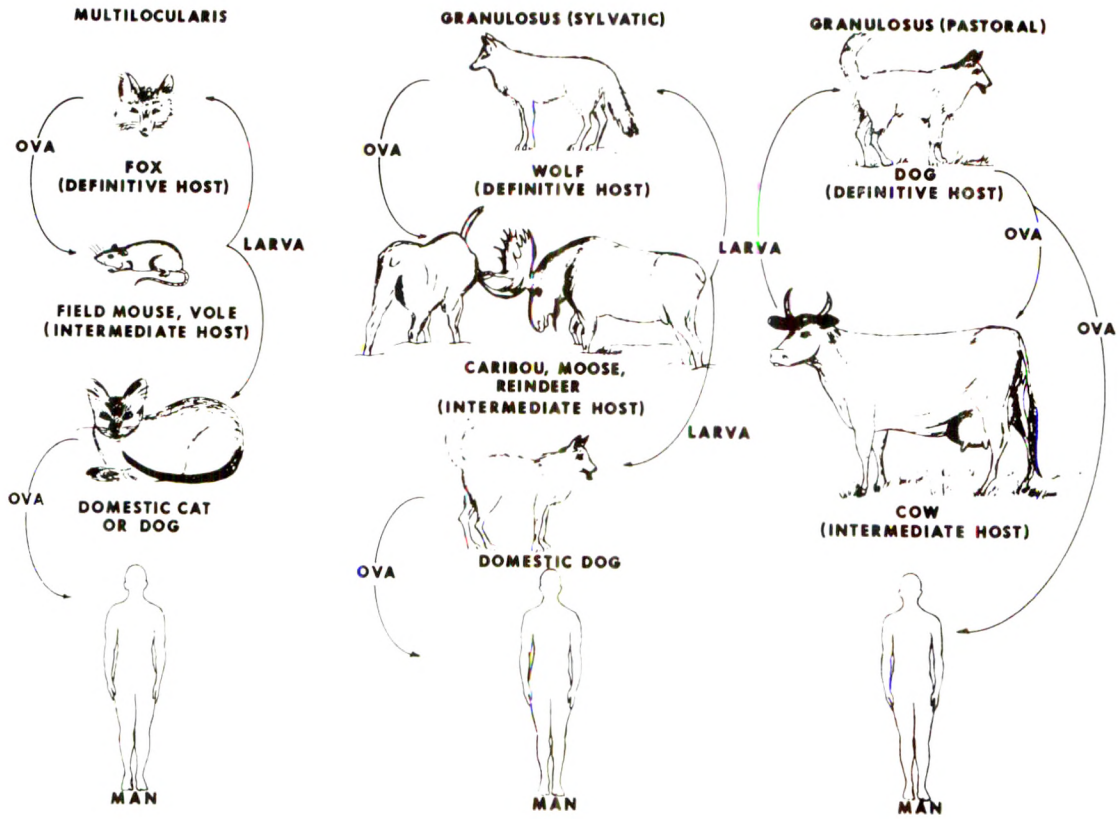


Fig. 1. Life cycles of the 3 forms of hydatid disease: *E. multilocularis*, *E. granulosus* (sylvatic) and *E. granulosus* (pastoral). The most common hosts are represented.

and calcification.[24,31–33,45] Elements of the larvae may also spread hematogenously and cause metastatic foci. In contrast, the larval *E. granulosus* is a fluid-filled cyst composed of the laminated membrane lined by the germinal membrane. The larva is enclosed by a dense pericyst derived from the tissue of the host.[8] Secondary cysts (daughter cysts) may be formed within the cyst-cavity, or may develop from protoscolices; when cysts are ruptured, these elements may cause secondary, disseminated disease. The lesions caused by both *E. granulosus* and *E. multilocularis* may calcify. Differences in pattern of growth and in the nature of the tissue reaction of the host account for the diverse clinical findings.[31–33,45]

The spectrum of clinical responses to the larval *E. multilocularis* ranges from benign to malignant. The patients may have the diagnosis made incidentally, from typical hepatic calcification seen on routine roentgenograms. However, others present with symptoms and findings similar to those of carcinoma of the pancreas.[44,45] The disease may ultimately be fatal because of hepatic failure or effects of metastatic lesions. Even in major endemic areas, the diagnosis may be difficult to establish without surgery. In contrast, there is usually little or no clinical response in the sylvatic type of cystic hydatid disease.[46] With the more pathogenic pastoral type, mortality ranging from 4 to 10 per cent is reported.[46]

The flocculation and hemagglutination tests are the best immunodiagnostic techniques for alveolar hydatid disease, but a low rate of positive reactions (15 to 30 per cent) is obtained in patients with either type of cystic hydatid disease.[18,46] The management of patients with alveolar hydatid disease has ranged from no treatment to

hepatic resection. Patients with hydatid disease caused by the sylvatic type of *E. granulosus* have been treated conservatively, but those with the disease of the pastoral type have required surgical intervention. There is no effective chemotherapy known for any form of hydatid disease.

There have been a number of reports concerning the roentgenologic characteristics of cystic hydatid disease,[8] but little has been written about the roentgenologic characteristics of alveolar hydatid disease. The radiologist should be familiar with the plain film roentgenographic findings in alveolar hydatid disease, because of its serious nature, and because the causative organism may become endemic throughout the United States.

MATERIAL

Twenty-seven cases of alveolar hydatid disease (*E. multilocularis*) have been diagnosed in Alaskan Natives. Eight of these were previously reported by West *et al.*[44,45] In North America, only 5 cases[12,16,21,39,40,42] have been reported from outside Alaska, with only 2 cases autochthonous; 1 from Canada[16] and 1 from St. Louis, Missouri.[12]

Examples will be presented from our 27 cases to illustrate the characteristic plain film roentgenographic findings.

REPORT OF CASES

Case 1. W.M., a 35 year old Eskimo male, was admitted on January 4, 1967, with a history of pain and a mass in his right upper quadrant of 5 months' duration. He owned a dog team and lived in northwestern Alaska, where *E. multilocularis* is most prevalent. Physical examination of his abdomen revealed a hard mass in the right upper quadrant 3 fingerbreadths below the right costal margin. The mass was not separable from the liver. His liver on the left extended 2 fingerbreadths below the costal margin, with slight tenderness noted. Laboratory studies indicated an alkaline phosphatase of 5.9 sigma units (normal 2.8) and a B.S.P. retention of 9 per cent (normal 5 per cent). The echinococcal hemagglutination titers were positive, the highest being 1:2,048. A titer of 1:128 is considered positive. Three flocculation titers were positive, in 1:40 and 1:20 dilutions (1:5 is considered positive). The echinococcal skin test was positive. A roentgenogram of the abdomen revealed a large homogeneous mass in the upper abdomen (Fig. 2). No calcifications were noted. Liver scan showed a large area of central replacement of the enlarged left lobe of the liver and areas of decreased activity in the right lobe. The patient underwent exploratory laparotomy and the diagnosis of alveolar hydatid disease involving both lobes of the liver was confirmed. The lesion was unresectable and the patient was discharged with a poor prognosis.

He had 2 follow-up visits, in 1968 and 1971, which showed signs of increasing disease with the alkaline phosphatase and hemagglutination and flocculation titers all increasing. His liver scans showed increased destruction of the liver, particularly in the left lobe. However, clinically he was no worse, and if anything, he had noted a reduction in pain, and the mass had not increased in size.

Comment. This case exemplifies alveolar hydatid disease presenting with gross hepatomegaly without hepatic calcifications. Even though the patient's long term prognosis is poor, he may survive a number of years, as have a number of the patients with similar findings and prognosis reported by West *et al.*[45]

Case 11. I. A., a 42 year old asymptomatic Eskimo female from northwestern Alaska, was admitted for evaluation of strongly positive echinococcal hemagglutination and flocculation titers. She had skinned many foxes, and there were many dogs in her village. She had noted occasional postprandial epigastric discomfort,

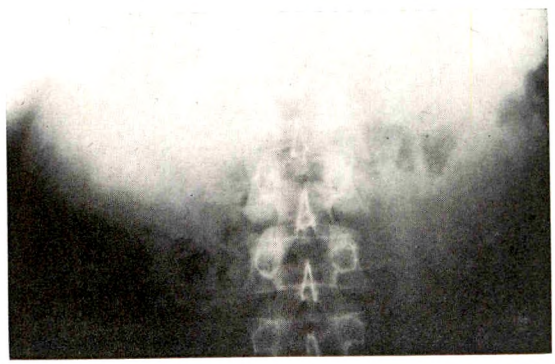


Fig. 2. Case 1. Plain roentgenogram of the abdomen shows a mass density in the upper abdomen, particularly on the right. Hepatomegaly was confirmed by physical examination and liver scanning.

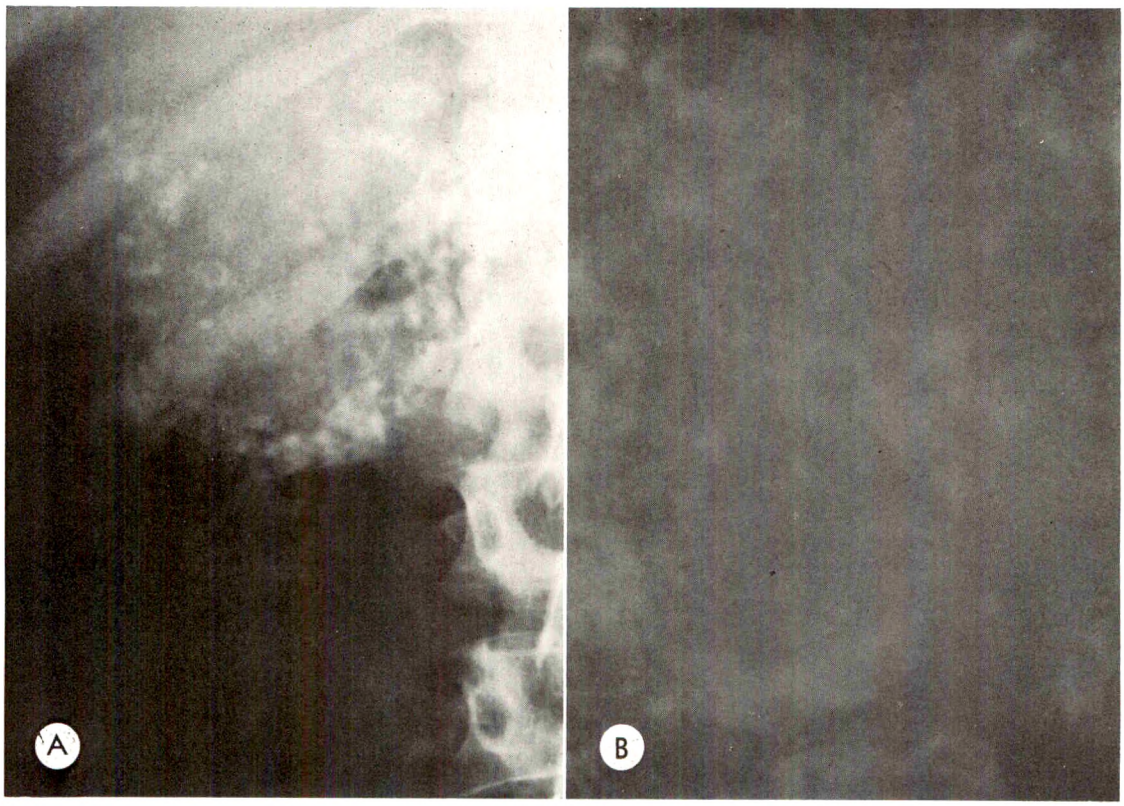


FIG. 3. Case 11. (*A*) Oblique roentgenogram of the right upper quadrant demonstrates typical hepatic calcification of alveolar hydatid disease. The calcified tuberculous nodes overlie the spine. (*B*) Enlargement of the center of *A* showing radiolucencies surrounded by faint calcification. This finding is thought to be diagnostic of alveolar hydatid disease.

but otherwise denied gastrointestinal symptoms. Review of systems and medical history were unremarkable except for a history of tuberculosis as a child. Physical examination revealed a diffusely nodular and slightly tender liver to be palpable 5 cm. below the right costal margin. The remainder of the examination was negative. The only abnormal laboratory values were an 8 per cent eosinophilia and the abnormal hemagglutination and flocculation titers, 1:2,048 and 1:80, respectively. Plain film roentgenograms of the abdomen and tomograms of the liver revealed calcifications within the anterior portion of the liver characteristic of *Echinococcus multilocularis* (Fig. 3, *A* and *B*). The calcifications were present on a lumbosacral spine roentgenogram made in 1967, and they had definitely increased in size over the ensuing 4 years. A number of other calcifications noted in the abdomen were thought to represent calcified tuberculous nodes. These had not changed since 1967. No obvious increase in hepatic size was noted. Liver scan showed a large area of decreased uptake anteriorly in the region of the porta hepatis. Cholelithiasis was demonstrated on oral cholecystogram, and the calcified mass was noted to be anterior to the gallbladder. Hepatic arteriogram showed an anterior avascular mass in the liver, which possibly involved both lobes. No additional lesions were identified. The patient underwent cholecystectomy. A pedunculated echinococcal lesion was noted to be attached to the anterior inferior edge of the liver, involving the right and left lobes without extensive parenchymal invasion in the bulk of the liver. The lesion was removed surgically (Fig. 4; and 5, *A* and *B*). The patient recovered and was discharged in satisfactory condition.

On gross pathologic examination, the resected hepatic tissue, which measured 10×8×8 cm., was virtually replaced by a large cyst with a thick and irregular wall. At the periphery of this large cyst were many small cysts, 5 to 6

mm. in diameter, many of which budded from the wall of the large cyst (Fig. 5, *A* and *B*), while some were discrete within the adjacent liver parenchyma. The large cyst was filled with thick, white, pasty material and contained translucent membranes, which formed small translucent spheres 2 to 4 mm. in diameter. The small peripheral cysts were lined by such membranes, and many contained clear fluid.

Microscopically, the material from the large cyst was amorphous, diffusely stippled with finely divided calcium, and contained pale eosinophilic laminated membranes which were acellular and were PAS positive and diastase resistant. The peripheral cysts were lined by identical membranes. The latter were surrounded by fibrous walls which were, in turn, separated from the liver parenchyma by a thin rim of granulation tissue and inflammatory cells. The calcification associated with these smaller cysts was primarily between the parasitic membranes and adjacent fibrous wall, both as finely divided calcium and as somewhat larger granules. Focally, there were small calcific deposits between laminae of the parasitic membrane. There were, in addition, focal granulomas in the hepatic parenchyma, with necrotic centers and peripheral epithelioid cells, multinucleated giant cells, and inflammatory cells. No germinal epithelium or scolices were identified (Fig. 6).

A calcified lymph node was removed. This lymph node contained fibrocalcific changes, obviously old and nonspecific, consistent with old mesenteric tuberculous lymphadenitis.

Comment. The majority of the calcific density

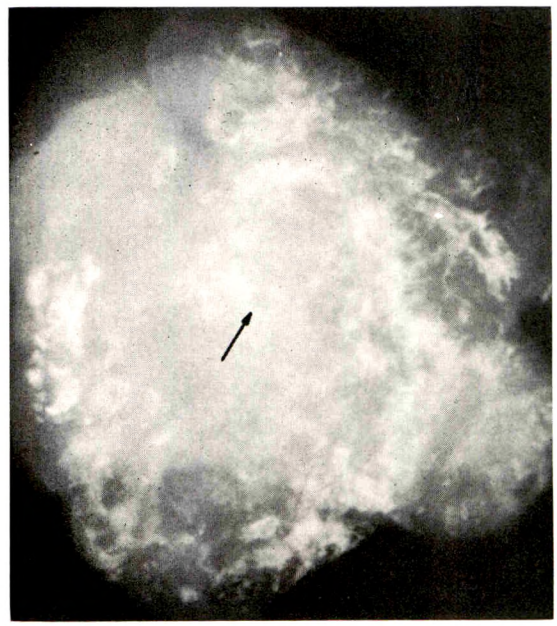


Fig. 4. Case 11. Roentgenogram of the resected echinococcal lesion. Both scattered amorphous nondiagnostic calcium and the small radiolucencies surrounded by calcium, thought to be diagnostic of alveolar hydatid disease (arrow), are demonstrated.

produced by this patient's lesion is nondiagnostic. However, the pattern produced by the calcification, demonstrated roentgenographically in the central area of the calcific mass, is typical of alveolar hydatid disease (Fig. 3, *A* and *B*). The small spherical lucencies rimmed by calcium represent cysts with reactive tissue surrounding the walls, which had become

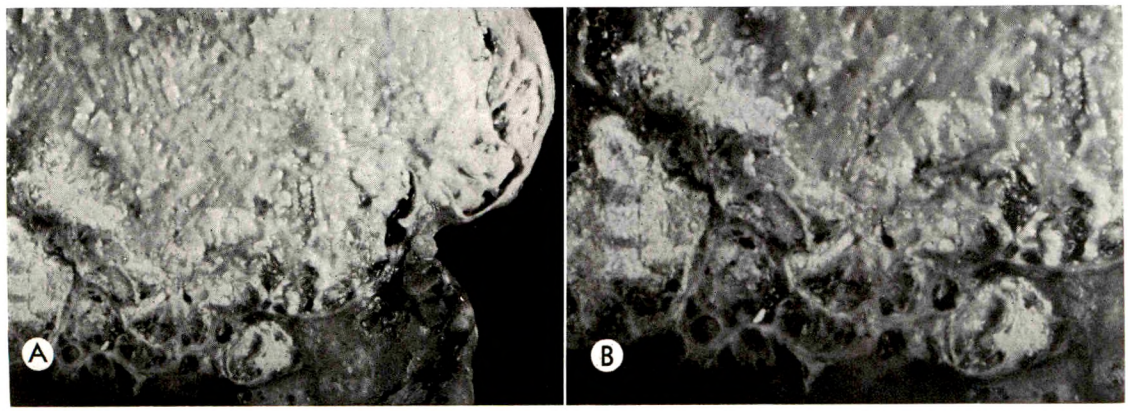


Fig. 5. Case 11. (*A*) Section of the resected echinococcal lesion showing necrotic amorphous material (center) and multiple small cysts (best demonstrated at the periphery of the lesion—bottom). (*B*) Enlargement of cysts at the periphery of the multilocular lesion.

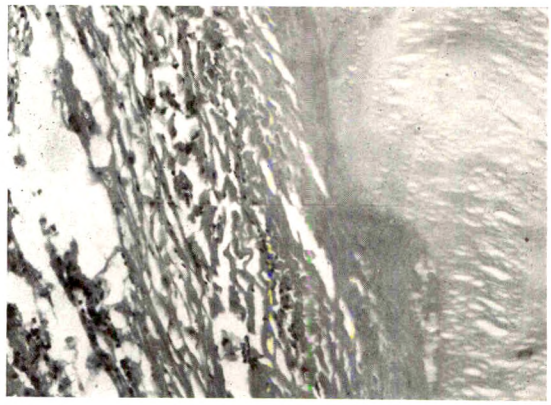


FIG. 6. Case II. Photomicrograph showing a portion of one cyst with the cyst wall (right) and surrounding reactive tissue (center). A few normal liver cells are seen in the lower left corner. Finely divided calcium deposits, poorly demonstrated in H and E stained section, occur in the reactive fibrous wall of the cyst (H and E stain; ×45).

calcified. These multiple ring-like calcified densities, which varied from 2 to 4 mm., are diagnostic of alveolar hydatid disease.

This patient had a fairly large mass which was asymptomatic and was diagnosed as alveolar hydatid disease preoperatively by plain film roentgenographic findings and the positive echinococcal hemagglutination and flocculation titers. Her social history was helpful in that she had had not only extensive exposure to dogs, but also direct contact with foxes. In addition to contracting the disease from fecal contamination from dogs, the accidental definitive host, one may ingest infective ova from fox pelts during skinning.[38]

CASE III. F.M., a 61 year old Eskimo female from northwestern Alaska, was admitted for the 5th time on September 29, 1970, for neurologic evaluation. Six weeks prior to this admission, she was admitted to an outlying hospital after an episode of clouded sensorium and bizarre behavior. Her behavior returned to normal in a few days and she was discharged. On September 26, 1970, she was readmitted to the outlying hospital after a tonic-clonic grand mal seizure. Her eyes were deviated to the left. A lumbar puncture revealed normal pressure, 260 lymphocytes/cu. mm., and protein of 47 mg. per cent. The remainder of her laboratory work was normal. She remained confused prior to her transfer to our hospital on September 29.

Past history revealed that she had taken prophylactic INH for tuberculosis for several months. She had a presumptive diagnosis of alveolar hydatid disease made at laparotomy in 1960. Previous echinococcal titers had been positive. Upper gastrointestinal examinations in 1965 and 1968 showed enlargement of the left lobe of the liver and calcifications in both lobes (Fig. 7). Liver scans showed hepatomegaly with gross enlargement of the left lobe, which contained a large mass with a central area of decreased uptake.

On admission, she was cachectic but oriented. Her temperature was 100°F. There was slight lateral nystagmus and ataxia. Her liver was enlarged, primarily in the epigastrium, and was not tender. The sedimentation rate was 58 mm. per min., and the albumin/globulin ratio was 3.2/4.5 mg. per cent (normal 4 to 5/2 to 3). Echinococcal hemagglutination and flocculation titers were 1:4,096 and 1:320, respectively. Several lumbar punctures were done, revealing normal pressures, 500 to 2,500 white blood cells per cu. mm. of which 80 per cent were lymphocytes, and protein which increased from 430 to 730 mg. per cent. A pneumoencephalogram showed atrophy of cortical tissue and enlargement of the lateral ventricles and basilar cistern. The 4th ventricle was not demonstrated. Electroencephalogram and RISA scans were abnormal but nondiagnostic. The remainder of her work-up was normal or unchanged. Studies included brain scan, carotid arteriogram, and liver chemistries. Initially, she improved on triple tuberculosis

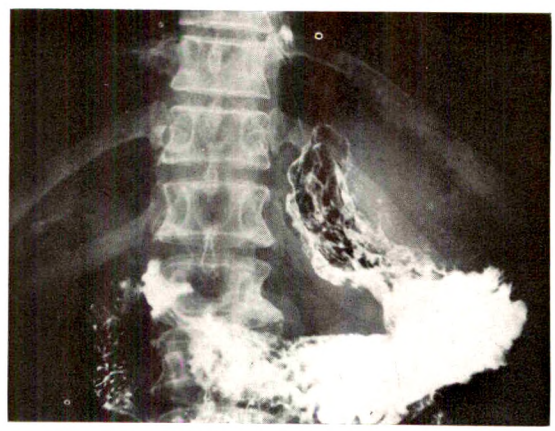


FIG. 7. Case III. The left lobe of the liver is enlarged compressing the fundus of the stomach. Small calcifications are demonstrated in the left and right lobes of the liver.

chemotherapy and steroids, but then her condition gradually deteriorated. Terminally, she developed 3rd, 4th, and 7th left cranial nerve palsies. She became obtunded and had marked fluctuation in her blood pressure and temperature. She expired on her 174th hospital day.

At autopsy, the liver was markedly enlarged. On section, the entire left lobe was replaced by a large cyst with a thick white fibrous wall, 2–3 cm. in thickness, lined by shaggy yellow material (Fig. 8). In the right lobe (Fig. 9) there were numerous white, slightly lobulated, gritty nodules, ranging from 1 to 5 cm. in diameter. A tumor-like mass was noted in the left cerebellar-pontine angle and a similar lesion was noted extending around the brain stem, deforming the midbrain and pons. The lesions resembled those in the liver. Histologically, these several lesions had the characteristic features of alveolar hydatid disease.

Comment. The calcifications in this patient's liver were small and scattered, although the patient had severe and extensive involvement of both lobes. A number of the calcifications had the characteristic appearance of small cysts, typical of alveolar hydatid disease. This patient's findings emphasize the point that there can be extensive involvement of the liver by *E. multilocularis* and no calcification, or only a small amount of calcification.

DISCUSSION

Nineteen of the 27 patients with the established diagnosis of alveolar hydatid disease had roentgenograms available for review (Table 1); 7 of these patients have expired. Eight of the 19 (42 per cent) had evidence of hepatomegaly on plain film roentgenograms of the abdomen and on barium examination of the gastrointestinal tract. Six of these 8 patients had verification of hepatomegaly by liver scan. The other 2 did not have liver scans. Four of the 19 patients underwent hepatic resection. Three of these had hepatomegaly clinically diagnosed prior to the surgery, and preoperative roentgenograms were not available for review. A total of 11 patients (58 per cent) had hepatomegaly.

Thirteen of the 19 (68 per cent) had hepatic calcification; 11 of the 19 (58 per cent) had calcification typical of alveolar hydatid disease which was thought to be diagnostic (Fig. 3, *A* and *B*; 4; 10, *A–D*; and 11, *A–C*). Three of the 13 with hepatic calcification

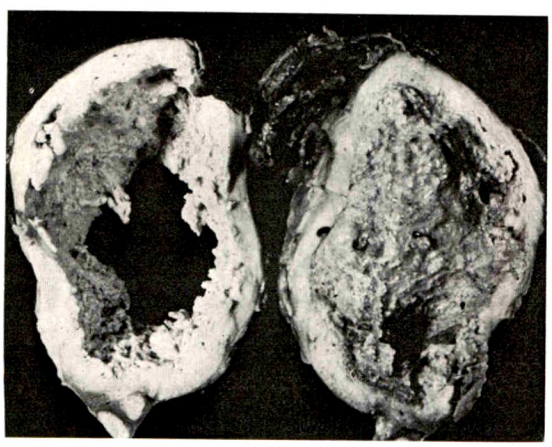


Fig. 8. Case III. Left lobe of the liver showing large *E. multilocularis* cyst with necrotic center. Fibrous cyst walls 2 to 3 cm. thick.

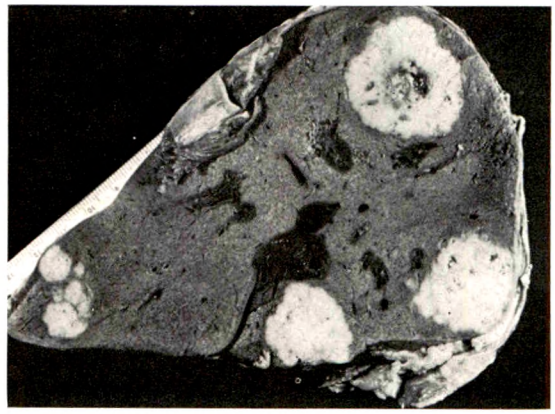


Fig. 9. Case III. Hemisection of the right lobe of the liver which contains a number of *E. multilocularis* lesions. They range in size from 1 to 5 cm. in diameter and one has a necrotic center.

Table I

ROENTGENOGRAPHIC FINDINGS IN 19 PATIENTS WITH THE ESTABLISHED DIAGNOSIS OF ALVEOLAR HYDATID DISEASE (*E. multilocularis*)

Finding	No.	Per Cent
Hepatomegaly	8	42
Hepatic calcification	13	68
Diagnostic calcification	11	58
Nondiagnostic calcification	2	11
Calcification in right and left lobes	3	16
Hepatomegaly and calcification	5	26

(16 per cent) had calcification in both right and left lobes. One of the 2 patients with nondiagnostic calcification had only small faint densities and the other had a large dense mass of calcification which was nonspecific. Five of the 19 (26 per cent) had both hepatomegaly and calcification.

Hepatomegaly and hepatic calcification are the plain film roentgenographic findings demonstrated in patients with alveolar hydatid disease. The hepatomegaly is due to the growth of the *E. multilocularis* organism, attending host reaction, and compensatory enlargement of the uninvolved portions of the liver.

The peculiar type of hepatic calcifications was the single most helpful roentgenologic finding in establishing the diagnosis of alveolar hydatid disease. The typical findings were small radiolucencies, outlined by calcific densities, ranging in size from 2 to 4 mm. in diameter. These lucencies were found scattered in the larger areas of amorphous calcium, which ranged in size from a few millimeters up to 10 to 12 cm. in diameter. These typical changes are thought to be diagnostic of alveolar hydatid disease, as similar lesions have not been previously reported in hepatic calcification due to other causes. The figures from Cases II and III and Figures 10, *A–D*, and 11, *A–C* illustrate these findings.

In pathologic specimens, calcification is seen diffusely in the amorphous debris within degenerating cysts, in the fibrous walls adjacent to the parasitic membranes of intact cysts, and in occasional foci between the laminae of the parasitic membranes. The majority of the calcification occurs scattered in the necrotic tissue, but the more important and diagnostic feature is the intact cyst with deposition of calcium surrounding it.

Grossly the large lesions appear as white to yellowish tumor masses which may replace liver parenchyma and which may present on the surface of the liver (Fig. 9). The centers of the large masses usually undergo necrosis, giving the mass the gross appearance of a large cavity filled with thick, pasty material, which has a firm wall up to several centimeters in thickness. A large single lesion may occupy half or more of the liver (Fig. 8). The liver may contain many lesions of different sizes (Fig. 9). Microscopically, eosinophilic laminated membranes appear as folded or collapsed masses within the amorphous debris of large cysts, while such membranes line the smaller peripheral cysts. At the periphery of the cysts, there is often a layer of reactive fibrous tissue which is separated, in turn, from the liver parenchyma by a layer of inflammatory cells and granulation tissue. There are also caseating granulomas within the liver parenchyma, with epithelioid cells, giant cells, and inflammatory cells surrounding the necrotic tissue. The small intact cysts are lined by an eosinophilic, laminated membranous layer, as in cystic hydatid disease. In alveolar hydatid disease in man, protoscolices are almost never found.

Tumoral calcification is dystrophic in type,[34] *i.e.*, it is the result of a change in the chemical structure of the intracellular substance. The mechanism by which this occurs is complex and not understood, but frequently the calcification occurs in necrotic areas. Why calcification occurs in some necrotic tumors and not in others is unknown.[34] It is more common in embryonic tumors in children than in carcinomas in adults, which may indicate that it occurs in more rapidly growing tumors which outstrip their blood supply, with consequent necrosis.[25,34] The hepatic calcification in alveolar hydatid disease is dystrophic in type, occurring in the necrotic tissue; however, the pattern of calcium deposition in the reactive fibrous tissue surrounding the membranes that form the wall of intact small parasitic cysts differentiates hepatic calcification of alveolar hydatid disease from other types of hepatic calcification.

Table II lists the reported causes of hepatic calcification. Most of these are quite rare and some of the disorders produce calcification thought to be typical for the disease.

Calcified hydatid cysts (*E. granulosus*),

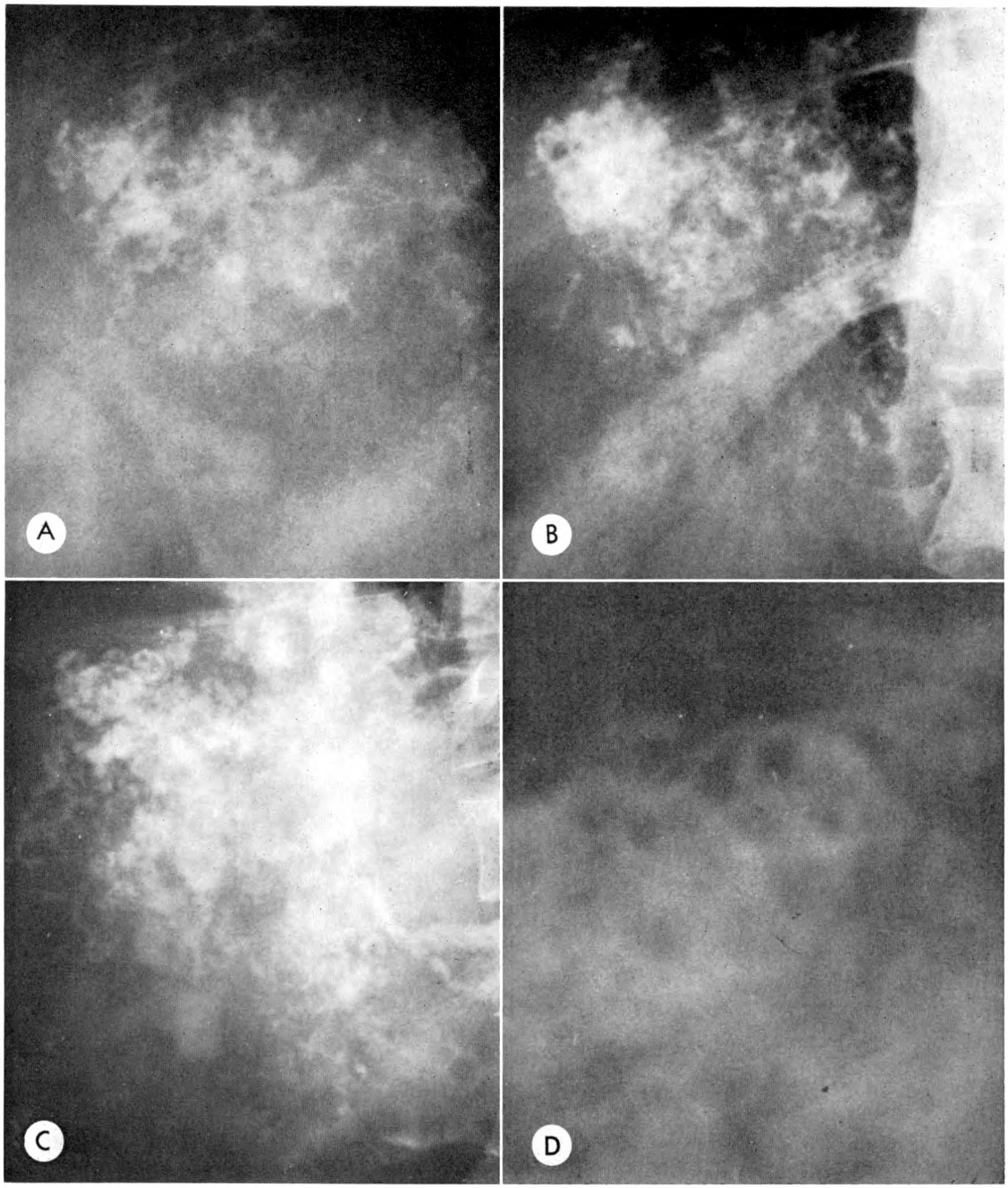


FIG. 10. Patient M.K. (*A*) Oblique roentgenogram of the right upper quadrant in a patient with surgically proven alveolar hydatid disease showing typical *E. multilocularis* hepatic calcifications. Roentgenogram was taken in 1959. (*B*) Same area in 1965; the calcifications have increased. (*C*) In 1971, further increase in the amount of calcifications is noted. (*D*) Enlargement of *C* showing small radiolucencies surrounded by calcification. This finding is thought to be diagnostic of alveolar hydatid disease.

both sylvatic and pastoral, are the most common cause of hepatic calcification. The calcification in an unruptured cyst is usually first seen as an arc, which gradually extends to form an oval or circle of calcium. Amorphous calcification occurs within the cyst. Polycyclic calcification in an unruptured cyst is pathognomonic of cystic hy-

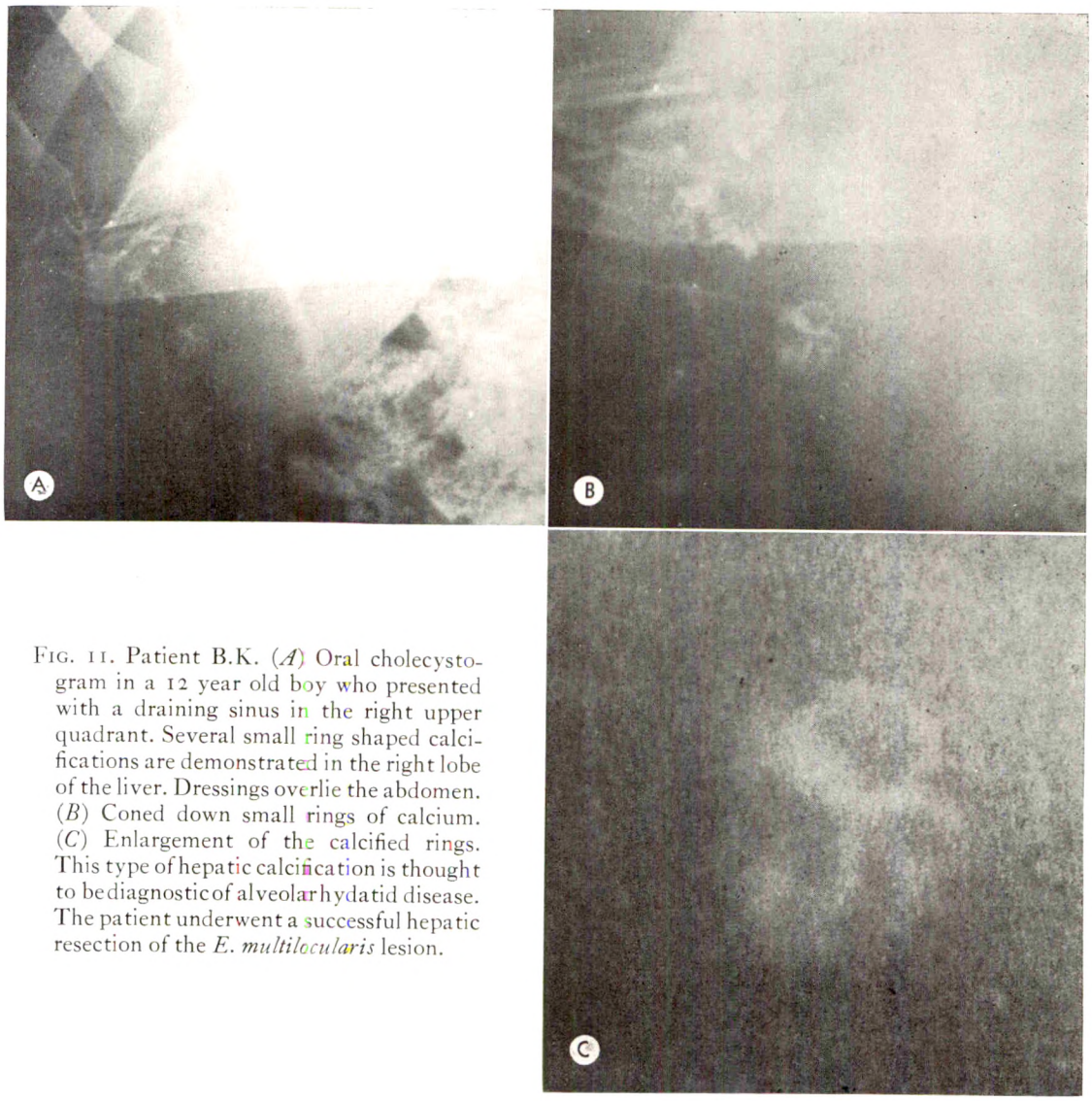


FIG. 11. Patient B.K. (*A*) Oral cholecystogram in a 12 year old boy who presented with a draining sinus in the right upper quadrant. Several small ring shaped calcifications are demonstrated in the right lobe of the liver. Dressings overlie the abdomen. (*B*) Coned down small rings of calcium. (*C*) Enlargement of the calcified rings. This type of hepatic calcification is thought to be diagnostic of alveolar hydatid disease. The patient underwent a successful hepatic resection of the *E. multilocularis* lesion.

datid disease. Not only is the mother cyst calcified, but also most of the daughter cysts are calcified as well. Ruptured cysts may have irregularly shaped calcification, but some of the calcium will usually be shaped in an arc.[8,10] The calcified lesions of cystic hydatid disease, both sylvatic and pastoral forms, are 5 to 10 times the size of the small calcified lesions diagnostic of alveolar hydatid disease.

Bassler and Peters[6] reported the incidence of hepatic calculi in association with cholelithiasis to be about 6 per cent. These calculi are rarely dense enough to be demonstrated on plain film roentgenograms and are usually discovered on T-tube or operative cholangiograms. When dense enough to be demonstrated on plain film roentgenograms, they are uniformly spherical and vary in diameter from 0.5 to 2.0 cm. with a rather dense shell around a less opaque area. They may be grouped in a mulberry pattern.

Calcification in the portal vein has been described by Haddow and Kemp-Harper[14] as linear densities of calcium crossing the vertebral column which are found in patients with portal hypertension. Localiza-

tion by anteroposterior and lateral roentgenograms helps to separate portal vein calcification from calcium in other organs; however, in some cases venography is necessary for accurate identification. Calcification in the splenic artery may help identify portal vein calcification, by association.[14]

Calcification of hepatic artery aneurysms is quite rare, but has been described as a multiloculated, ovoid, calcific density, sharply defined, with the periphery being egg-shell in character and the center less dense and mottled.[17] This is similar to splenic and renal artery aneurysmal calcification, but the relative positions differentiate them.

Karras *et al.*[19] mentioned the calcifications in liver abscesses as being dense, mottled, and irregular, but they can be arranged in a circular pattern. The patient is usually asymptomatic at the time the calcium is noted.

Tuberculosis and histoplasmosis calcifications in the liver are usually small, multiple, dense, discrete, and scattered throughout the organ.[5] The spleen is usually involved in histoplasmosis and less frequently in tuberculosis.[19]

Brucellosis has been reported to cause hepatic calcifications. Spink[41] described these as fluffy spots looking similar to snowflakes. Similar lesions may also occur in the spleen.

Alergant,[1] and Karras *et al.*[19] reported cases of gummas of the liver with calcification. The massive calcification of the liver reported by Heilbrun and Klein[15] may also have been due to a gumma and not alveolar hydatid disease as suspected. The patient reported had a positive serology and negative echinococcal hemagglutination titers. The calcification in a gumma is dense, irregular and well defined. Calcified gummas are extremely rare.

Primary hepatic tumors, both benign and malignant, may show calcific change. There have been a number of reports of calcified cavernous hemangiomas.[4,19,29] Plachta[29] described a characteristic pattern of spicules

Table II

REPORTED CAUSES OF HEPATIC CALCIFICATION

1. Cystic hydatid disease[7,8,10,22]
2. Hepatic duct calculi[6]
3. Portal vein calcification[14]
4. Calcified aneurysms of the hepatic artery[17]
5. Calcified liver abscess (pyogenic or amebic)[14,19]
6. Tuberculosis and histoplasmosis granulomas[5]
7. Brucellosis granulomas[41]
8. Calcified gumma[1,14,19]
9. Primary liver tumors
   - Benign—hemangioma[4,29]
   - Malignant—hepatoma (liver cell carcinoma),[14,19,25] cholangioma (bile duct carcinoma),[2,14,19] hemangioendothelioma[11,30]
10. Calcification of liver metastasis from ovary,[27] breast,[35] ocular melanoma,[19] neuroblastoma,[34] mesothelioma,[28] and colon[3,9,19,20,26,37,43]
11. Alveolar hydatid disease (15*),[44,45]
12. Congenital cysts of the liver†

15 * Calcification may have been due to calcified gummas.
† Anton, H. C. *Brit. J. Radiol.*, 1972, *45*, 146–147.

in the form of numerous radiating trabeculations, from a central area toward the periphery of the lesion. Primary hepatic carcinomas may calcify in a variety of patterns, ranging from spherical discrete calculi resembling cholelithiasis to irregular indistinct stippled calcification or spiculated calcification which has the appearance of a sunburst.[14] This last type is similar to that seen in hemangiomas as described by Plachta.[29] Calcification is more common in malignant hepatic tumors in children.[19,25] Biliary duct carcinoma may calcify, and has been described as amorphous collections of calcium with sharply defined borders which are spherically shaped, resembling calculi.[19] Rakov *et al.*[30] described a case of hemangioendotheliosarcoma occurring in a patient who previously had intravenous injections of thorotrast for a hepatic ailment. Tiny punctate stippled densities, which were typical deposits of thorium dioxide, were noted in his liver, spleen, and lymph nodes. Davis[11] reported a patient with the same type of tumor who had diffuse calcification throughout his lungs and liver. The calcifications were thought to be in metastatic lesions. This patient had not received injections of thorotrast.

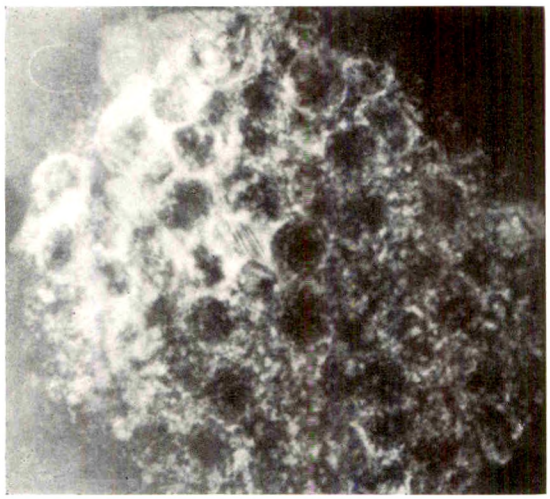


FIG. 12. Roentgenogram of a model of an *E. multilocularis* lesion containing calcium. Model made by mixing salmon caviar and barium in a viscous medium and freezing the mass in a block of ice.

Carcinoma of the colon is the most frequent primary tumor causing calcified metastatic lesions in the liver. The calcifications are diffuse, finely granular, closely spaced, and occupy an irregular area of the liver. They are discrete, average 2 to 4 mm. in diameter, have been likened to poppy seeds, and appear to be typical for colonic metastases.[19,26] Calcified hepatic metastases have been described in carcinoma of the breast[35] and ovary,[27] neuroblastoma,[34] ocular melanoma,[19] and mesothelioma.[28] The metastatic lesions from these primary tumors usually show much larger and denser calcifications than colonic metastatic lesions. There are usually other areas of intraabdominal calcification from metastatic carcinoma of the ovary and neuroblastoma, which may help in making the ultimate diagnosis.

Calcification in the pancreas, lymph nodes, aorta, and pleura, as well as cholelithiasis, must all be considered in the differential of upper abdominal calcification. Usually all of these can be diagnosed by their location and typical morphology.

If only a small amount of hepatic calcification is present, it is difficult to differentiate the calcification secondary to alveolar hydatid disease from that secondary to metastatic disease, tuberculosis or histoplasmosis, brucellosis, and primary hepatic tumors. However, a history of living in or visiting an endemic area, skin tests, hemagglutination and flocculation titers, and a work-up to rule out the other causes of hepatic calcification should establish the diagnosis.

The hepatic calcification of alveolar hydatid disease is significantly different from that caused by other disease processes, particularly when extensive. The occurrence of areas of radiolucency rimmed by calcium is the important differentiating feature. These apparently represent the calcification seen in the reactive tissues surrounding the membranous walls of the parasitic cysts. These lucencies may vary in size from 2 to 4 mm. and are found scattered throughout the affected areas of the liver.

Figure 12 is a roentgenogram made of a model produced by mixing barium sulfate and salmon caviar in a viscous medium and then freezing the mass in the center of a block of ice. It closely resembles the amorphous calcium, as well as the calcified multilocular cysts which occur in patients with alveolar hydatid disease. The model helps one understand the pathogenesis of the characteristic roentgenographic pattern of alveolar hydatid disease.

SUMMARY

1. The plain film roentgenographic findings of alveolar hydatid disease (*E. multilocularis*) in 19 patients are presented.

2. Hepatomegaly (in 42 per cent) and hepatic calcifications (in 68 per cent) are the 2 plain film roentgenographic findings.

3. The small radiolucencies surrounded by calcification, forming spheres which range in size from 2 to 4 mm. in diameter, are diagnostic (58 per cent) of alveolar hydatid disease.

4. The 3 different forms of hydatid disease: (1) alveolar hydatid disease (*E. multilocularis*), (2) sylvatic cystic hydatid disease (*E. granulosus*), and (3) pastoral cystic hydatid disease (*E. granulosus*), are discussed.

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REFERENCES

1. Alergant, C. D. Gumma of liver with calcification. *A.M.A. Arch. Int. Med.*, 1956, *98*, 340–343.
2. Allen, R. W., and Holt, A. H. Calcification in primary liver carcinoma. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *99*, 150–152.
3. Appleby, A., and Hacking, P. M. Calcification in hepatic metastases. *Brit. J. Radiol.*, 1958, *31*, 449–450.
4. Aspary, M. Calcified hemangiomas of liver. Am. J. Roentgenol. & Rad. Therapy, 1945, *53*, 446–453.
5. Astley, R., and Harrison, N. Miliary calcifications of liver. *Brit. J. Radiol.*, 1949, *22*, 723–724.
6. Bassler, A., and Peters, A. G. Hepatic calculi. *Am. J. M. Sc.*, 1947, *214*, 422–430.
7. Bockus, H. L. Gastroenterology. Volume III. W. B. Saunders Company, Philadelphia, 1966, pp. 27–30.
8. Bonakdarpour, A. Echinococcus disease. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *99*, 660–667.
9. Case record of Massachusetts General Hospital: case 41452. *New England J. Med.*, 1955, *253*, 828–831.
10. Clasessen, G. Roentgen diagnosis of Echinococcus tumors. *Acta radiol.*, 1928, Suppl. 6, 1–139.
11. Davis, G. M. Hemangioendothelioma of liver with unusual roentgenologic findings. *Gastroenterology*, 1961, *40*, 253–258.
12. Dean, D. V. Echinococcus multilocularis of liver. *St. Louis Med. & Surg. J.*, 1877, *14*, 420–424.
13. Fraser, R. G., and Paré, J. A. P. Diagnosis of Diseases of the Chest. W. B. Saunders Company, Philadelphia, 1971, pp. 712–715.
14. Haddow, R. A., and Kemp-Harper, R. A. Calcifications in liver and portal system. *Clin. Radiol.*, 1967, *18*, 225–236.
15. Heilbrun, N., and Klein, A. J. Massive calcification of liver. Am. J. Roentgenol. & Rad. Therapy, 1946, *55*, 189–192.
16. James, E., and Boyd, W. Echinococcus alveolaris with report of case. *Canad. M. A. J.*, 1937, *36*, 354–356.
17. Jarvis, L., and Hodes, P. J. Aneurysm of hepatic artery demonstrated roentgenographically. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1954, *72*, 1037–1040.
18. Kagan, I. J. Review of serological tests for diagnosis of hydatid disease. *Bull. World Health Organ.*, 1968, *39*, 25–37.
19. Karras, B. G., Cannon, A. H., and Zanon, B., Jr. Hepatic calcification. *Acta radiol.*, 1962, *57*, 458–468.
20. Khilnani, M. T. Calcified liver metastasis from carcinoma of colon. *Am. J. Digest. Dis.*, 1961, *6*, 229–232.
21. Lafond, D. J., Thatcher, D. S., and Handeyside, R. G. Alveolar hydatid disease. *J.A.M.A.*, 1963, *186*, 35–37.
22. Latham, W. J. Hydatid disease. Part 1. *J. Fac. Radiologists*, 1953, *5*, 65–81.
23. Leiby, P. D., Carney, W. P., and Woods, C. E. Studies on sylvatic enchinococcosis. III. Host occurrence and geographic distribution of *Echinococcus multilocularis* in north central United States. *J. Parasitol.*, 1970, *56*, 1141–1150.
24. Lukashenko, N. P. Comparative biologic and pathologic studies of *Echinococcus multilocularis*. *Arch. Environ. Health*, 1968, *17*, 676–680.
25. Margulis, A. R., Nice, C. M., Jr., and Rigler, L. G. Roentgen findings in primary hepatoma in infants and children. *Radiology*, 1956, *66*, 809–817.
26. Miele, A. J., and Edmonds, H. W. Calcified liver metastases: specific roentgen diagnostic sign. *Radiology*, 1963, *80*, 779–785.
27. Nathanson, L. Calcified metastatic deposits in peritoneal cavity, liver and right lung field from papillary cystadenocarcinoma of ovary. Am. J. Roentgenol. & Rad. Therapy, 1950, *64*, 467–469.
28. Persard, V., Batson, E. M., and Bankay, C. D. Pleural mesothelioma associated with massive hepatic calcification and unusual metastases. *Cancer*, 1970, *26*, 920–928.
29. Plachta, A. Calcified cavernous hemangioma of liver. *Radiology*, 1962, *79*, 783–788.
30. Rakov, H. L., Smalldow, T. R., and Derman, H. Hepatic hemangioendotheliosarcoma. *A.M.A. Arch. Int. Med.*, 1963, *112*, 173–178.
31. Rausch, R., and Jentoft, V. L. Studies on helminth fauna of Alaska. XXXI. Observations on propagation of larval Echinococcus multilocularis Leuckart, 1963, in vitro. *J. Parasitol.*, 1957, *43*, 1–8.
32. Rausch, R. L. On ecology and distribution of Echinococcus spp. (Cestoda: Taeniidae) and characteristics of their development in inter-

mediate host. *Ann. de parisitol. Paris*, 1967, *42*, 19–63.

33. Rausch, R. L. Taxonomic characters in genus Echinococcus (Cestoda: Taeniidae). *Bull. World Health Organ.*, 1968, *39*, 1–4.
34. Ross, P. Calcification in liver metastases from neuroblastoma. *Radiology*, 1965, *85*, 1074–1079.
35. Saghatoeslami, M., Khdarahmi, K., and Epstein, B. S. Calcified intrahepatic metastases from carcinoma of breast. *J.A.M.A.*, 1962, *181*, 1139–1140.
36. Samuels, S., and Fosmoe, R. Alveolar hydatid disease with involvement of inferior vena cava. *Am. Surgeon*, 1970, *36*, 698–701.
37. Sang Oh, K. Mottled areas of intra-abdominal calcification. *J.A.M.A.*, 1969, *208*, 521–523.
38. Schiller, E. L. Echinococcosis in North America. *Ann. Int. Med.*, 1960, *52*, 464–476.
39. Sereda, M. M., Sherman, L., and Smith, E. M. Alveolar echinococcosis. *Canad. M. A. J.*, 1961, *84*, 1138–1139.
40. Smith, E. M., and Hansen, H. Alveolar echinococcosis. *Am. J. Clin. Path.*, 1961, *35*, 160–165.
41. Spink, W. W. Suppuration and calcification of liver and spleen due to long standing infection with Brucella suis. *New England J. Med.*, 1957, *257*, 209–210.
42. Weinberg, T. Echinococcus alveolaris infection of human liver. *J. Mt. Sinai Hosp.*, 1947, *13*, 331–336.
43. Wells, J. Calcified liver metastases. *New England J. Med.*, 1956, *255*, 639–640.
44. West, J. T. Malignant Echinococcus disease of liver. *Alaska Med.*, 1959, *1*, 107–113.
45. West, J. T., Hillman, F. J., and Rausch, R. L. Alveolar hydatid disease of liver: rationale and technics of surgical treatment. *Ann. Surg.*, 1963, *157*, 548–559.
46. Wilson, J. F., Diddams, A. C., and Rausch, R. L. Cystic hydatid disease in Alaska. *Am. Rev. Resp. Dis.*, 1968, *98*, 1–16.

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RADIONUCLIDE HEPATIC FLOW STUDY AND LIVER SCAN IN TWO CASES OF NON-PARASITIC HEPATIC CYSTS*

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NONPARASITIC hepatic cysts occur with sufficient rarity to merit the reporting of each case. Less than 300 cases have been reported so far in the literature. Most of these were retention cysts of bile duct origin and were discovered only at autopsy.[3,4,7,10,17,18]

The opportunity to diagnose and treat surgically such lesions in 2 female patients has prompted these case reports. Moreover, these cases are described in detail to show the contribution of the Tc^{99m} pertechnetate hepatic flow study to the liver scanning and other radiologic procedures in arriving at a differential diagnosis.

REPORT OF CASES

CASE 1. In May 1967, a 72 year old female harboring a cystocele and subject to recurrent cystitis was seen again because of burning on urination, urgency and frequency of micturition of 10 days' duration. No hematuria, dysuria, fever or renal pain was present, but upon physical examination an epigastric mass was found which prompted her hospitalization for further investigation.

Past medical history revealed hospitalization in 1959 for epigastric pain. A cholecystogram at that time showed a gallstone in an enlarged gallbladder. A barium meal examination was normal and a barium enema study visualized the cecum under the hepatic border. Malposition of the cecum, gallstone and functional troubles of the digestive tract were retained as final diagnoses. In 1964, the patient was admitted to another hospital for cholelithiasis. A cholecystectomy, an appendectomy and a right oophorectomy for a cyst were performed. A uterine prolapse and an asymptomatic cystocele were also noted. The surgical exploration did not reveal any other feature.

The outstanding physical finding in May 1967 was an enlarged liver palpable 6 cm. below the costal margin and extending into the epigastrium and left upper quadrant. The patient was unaware of its presence and there were no symptoms referable to the gastrointestinal tract. All liver function tests gave results within normal limits.

A barium meal examination, barium enema study and intravenous pyelography ruled out gastrointestinal or urinary tract involvement (Fig. 1, *A–C*). However, compression and displacement of the stomach and duodenum to the left of the midline, and lowering of the hepatic flexure, the transverse colon and the right kidney, pointed to either a large hepatomegaly or a huge right upper quadrant mass.

Radioactive colloidal gold liver scanning revealed what appeared to be a liver of normal size and shape entirely displaced to the left by a huge cold lesion in the right upper quadrant (Fig. 2*A*). The irregularity of uptake over the left part was attributed to abnormal position of the liver, rather than to an intrinsic abnormality in the liver.

The patient was released from hospital 13 days after admission with a presumptive diagnosis of congenital malformation of the liver. Elements substantiating this opinion were: an excellent general condition and the absence of symptoms and laboratory findings indicative of any active hepatic involvement in a patient harboring a huge silent mass in the right upper quadrant displacing the functional liver to the left.

Two and a half years later, the patient was readmitted to the hospital complaining of abdominal distention and tenderness on palpation of 6 months' duration. No symptoms referable to the digestive tract were present. The vital signs were normal; the patient was in excellent condition and again the laboratory tests were within normal limits.

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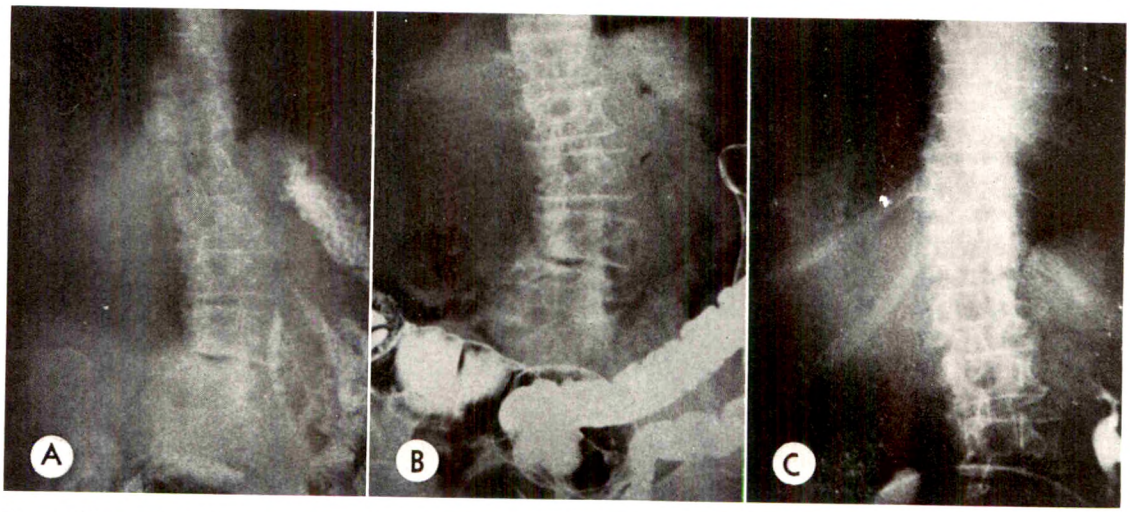


FIG. 1. Case 1. (*A*) Compression and displacement of the stomach and duodenum to the left on barium meal examination. (*B*) Lowering of the hepatic flexure on barium enema study. (*C*) Lowering of the right kidney on intravenous pyelography.

Abdominal distention was obvious. The right quadrant mass was felt to be "cystic on palpation" and filled the entire right abdomen displacing the liver to the left. A colloidal gold hepatic scan showed the presence of a cold mass in the right upper quadrant (Fig. 2*B*). The dominant feature of the scan was the greater displacement of the functional liver to the left, as compared to the hepatic scan performed $2\frac{1}{2}$ years previously, and thus substantiating the history of abdominal distention given by the patient.

A Tc^{99m} pertechnetate hepatic flow study was performed in the following manner to help define the degree of vascularity of the lesion and outline the lesion in relation to the liver and other structures in the abdomen and the chest:

A Nuclear Chicago Pho-Gamma scintillation camera equipped with a divergent collimator was used. A landmark scan was first obtained making use of the colloidal gold previously injected into the patient for the gold hepatic scan (Fig. 3*A*). Then, 10 millicuries of radiopertechnetate, contained in less than 2 ml. of normal saline, was rapidly injected into an antecubital vein, and sequential exposures of the circulation of the radionuclide were simultaneously recorded both on Polaroid Paper-Film (Fig. 3, *B–H*) at the rate of 2 second exposures for 16 consecutive pictures and on x-ray type film

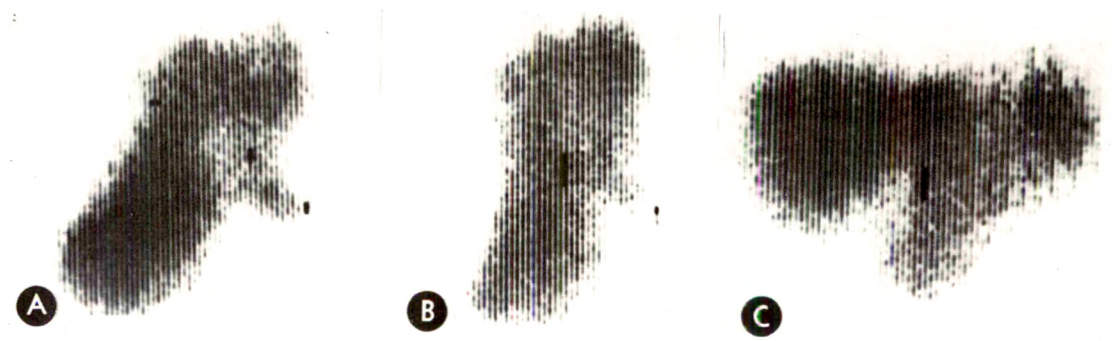


FIG. 2. Case 1. Anterior views of colloidal gold scans. (*A*) In 1967, the rotation of the liver and its global displacement to the left by the silent mass in the upper right quadrant stood out. (*B*) In 1969, the increase in size of the silent mass was assumed from the greater displacement of the liver to the left and the greater opening of its upper curvature as compared to the 1967 scan. (*C*) The early postoperative scan showed the liver back in its relatively normal position; this became the base line scan for further assessment of possible recurrence.

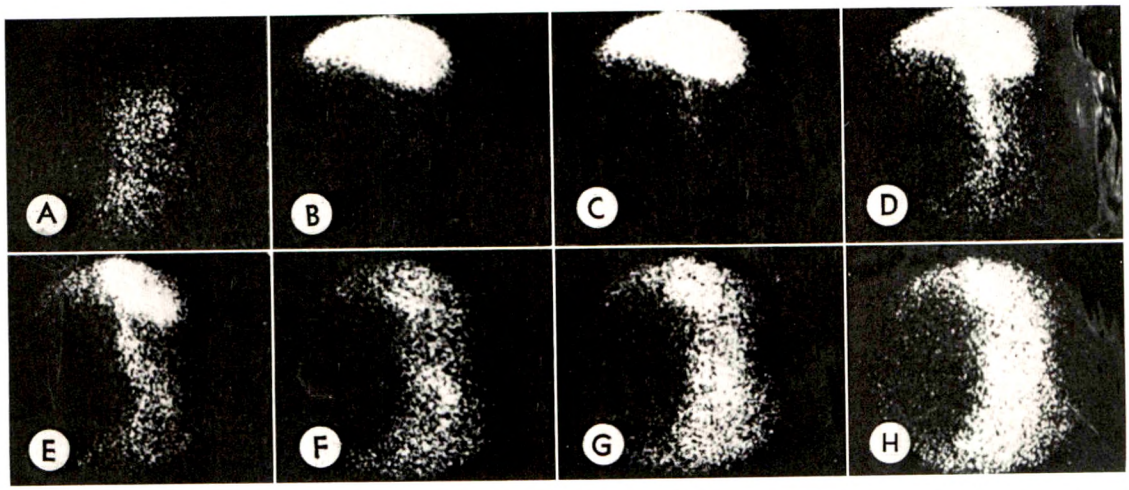


Fig. 3. Case I. Radiopertechnetate flow study. (*A*) A landmark colloidal gold liver scan, same as that shown in Figure 2*B*, was first obtained on the Phc-Gamma, in the actual position for the pertechnetate flow study. It was obtained immediately prior to the dynamic study, with the camera spectrometer set on the gold energy. Then, the spectrometer was set on the technetium energy, nullifying for our purpose the detection of gold in later views. The flow study was initiated in the following manner: (*B–H*) Sequential 2 second exposures of radiopertechnetate flow following rapid injection into antecubital vein were obtained. Note sequential outlining of chest (*B*) and abdominal structures (*C–H*), allowing their identification and actual correlation. Easy delineation of the mass and absence of perfusion over it were dominant features of the study. Actual relation to functional liver was obtained by comparison with the landmark scan (*A*).

using the photoscope, at the rate of 20 second exposures for 4 consecutive frames. Equivalent information was achieved with both the Polaroid paper and x-ray type film pictures. However, the x-ray type films, allowing for superimposition of the different exposures, were found very helpful in correlating, point by point, structures delineated on the landmark colloidal gold scan and the various serial radiopertechnetate studies. Accurate delineation of the cold lesion and absence of perfusion over it in the right abdomen, as compared to normal perfusion of the liver located in the epigastrium and left upper quadrant, were the dominant features of the study.

At surgery, an enormous cyst of the liver was found filling the right upper quadrant, displacing the liver to the left and the right kidney downwards (Fig. 4). The cyst was lined with a serous membrane; it was easy to delineate by palpation its superior and posterior aspects. It was affixed to the inferior part of the left portion of the liver. Almost 3 liters of fluid was aspirated. During its dissection, bleeding of the liver occurred and forced a marsupialization of that portion of the wall of the cyst in relation with the liver. An unsuspected second cyst of the same nature, 10 cm. in diameter, was then seen posteriorly and was dealt with in the same manner as the first.

Culture and staining of the cyst fluid were negative for bacteria. Chemical analysis measured 33 units of amylase.

Histologic examination of the wall of the cyst revealed connective tissue containing hepatic elements such as hepatic cells and biliary ducts. These elements were infiltrated by a moderate number of inflammatory cells. The final diagnosis was benign cysts of the liver with slight inflammatory reaction.

On December 2, 1969, 1 week after laparotomy, a radiogold hepatic scanning (Fig. 1*C*) and a radiopertechnetate flow study were performed. Both reflected the new relatively normal position of the liver.

A follow-up technetium sulfur colloid liver scanning was performed 10 months later and ruled out recurrence for the time being.[3]

Case II. This case entails a roentgenologic finding in a 60 year old female admitted into another hospital for a zona. Routine roentgenograms at that time revealed a mass located between the liver and right kidney. The patient was unaware of its presence and had no related complaints. She was then referred to this hos-



Fig. 4. Case I. At surgery, the liver (l) and the huge right upper quadrant cyst (m) were in the same position as shown on the preoperative scan (Fig. 2*B*; and 3, *A–H*).

pital for further investigation and appropriate treatment.

Medical antecedents revealed hysterectomy and appendectomy 23 years previously and an episode of rheumatoid arthritis 6 years ago. There had been no history of jaundice.

On examination, a round, mobile, orange size mass was palpable in the lower part of the right upper quadrant. A colloidal gold scan showed the mass to be a cold lesion embedded in the lower portion of the liver, raising the right lobe towards the diaphragm and pushing the left lobe towards the epigastrium (Fig. 5, *A* and *B*). A radiopertechnetate hepatic flow study was performed (Fig. 6, *A–G*). Upon completion of the flow study, the intensity knobs on both oscilloscopes were lowered to scan settings and an early scan was obtained (Fig. 6*H*). The avascularity of the mass stood out between the normal vascularity of the liver and right kidney on the flow study pictures and the early scan. At this point, an avascular mass, either solid or cystic, was thought to be present.

Its exact nature remained somewhat of a puzzle until laparotomy, although many different roentgenologic techniques were performed to untangle it (Fig. 7, *A–F*). Cholecystography revealed a normal gallbladder with slightly stretched cystic and common bile ducts (Fig. 7*A*). The inferior vena cavagram was normal (Fig. 7*B*). Selective right renal arteriography (Fig. 7, *C* and *D*) showed a right kidney tilted from above and away from the midline and displaced downward; the upper interlobar arteries appeared stretched. Surprisingly, the right adrenal gland did not follow the kidney in its descent. The contours of the kidney were normal. At aortography (Fig. 7, *E* and *F*), the

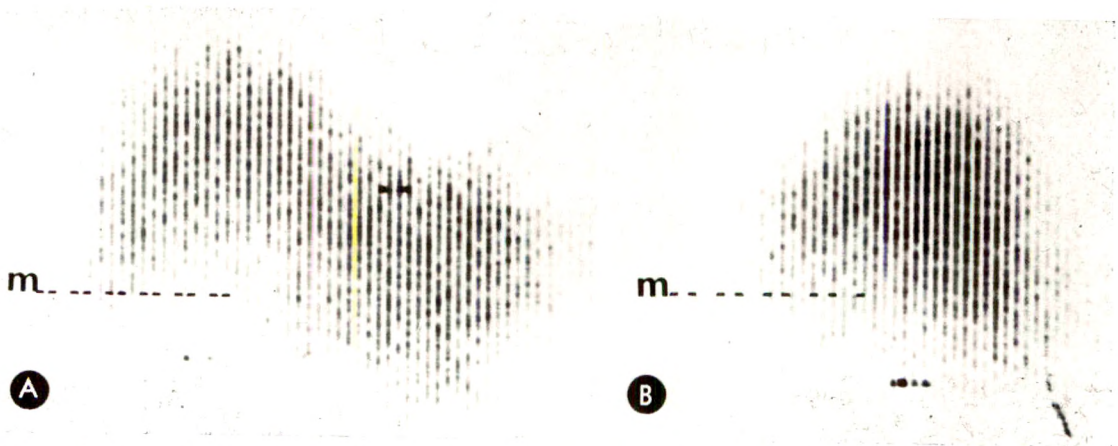


Fig. 5. Case II. (*A*) Anterior and (*B*) right lateral views of the colloidal gold liver scanning show a region of decreased activity over the projection of the mass (m). The shape of the liver impinged upon by the occupying lesion shows a right lobe raised upward and a left lobe displaced toward the epigastrium.

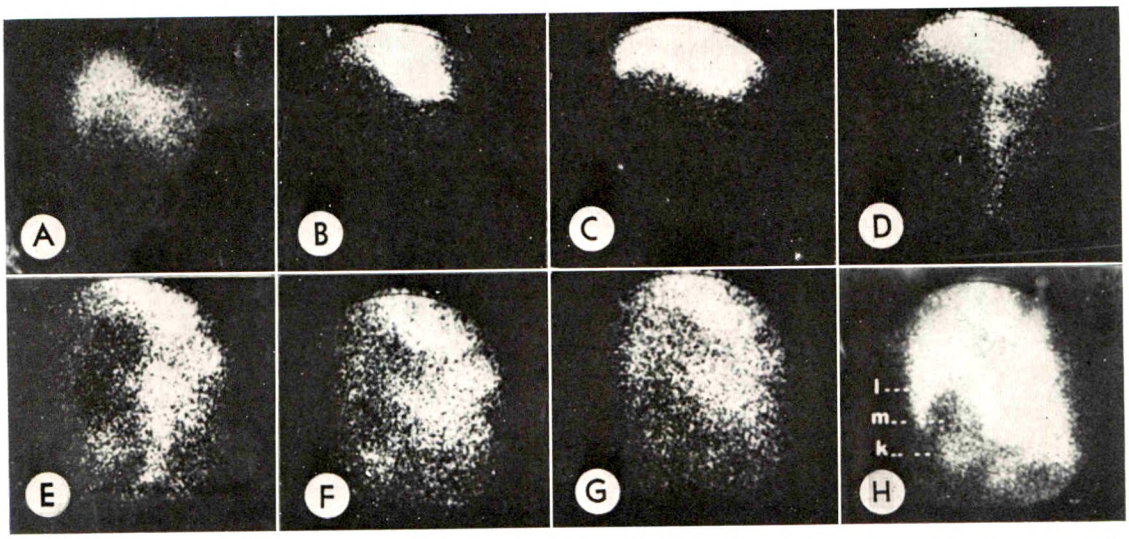


Fig. 6. Case II. Radiopertechnetate flow study. (*A*) Landmark colloidal gold liver scan. (*B–G*) Radiopertechnetate flow study. (*H*) Radiopertechnetate scan obtained in the minutes following the flow study. Note the nonfilling pattern of the mass (m) standing out between the filling pattern of the right kidney (k) and the liver (l) in all radiopertechnetate views (*E–H*).

hepatic artery was seen originating from the superior mesenteric artery. The hepatic vessel was stretched and its secondary branches delineated an important avascular mass.

Exploration revealed a smooth-walled cystic mass presenting from the inferior aspect of the right lobe of the liver and displacing the hepatic artery and the common bile duct (Fig. 8). The cyst was affixed to the base of the right lobe of the liver. The right kidney and right adrenal were completely independent of that mass. The cyst was removed *in toto*, without being ruptured. It measured 12 by 8 cm. and was filled with clear brownish fluid (Fig. 9*A*).

The surgical specimen consisted of 2 incompletely separated cysts (Fig. 9*B*), lined by a simple epithelium containing some goblet and some argentaffine cells. The epithelium lining was replaced in some regions by small deposits of fibrin. The epithelial lining was lying on connective tissue in which some smooth muscle fibers could be found (enteroid cyst).

DISCUSSION

Symptoms and signs. A review of the literature indicates that there are neither specific symptoms nor pathognomonic physical signs of nonparasitic hepatic cysts.[4,7,17,18] Most hepatic cysts are asymptomatic; their discovery is usually an autopsy finding. When there are roentgenographic findings, most often the diagnosis is suggested by displacement of adjacent organs noted during contrast material examination of the gastrointestinal tract. Occasionally, visualization of a radiopaque mass, as in our Case II, is the trigger feature. The symptoms which occur are almost always, in the absence of complications, the result of the space occupying effect of the cyst. Abdominal distention, the presence of a mass and right upper quadrant or epigastric pain are the most frequent complaints.

Complications. Complications are: rapid growth of the cyst; hemorrhage into it; torsion of its pedicle; secondary bacterial invasion with inflammation and development of infection; spontaneous or traumatic rupture into the peritoneal cavity; and jaundice which is the result of common bile duct obstruction by pressure.[4,7] In our Case II, pressure on the common bile duct was noted at laparotomy and the histologic examination revealed previous hemorrhage into part of the multilocular cyst. However, these 2 complications remained subclinical during the life of the patient. Major complications are infrequent. Jaundice is only rarely described; it occurs in approximately 9 per

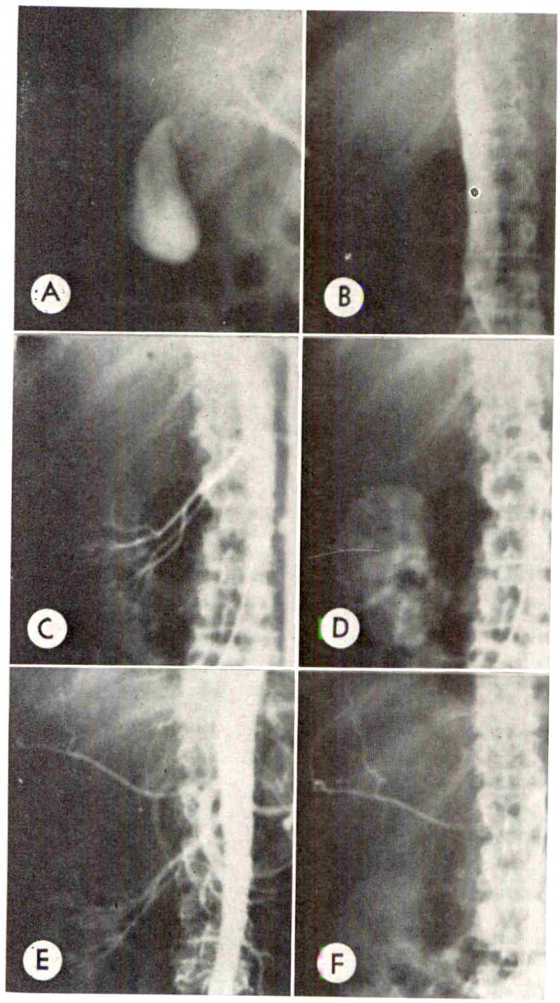


Fig. 7. Case II. (*A*) Cholecystography reveals a normal gallbladder with slightly stretched cystic and bile ducts. (*B*) Normal inferior vena cavagram. (*C* and *D*) Selective right renal arteriograms show a vertical kidney from above and away from the midline and displaced downward. (*E* and *F*) On aortography, the hepatic vessel is stretched and its secondary branches delineate an important avascular mass.

cent of cases of nonparasitic liver cyst requiring surgical treatment.[7]

Growth. Most hepatic cysts are slow growing. Henson *et al.*[10] reported that the interval between the onset of symptoms and admission to a hospital is 3 years. Flagg and Robinson[7] described the case of a 90 year old woman, who presented with a huge unilocular cyst of the left lobe of the liver. The patient had an attack of acute cholecystitis 2 years previously that necessitated a cholecystotomy. Our Case 1 underwent laparotomy for cholecystectomy in 1964. The first indication of the abnormal mass was a physical examination finding 3 years later and the first complaint by the patient appeared 2 years later.

Differential diagnosis. It is extremely difficult to make an accurate preoperative diagnosis in nonparasitic solitary cysts. Differentiation from parasitic cysts, choledochal cysts, polycystic disease, hydrops, mucocele or carcinoma of the gallbladder, cholecystitis, hepatoma, pancreatic pseudocyst, mesenteric, ovarian, renal or adrenal cyst and carcinoma of the liver or stomach must be considered.[4,7,10] Roentgenologic procedures are of value in the detection and in the assessment of these patients. However, a definitive diagnosis is not always arrived at, even with the sophisticated techniques such as celiac angiography.[9]

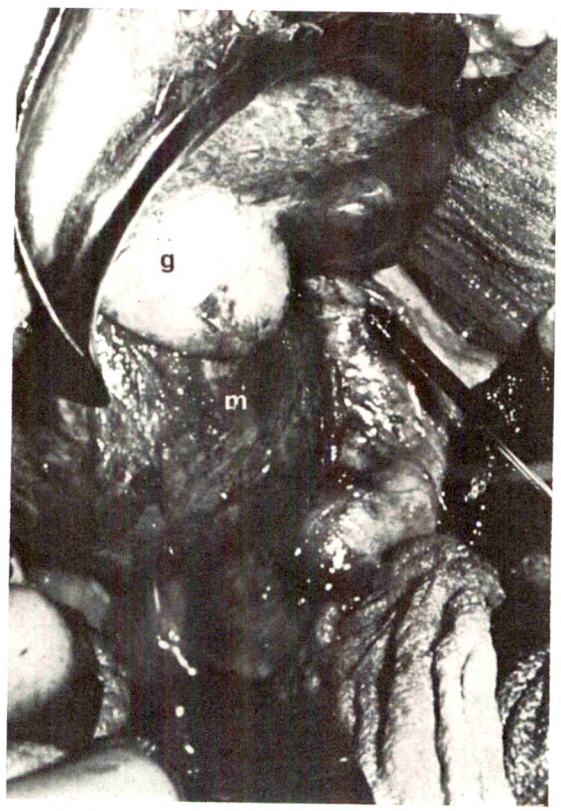


Fig. 8. Case II. At surgery, the mass (m) was underneath the gallbladder (g).

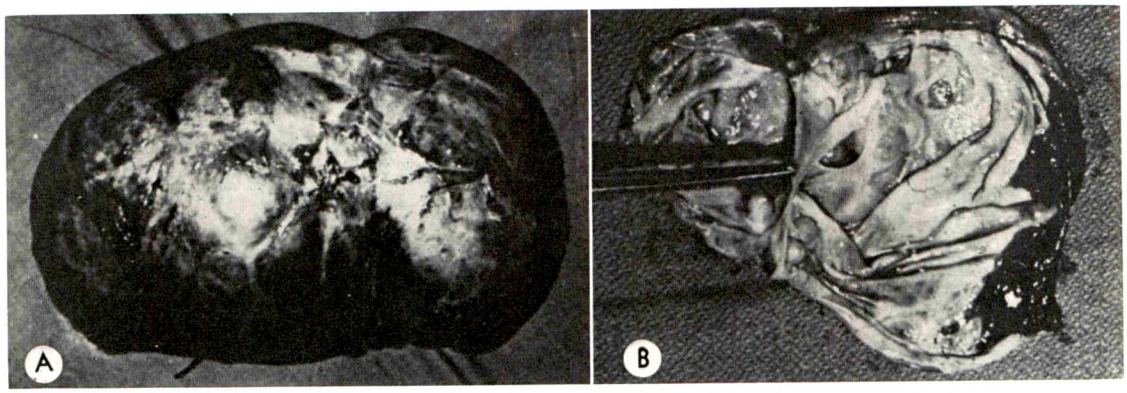


Fig. 9. Case 11. Cyst, as removed (*A*) *in toto*, and (*B*) upon opening.

Our second patient is a case in point.

Liver scan. Radiocolloid or rose bengal liver scans are particularly valuable for morphologic assessment of the liver and follow-up of lesions. Cystic lesions in the liver show as regions of diminished or absent uptake by this method.[1–4] Noteworthy, however, is the fact that liver morphology is easily modified when impinged upon by disease processes in neighbouring structures.[8] The resulting liver scan can be quite confusing and, at times, may even lead to false positive diagnosis of hepatic involvement. When a conflicting state of affairs occurs and additional data are required, such as a better outlining of the lesion and liver or an assessement of their relative degree of vascularity, various combinations of radionuclides are used.[9,13,16] We use the Tc^{99m} pertechnetate hepatic flow study as an alternative.

In our 2 cases, the flow study contributed to better delineation of the lesions respective to hepatic tissue and surrounding structures and pointed out their avascularity by their nonfilling patterns. The flow study, however, could not unravel whether these avascular masses were solid or cystic. Delayed pertechnetate scans were not obtained. It is contemplated that in such cases, distribution patterns of the radionuclide related to time should be attempted.

In a case of liver metastasis from a carcinoma of the rectosigmoid (Fig. 10, *A–F*), the landmark colloidal gold hepatic scan showed 3 lesions as cold spots; the pertechnetate hepatic flow study demonstrated their avascularity by the early nonfilling pattern, but 4 hours later, a delayed scan of the pertechnetate showed filling of the holes. By that time, an even density had been achieved over the liver and lesions. Later delayed scans, to follow washout, were not obtained.

Actual data on the kinetics of radiopertechnetate in lesions of the liver are very scanty, but, if assumptions based on radiopertechnetate kinetic studies in brain and chest lesions apply,[5,6] delayed radiopertechnetate scans should be of value.

In the meantime, when a differential diagnosis of solid *versus* cystic mass is contemplated, ultrasonic studies should be performed.[11,12,14,15]

SUMMARY

Two cases of nonparasitic hepatic cysts are described in detail.*

A radiopertechnetate hepatic flow study was combined with the conventional radiogold hepatic scan in an attempt to achieve positive diagnosis. Better delineation of the

* Recently 2 additional patients were studied: a patient with a huge dermoid cyst and a patient with an hepatobiliary cyst producing abnormal liver scans. The radiopertechnetate flow study revealed absence of circulation. The 4 hour and the 24 hour delayed radiopertechnetate scans showed absence of diffusion of the radiopertechnetate into the cysts. Ultrasonic studies were also obtained and were indicative of the cystic nature of the lesions in both cases.

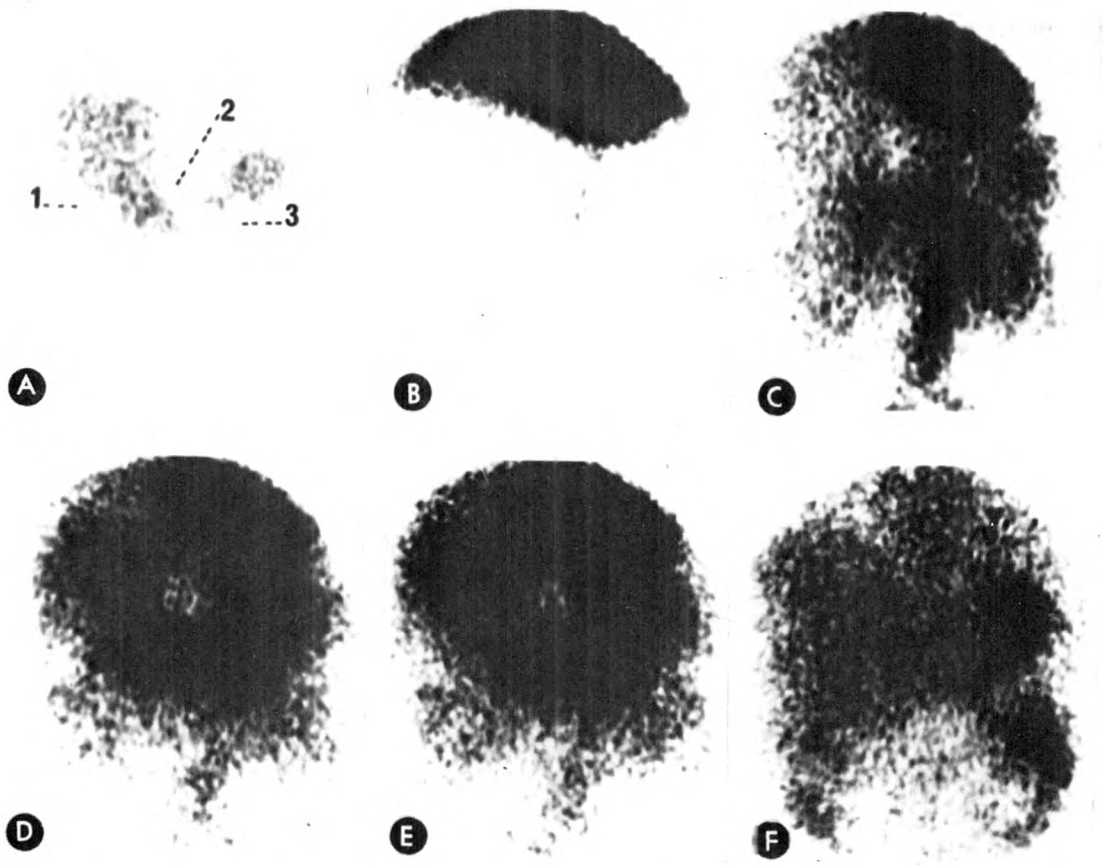


FIG. 10. Radiopertechnetate flow study and delayed scan in a patient harboring an adenocarcinoma of the rectosigmoid. (*A*) The landmark colloidal gold scan shows 3 lesions (1, 2, 3). (*B–E*) The radiopertechnetate flow studies show the nonfilling pattern of the 2 unmasked lesions (1, 2). (*F*) The radiopertechnetate 4 hour delayed scan shows late filling of the lesions.

lesions and evidence of their avascularity were noted.

Definitive proof of their cystic nature, however, remained somewhat controversial until surgery, although it was intimated by the history and various tests.

It is suggested that delayed pertechnetate scanning might help, and ultrasonic studies should not be neglected in any attempt to achieve pathognomonic features.

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REFERENCES

1. ARNOLD, J. S. Liver cyst and tumor differentiation by ^{99m}Tc bolus injection. *J. Nuclear Med.*, 1969, *10*, 315.
2. ARNOLD, J. S. Hepatic and renal cyst tumor differentiation by Anger camera observed with Tc^{99m} bolus perfusion. *Am. J. Clin. Path.*, 1969, *52*, 750.
3. BELCHER, H. V., and HULL, H. C. Nonparasitic cysts of liver: report of three cases. *Surgery*, 1969, *65*, 427–431.
4. CLARK, D. D., MARKS, C., BERNHARD, V. M., and BUNKFELDT, F. Solitary hepatic cysts. *Surgery*, 1967, *61*, 687–693.
5. CONWAY, J. J., and SHERMAN, J. O. Evaluation of chest masses in children with early and delayed radionuclide angiography. AM. J.

Roentgenol., Rad. Therapy & Nuclear Med., 1970, *108*, 575–581.

6. Fish, M. B., Pollycove, M., O'Reilly, S., Khentigan, A., and Kock, R. L. Vascular characterization of brain lesions by rapid cranial scintiphotography. *J. Nuclear Med.*, 1968, *9*, 249–259.
7. Flagg, R. S., and Robinson, D. W. Solitary nonparasitic hepatic cysts. *A.M.A. Arch. Surg.*, 1967, *95*, 964–973.
8. Freeman, L. M., Meng, C. H., Johnson, P. M., Bernstein, R. G., and Bosniak, M. A. False positive liver scans caused by disease processes in adjacent organs and structures. *Brit. J. Radiol.*, 1969, *42*, 651–656.
9. Freeman, L. M., Bernstein, R. G., Katz, M. C., Derman, A., and Meng, C. H. Combined diagnostic approach of hepatic scanning and celiac angiography in investigation of liver disease. *J. Nuclear Med.*, 1969, *10*, 628–632.
10. Henson, S. W., Jr., Gray, H. K., and Docherty, M. B. Benign tumors of liver: solitary cysts. *Surg., Gynec. & Obst.*, 1956, *103*, 607–612.
11. Holmes, J. H. Ultrasonic diagnosis of liver disease. In: Diagnostic Ultrasound. Proceedings of the First International Conference, University of Pittsburgh, 1965. Plenum Press, New York, 1966, pp. 249–263.
12. Kovaleski, B., Coppler, M., and Franco, J. Combined isotopic and ultrasonic studies in selected patients with unusual liver disease. *J. Nuclear Med.*, 1970, *11*, 411.
13. Lubin, E., Laor, J., Shimeoni, A., Lewitus, Z., Pick, A. I., Kadish, U., Dintsman, M., and Garti, I. Two stage scintiscanning in differential diagnosis of vascularized and nonvascularized intrahepatic space occupying lesions. *J. Nuklear Medizin*, 1970, *9*, 17–24.
14. McCarthy, C. F., Rhys Davis, E., Wells, P. N., Ross, F. G., Follet, D. H., Muir, K. M., and Read, A. E. Comparison of ultrasonic and isotope scanning in diagnosis of liver disease. *Brit. J. Radiol.*, 1970, *43*, 100–109.
15. Rand, E. Differentiation of renal masses with combined use of radioisotope scintiphotography and ultrasonography. *J. Nuclear Med.*, 1969, *10*, 367.
16. Riccioni, N., Becchini, M. G., Navalesi, R., and Lofaro, A. Use of radiofibrinogen in evaluation of cold areas of hepatic scans. *J. Nuclear Biol. & Med.*, 1968, *12*, 101–107.
17. Rosenberg, G. V. Solitary non-parasitic cysts of liver. *Am. J. Surg.*, 1956, *91*, 441–444.
18. Warren, K. W., and Polk, R. C. Benign cysts of liver and biliary tract. *S. Clin. North America*, 1958, *38*, 707–728.

A RADIOLOGIC APPROACH TO THE DIFFERENTIAL DIAGNOSIS OF SURGICAL AND NON-SURGICAL JAUNDICE*

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THE differentiation between patients with jaundice caused by a surgically correctable lesion and those without such a lesion remains a very challenging clinical problem. Numerous laboratory determinations of liver function are available to aid in this differential, and while these tests accurately separate surgical and nonsurgical jaundice in the majority of these patients, a substantial number remain in whom the test results are inconclusive or misleading. Oral cholecystography and intravenous cholangiography in these patients usually result in nonvisualization and are rarely helpful.

The purpose of this report is to present our experience with a combined approach to this differential diagnosis, utilizing I^{131} rose bengal blood clearance and sequential liver scanning, followed by percutaneous transhepatic cholangiography when indicated.

Rose bengal (tetraiodo tetrachlor fluorescein) has been available as a test of excretory liver function since 1924.[2] The dye is cleared quantitatively from the blood by the functional polygonal cells of the liver and is then excreted into the biliary tract and passes through the common duct and into the small bowel. The activity of the polygonal cells, the rate of blood flow through the liver and patency of the biliary tree are factors influencing the rate of blood clearance and biliary excretion of the rose bengal. The usefulness of the original rose bengal test, however, was limited, since it was dependent upon a colorimetric analysis of plasma, and elevated levels of serum bilirubin interfered with the results. Bromsulphalein (sulfobromophthalein) was later found to be cleared by the hepatic polygonal cells and replaced rose bengal as a liver function test.

After rose bengal was labeled with I^{131} by Taplin *et al.* in 1955,[10] the dye became more useful as a liver function test with the substitution of external counting for the colorimetric analysis of serum. Nordyke and Blahd in 1958[7] described the use of rose bengal with a dual probe technique to evaluate first the clearance of I^{131} rose bengal from the blood and then the patency of the common duct. Probes placed over the skull and over the left lower quadrant of the abdomen showed a steady decrease in activity as the liver cleared the rose bengal from the blood. A cholecystogogue was given, and increase in activity as measured by the abdominal probe signaled entry of the isotope into the small bowel indicating patency of the common duct. No increase in intestinal activity was seen in those patients with extrahepatic obstruction.

Sequential scanning of the liver and abdomen after I^{131} rose bengal injection has been described by Eyler *et al.*,[4] and by Freeman and Kay[5] to evaluate common duct patency. A combination of the rose bengal clearance and the sequential scans could eliminate possible errors due to probe placement and provide a pictorial anatomic presentation of the liver and of the progress of the isotope through the biliary tree and through the intestine. This combination gives functional and anatomic information about the liver in addition to testing patency of the common duct.

Further anatomic information can be ob-

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tained, particularly in those cases where the clearance and scan results indicate extrahepatic obstruction, by the use of percutaneous transhepatic cholangiography. The biliary tree can be visualized by this technique in over 80 per cent of patients with dilated obstructed ducts and approximately 50 per cent of patients without dilatation of the biliary tree.[11] The percutaneous cholangiogram should pinpoint the exact anatomic location and etiology of the obstruction. This information is helpful preoperatively, allowing the surgeon to plan the type of by-pass or definitive operation to be done without using time under general anesthesia.

TECHNIQUE

Three hundred microcuries of I^{131} rose bengal was injected intravenously and 1 minute count rates obtained every other minute for the first 20 minutes after injection with a probe positioned over the right ear. The first liver scan is obtained after the count rates are completed, and the sequential scans of the liver and abdomen are made at 3 and 24 hours after injection. The technique of percutaneous cholangiography has been discussed in the literature.[1,3,12]

INTERPRETATION

A smoothed curve is constructed from the clearance data (Fig. 1). Nordyke and Blahd[8,9] have recommended using a ratio expressed as a percentage obtained by dividing the 20 minute count rate by the 5 minute count rate. This represents the blood retention or residual isotope activity within the blood not as yet cleared from the circulation by the liver. Normally the blood retention is 45 to 50 per cent (Fig. 2*A*). In a patient with a nonobstructed duct, the sequential scans show isotope activity in the intestine within 2 hours after intravenous injection (Fig. 3, *A* and *B*).

In cases of primary hepatocellular dysfunction such as cirrhosis or hepatitis, blood retention of 75 per cent or greater would be expected (Fig. 2*B*). A high blood

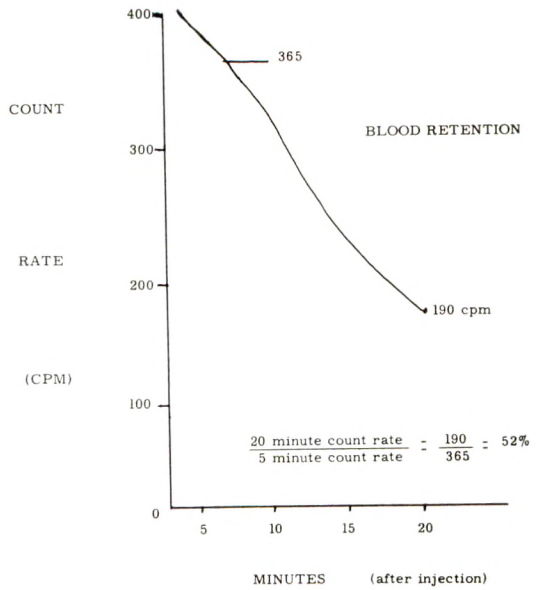


FIG. 1. Representative smoothed curve constructed from clearance data in normal patient and illustrative blood retention ratio.

retention indicates inability of the liver to clear the I^{131} rose bengal from the blood. The more severe the hepatocellular dysfunction, the higher the blood retention. In cases of mild to moderate hepatocellular dysfunction without significant intrahepatic cholestasis, isotope activity will appear within the intestines on the sequential scans, although there may be some delay depending upon the degree of hepatocellular dysfunction.

Patients with extrahepatic obstruction characteristically show no evidence of intestinal activity on the 24 hour scan. All isotope activity remains within the liver (Fig. 4, *A* and *B*). Blood retentions are usually between 60–75 per cent, intermediate between a normal of 50 per cent, and the 85–90 per cent range indicating severe hepatocellular dysfunction (Fig. 2*C*). The degree of blood retention in these cases depends on the degree of hepatocellular dysfunction secondary to the obstruction which is dependent on the length of time of the obstruction. Longstanding extrahepatic obstruction may cause significant liver cell dysfunction.

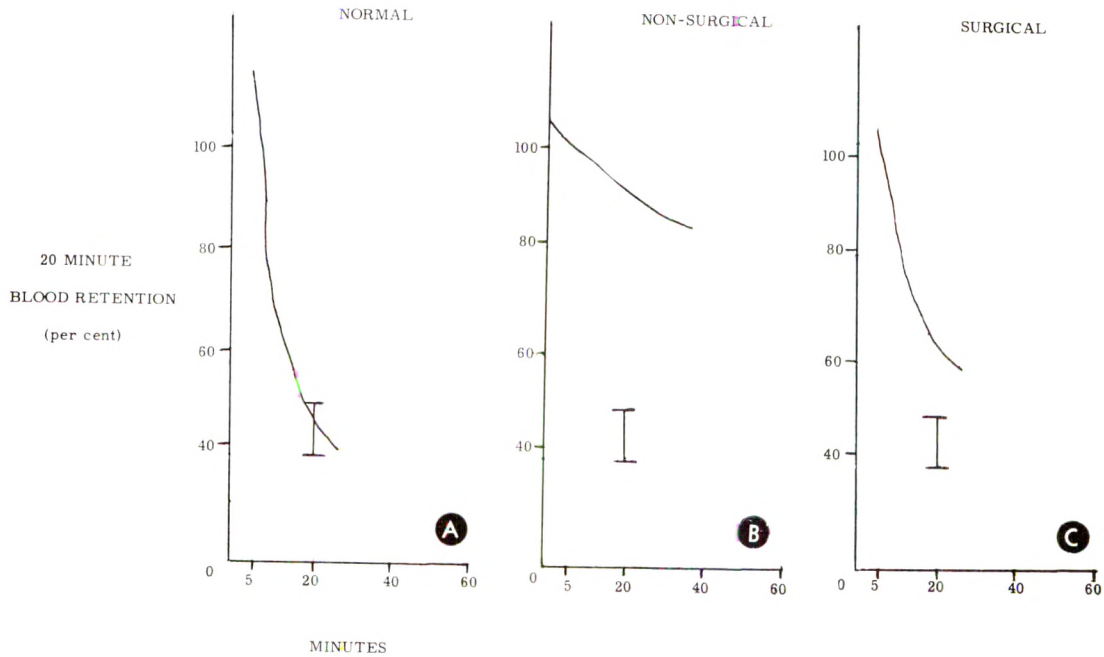


Fig. 2. Typical blood clearance curves in (*A*) normal, (*B*) nonsurgical, and (*C*) surgical patients. (Reproduced with permission from *J.A.M.A.*[9])

Patients with significant intrahepatic cholestasis caused by primary hepatocellular disease will also show a sequential scan picture of retained isotope within the liver at 24 hours, with no evidence of isotope within the intestines (Fig. 5, *A–C*). In our experience, patients with intrahepatic cholestasis who have presented this scan pic-

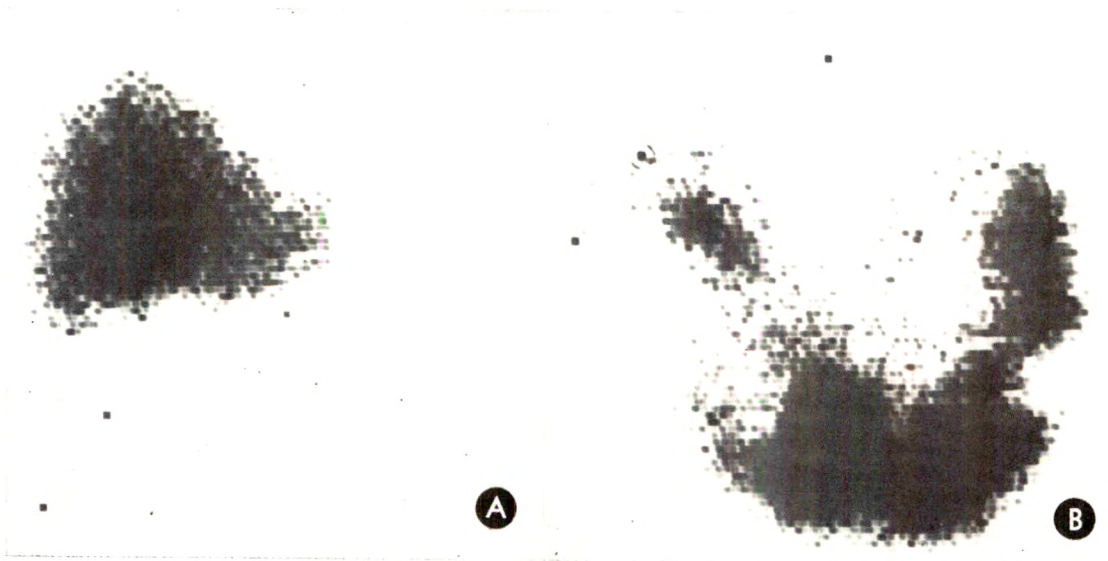


Fig. 3. Normal scan: (*A*) 30 minutes after isotope injection—normal appearing liver; (*B*) 3 hours after injection—all isotope in intestines. Blood retention 48 per cent.

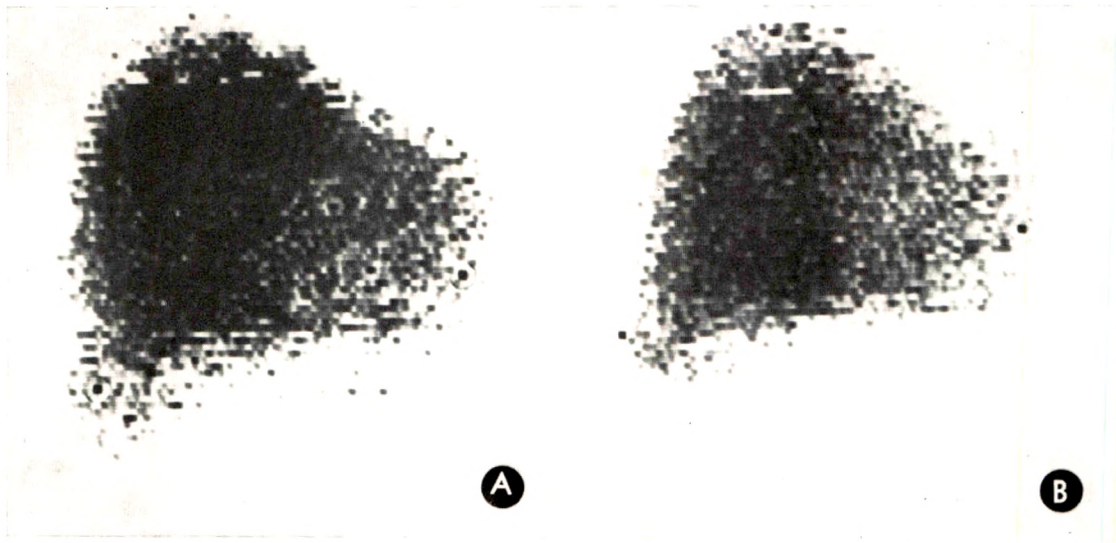


FIG. 4. Obstruction of common duct by carcinoma of common duct: (*A*) 30 minutes after isotope injection; (*B*) 24 hours after injection. All isotope activity remains in the liver. No isotope activity in the intestines. Blood retention 65 per cent.

ture have had blood retentions of greater than 90 per cent, and this has been important in making the correct diagnosis.

RESULTS

Forty patients showed biliary patency. Thirty-eight of these patients proved to have nonsurgical jaundice. These cases were proven by clinical follow-up, liver biopsy or autopsy. The 2 misdiagnoses in this group were both cases of ball-valve type intermittent obstruction of the common duct, 1 with a floating common duct stone and 1 with an adenoma of the ampulla of Vater. Nineteen patients showed retention of all of the isotope within the liver and no evidence of intestinal activity on the 24 hour scan. Fifteen of these patients were diagnosed as having extrahepatic obstruction, and these cases were proven by surgery or autopsy. The other 4 patients had intrahepatic cholestasis. One of these patients had been diagnosed as an extrahepatic obstruction on the basis of the



FIG. 5. Alcoholic hepatitis with intrahepatic cholestasis: (*A*) 30 minutes after isotope injection; (*B*) 3 hours after injection; (*C*) 24 hours after injection. All isotope retention in liver and no evidence of intestinal activity. Blood retention 94 per cent.

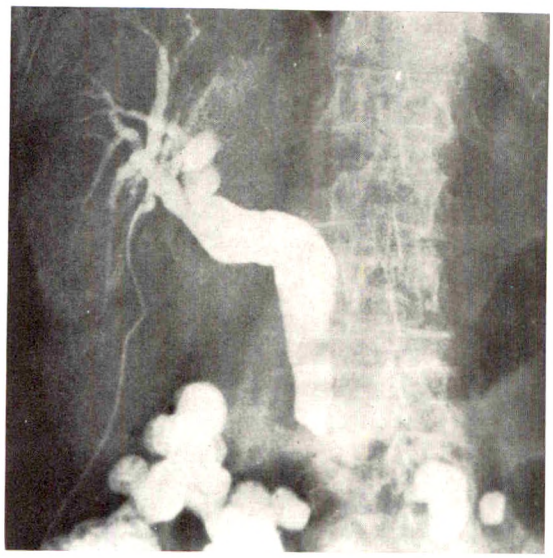


Fig. 6. Percutaneous transhepatic cholangiography demonstrating common duct stones with obstruction.

sequential scans. This patient was one of the earlier cases in the series and only had the sequential scans without the additional information to be gained from the clearance studies. Three other cases showing a scan picture of obstruction and a blood retention of over 90 per cent proved to have nonsurgical jaundice.

DISCUSSION

Patency of the common duct can be readily demonstrated by the sequential scan technique, even in the presence of elevated or rising bilirubin levels. This ability to rule out surgical jaundice in these patients is of great importance because of the risk of surgery in patients with primary liver disease.[6]

Nondilated biliary ducts in patients with a patent common duct are not as readily demonstrated by percutaneous transhepatic cholangiography as are the dilated ducts in patients with an obstructed ductal system, and the isotope procedure is helpful in defining which patients should have percutaneous cholangiography.

The percutaneous cholangiogram has demonstrated the site of obstruction in all cases in which the procedure was performed. Two illustrative cases are shown in Figures 6 and 7, 1 case of multiple common duct stones and 1 case of carcinoma of the common duct.

The additional information gained by the clearance data was helpful in differentiating between extrahepatic obstruction and intrahepatic cholestasis. Three cases of primary hepatocellular disease with intrahepatic cholestasis were correctly diagnosed by the combined blood clearance study and sequential scans. One case of chronic hepatitis was diagnosed as extrahepatic obstruction. However, as stated above, only the sequential scans were done in this case and clearance studies are necessary to make this differentiation.

A case of adenoma of the ampulla of Vater and a case of a floating common duct stone were not correctly diagnosed by this combined testing procedure. These cases of intermittent ball-valve type obstruction still present a problem. Of the total of 59



Fig. 7. Percutaneous transhepatic cholangiography demonstrating obstruction by a carcinoma of the common duct.

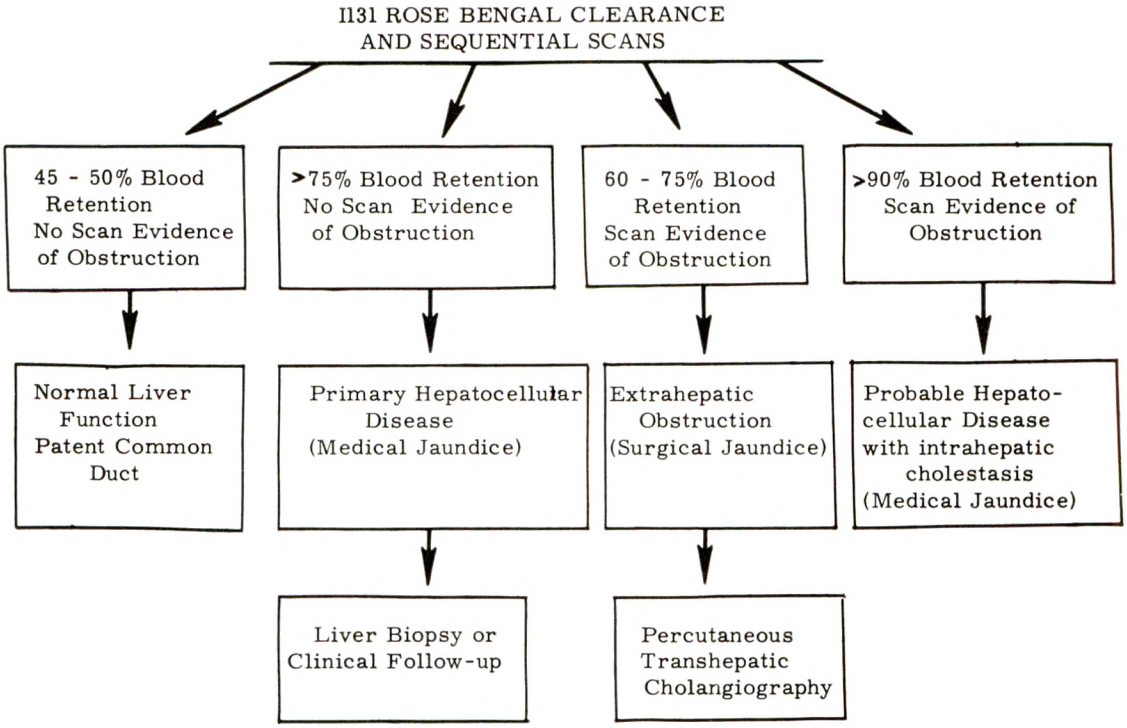


FIG. 8. Summary of most reliable findings of combined blood clearance studies and sequential scans.

cases, 56 were correctly diagnosed as either surgical or nonsurgical jaundice. This diagnostic accuracy of 95 per cent is comparable to that reported by Nordyke and Blahd,[8] and by Whiting and Nusynowitz,[13] using radioactive rose bengal for this differential diagnosis.

Figure 8 presents a summary of the most reliable findings utilizing this approach to the differential diagnosis of jaundice.

SUMMARY

The combination of liver clearance of I^{131} rose bengal from the blood and sequential liver scanning supplemented by percutaneous transhepatic cholangiography when indicated has proven to be very helpful in the differential diagnosis of surgical *versus* nonsurgical jaundice, and in a number of cases has given us information that was not readily available in any other way.

This approach has been invaluable in obviating surgery when not indicated, and in giving the surgeon important preoperative information on which to base his surgical approach.

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REFERENCES

1. CARLSON, H. C. Percutaneous transhepatic cholangiography. *Med. Clin. North America*, 1970, *54*, 875–879.
2. DELPRAT, G. D., EPSTEIN, N. W., and KERR, W. J. New liver function test: elimination of rose bengal when injected into circulation of human subjects. *Arch. Int. Med.*, 1924, *34*, 533–541.
3. DODD, G. D. Percutaneous transhepatic cholangiography. *Surg. Clin. North America*, 1967, 47, 1095–1114.
4. EYLER, W. R., DUSAULT, L. A., POZNANSKI, A. K., and SCHUMAN, B. Isotope scanning in evaluation of jaundice patients. *Radiol. Clin. North America*, 1966, *4*, 589–603.
5. FREEMAN, L. M., and KAY, C. Radioactive rose bengal abdominal scanning in jaundice patients. *New York J. Med.*, 1966, *66*, 1778–1781.
6. HARVILLE, D. D., and SUMMESKILL, W. H. Sur-

gery in acute hepatitis. *J.A.M.A.*, 1963, *184*, 257–261.

7. Nordyke, R. A., and Blahd, W. H. Differential diagnosis of biliary tract obstruction with radioactive rose bengal. *J. Lab. & Clin. Med.*, 1958, *51*, 565–579.
8. Nordyke, R. A., and Blahd, W. H. Blood disappearance of radioactive rose bengal: rapid simple test of liver function. *J.A.M.A.*, 1959, *170*, 1159–1164.
9. Nordyke, R. A. Surgical vs. non-surgical jaundice: differentiation by combination of rose bengal I^{131} and standard liver function tests. *J.A.M.A.*, 1965, *194*, 949–953.
10. Taplin, G. V., Meredith, O. M., Jr., and Kade, H. Radioactive I^{131}-tagged rose bengal uptake-excretion test for liver function using external gamma-ray scintillation counting techniques. *J. Lab. & Clin. Med.*, 1955, *45*, 665–678.
11. Thorbjarnarson, B. Anatomical diagnosis of jaundice by percutaneous cholangiography and its influence on treatment. *Surgery*, 1967, *61*, 347–354.
12. Turner, F. W., and Costopoulos, L. B. Percutaneous transhepatic cholangiography: study of 115 cases. *Canad. M. A. J.*, 1968, *99*, 513–521.
13. Whiting, E. G., and Nusynowitz, M. L. Radioactive rose bengal in differential diagnosis of jaundice. *Surg., Gynec. & Obst.*, 1968, *127*, 729–733.

INDICATION OF SELECTIVE HEPATIC ANGIOGRAPHY*

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IT HAS recently become evident that selective celiac angiography is an important method for diagnosing diseases of the upper abdominal organs. In evaluating the examination of the celiac vessels, however, one may experience the limitations of a conventional method; *i.e.*, optimal visualization of the finer vessels of the organ examined is not always achieved and there is often superimposition of other vessels on the organ. When the artery supplying the organ or a portion of the organ in question is directly catheterized by passing a catheter beyond the celiac axis, these limitations can be overcome. The reports by Boijsen,[6] Judkins *et al.*,[8] Reuter,[10] Rösch and Grollman,[11] and Takashima *et al.*[13] have already indicated that superselective celiac angiography is a useful method to visualize the small intrapancreatic vessels. Similarly, in diseases of the liver and gallbladder, the superselective celiac examinations, *e.g.*, proper hepatic and right or left hepatic angiography, are of paramount value. However, the method should not be considered as a replacement, but rather a supplement of conventional celiac angiography.

The purpose of this paper is to discuss the indication of selective hepatic angiography.

METHOD

Selective hepatic angiography was performed by the Seldinger technique of percutaneous catheter introduction through the femoral route. The details of superselective celiac catheterization have already been described.[15] Before the superselective catheterization, a routine celiac angiogram is made with the Kifa red catheter.

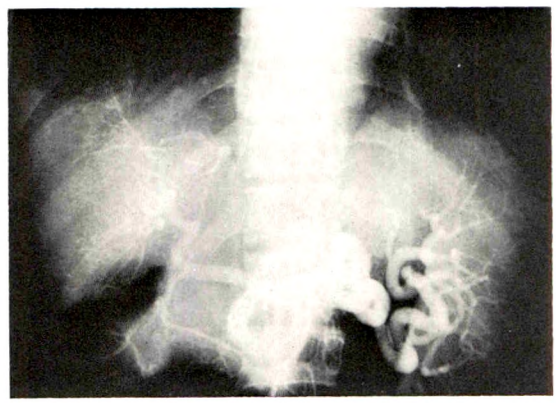


FIG. 1. Case 1. Celiac angiogram. The tortuous and dilated splenic artery and splenomegaly are seen. A large mass in the right lobe of the liver can be noted by displacement and stretching of the surrounding vessels. Tumor vessels, however, can hardly be appreciated.

Ten to 15 ml. of Conraxin-H* for proper hepatic angiography and 7 to 10 ml. for right or left hepatic angiography, respectively, is injected with pressure. Films are exposed at the rate of 4 per second for 3 seconds, 1 per second for 4 seconds, and then 1 every other second for 4 seconds, in the anteroposterior projection and occasionally in the lateral projection.

In cases with an accessory hepatic artery originating from the superior mesenteric artery, a selective accessory hepatic artery catheterization is performed using the same technique.

REPORT OF CASES

CASE 1. S.O., a 48 year old male, presented with hepatomegaly. Liver scan showed an enlarged liver and a large defect in the right lobe.

Celiac angiography (Fig. 1). A moderately

* Sixty per cent of sodium and N-methylglucamine salt of iodamide. Takeda Co., Osaka, Japan.

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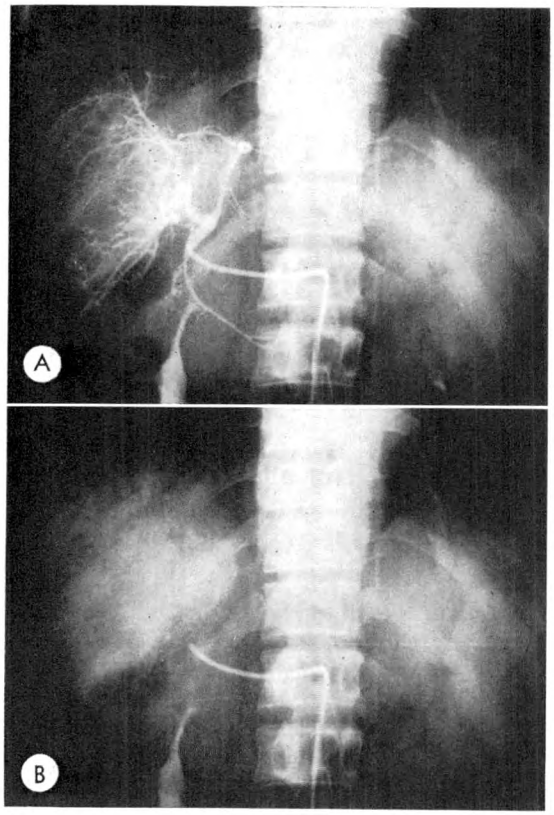


FIG. 2. Case 1. (*A*) Selective hepatic angiogram. Numerous tumor vessels are demonstrated in the mass compared with the conventional study. (*B*) Selective hepatic angiogram The capillary phase shows a dense tumor stain.

tortuous, dilated splenic artery and a large mass with displacement and stretching of the neighboring arteries in the right lobe of the liver were demonstrated. Because of the excessive contrast material flowing into the splenic artery, the tumor vessels in the right lobe of the liver were not clearly visualized.

Selective hepatic angiography (Fig. 2, *A* and *B*). The numerous tumor vessels and the displacement of the adjacent arteries were more clearly demonstrated than with the celiac injection.

Pharmacologic (norepinephrine) *hepatic angiography* (Fig. 3, *A* and *B*). A small amount of norepinephrine (10–20 ug.) was injected through the catheter before the injection of the contrast medium. The identification of the tumor vessels was further improved by the intra-arterial administration of norepinephrine.

Arteriographic diagnosis. Cirrhosis of the liver with hepatoma.

Surgical findings. Hepatoma located in the right lobe of the liver.

CASE II. K.T., a 61 year old male, presented with a palpable liver, the lower border extending 5 cm. below the xiphoid process. The liver scan showed a small defect in the dome of the right lobe with an enlarged left lobe.

Celiac angiography. (Fig. 4, *A* and *B*). A solitary vascularized tumor was faintly seen in the upper part of the right lobe. An enlargement of the left lobe was suspected by the stretching of the vessels in the lobe.

Selective hepatic angiography (Fig. 5, *A* and

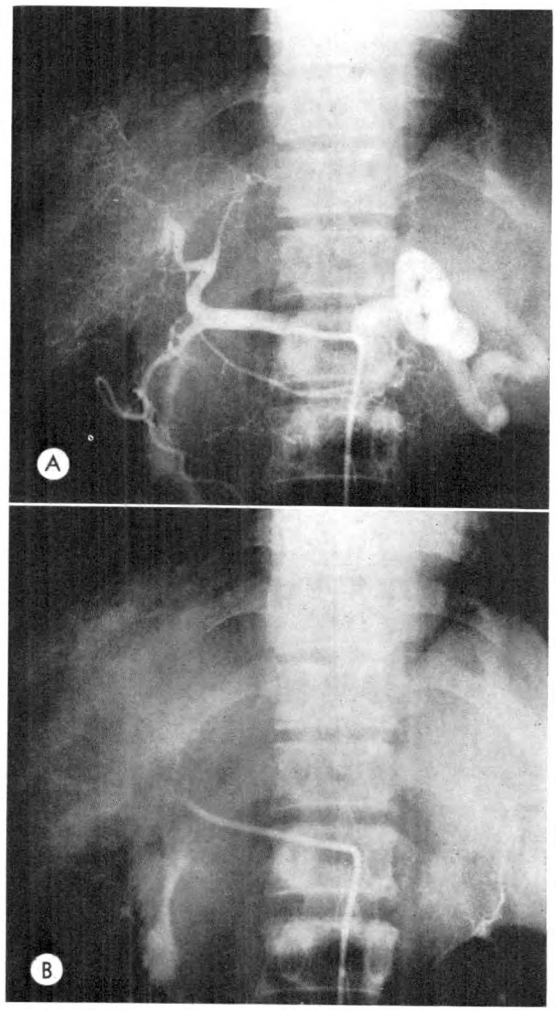


FIG. 3. Case 1. (*A*) Pharmacologic (norepinephrine) hepatic angiogram. There is further improvement in identifying the tumor vessels in the right lobe mass. (*B*) Pharmacologic hepatic angiogram. Fine tumor vessels are best visualized in the early capillary phase.

B). The lesion of the right lobe was better visualized than with the celiac injection. Nodular metastases, not detected in the celiac angiogram, were distinctly seen in the left lobe.

Angiographic diagnosis. Nodular form of hepatoma with metastases in the left lobe.

Autopsy findings. Metastatic carcinoma of the right and left lobe of the liver from probable bile duct carcinoma.

Case III. M.T., a 57 year old male, presented with low grade fever. The liver scan showed a small defect in the upper part of the liver.

Celiac angiography (Fig. omitted). An avascular lesion was suspected in the right side of the left lobe.

Left hepatic angiography. The frontal view (Fig. 6) demonstrated a large avascular mass with displacement and stretching of the neighboring hepatic arteries. The lateral view (Fig. 7) revealed that the mass was located anteriorly in the left lobe.

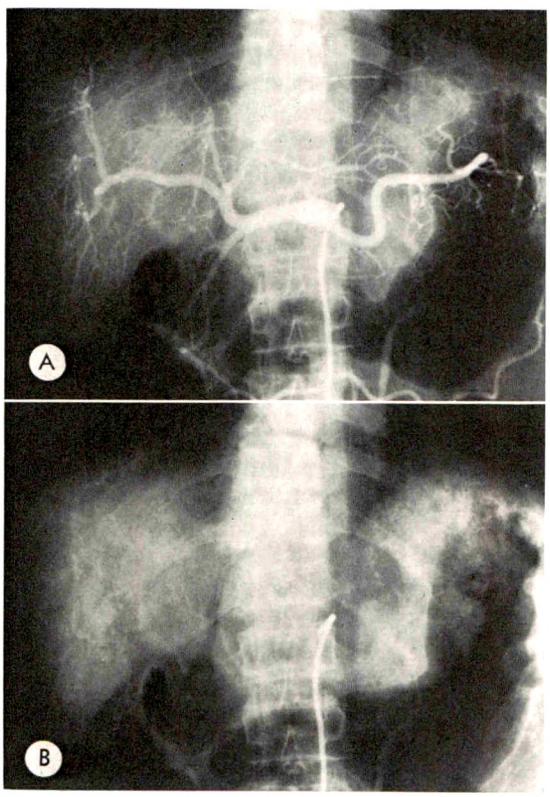


Fig. 4. Case II. (*A*) Celiac angiogram. A vascularized tumor faintly seen in the right lobe appears to be solitary. An enlargement of the left lobe is suggested by stretching of the vessels in the lobe. (*B*) Celiac angiogram. A second lesion in the left lobe is obscured by fundic blush of the stomach and the opacified spleen in the capillary phase.

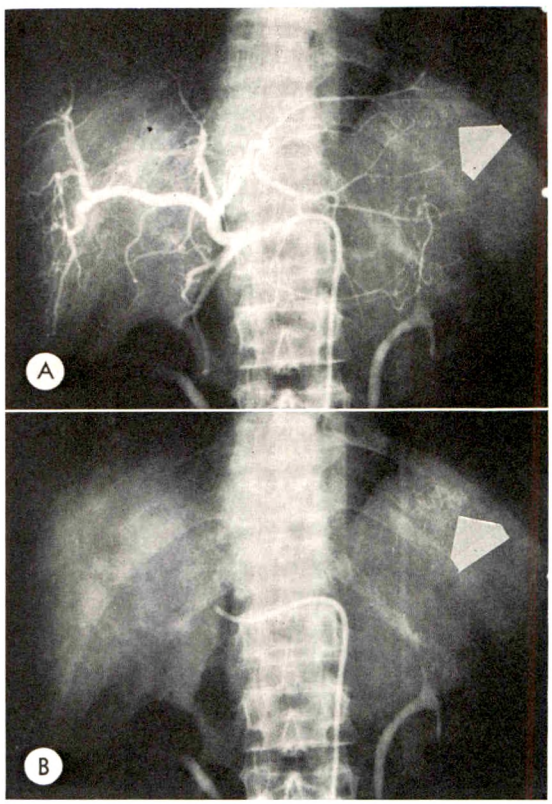


Fig. 5. Case II. (*A*) Selective hepatic angiogram. The tumor in the right lobe is well visualized and another focus of hypervascularity in the left lobe is now distinct (white marker). (*B*) Selective hepatic angiogram. The capillary phase shows tumor stain (white marker) in the left lobe with lack of superimposition of other organs.

Angiographic diagnosis. Metastasis, cyst, or abscess?

Surgical findings. Huge abscess of the anterior portion of the left lobe, with periappendiceal abscess.

Case IV. Y.K., a 61 year old female, presented with obstructive jaundice and enlarged liver.

Celiac angiography (Fig. omitted). A right aberrant hepatic artery from the superior mesenteric artery was noted.

Superior mesenteric angiography (Fig. 8, *A* and *B*). No pathology was found except hepatomegaly.

Selective hepatic angiography (Fig. 9, *A* and *B*). There were numerous nodular shadows with

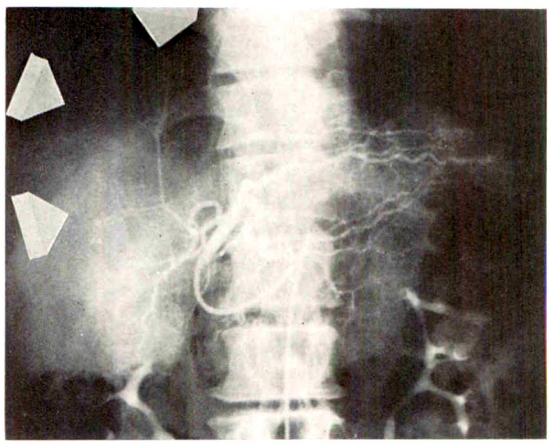


FIG. 6. Case III. Left hepatic arteriogram (frontal view). A large avascular mass (white markers) is demonstrated by displacement and stretching of adjacent hepatic arteries.

pooling of contrast material throughout the right lobe.

Angiographic diagnosis. Metastatic liver cancer from unknown primary.

Surgery. Not done.

INDICATIONS OF SELECTIVE HEPATIC ANGIOGRAPHY

The value of celiac angiography in the diagnosis of liver disease has been well established. The most common indication for celiac angiography in our department is for diagnosis of primary and metastatic tumors of the liver. In reviewing 300 celiac axis angiograms obtained for this purpose, the method has proven to be of definite, but partly limited value.

One of the more promising facets in the current vascular roentgenology might be the development of a technique which enables introduction of a catheter closer to the target organ; *i.e.*, the superselective technique. However, in our opinion, a superselective study should not be considered as a replacement for conventional celiac angiography, but a supplement to it. The indication arises only when an injection of the main trunk does not show sufficient diseased processes.

The injection of hepatic arteries is indicated: (1) when celiac injection does not show the optimal filling of the peripheral hepatic vessels (*e.g.*, when the splenic artery is dilated, Case I); (2) when pathology is suspected in the left lobe of the liver (in a case where partial hepatectomy is contemplated, Case II); (3) when a lateral view of hepatic arteriography is required for the surgical approach (Case III); (4) when pharmacologic angiography is indicated for the intrahepatic vessels (Case I); (5) when a superior mesenteric artery injection does not achieve optimal filling of the small hepatic arteries in cases with an aberrant right hepatic artery from the superior mesenteric artery (Case IV); and (6) when the target organ is the gallbladder.

DISCUSSION

Because the primary, as well as some of the secondary tumors of the liver are almost exclusively supplied by the hepatic artery, hepatic arteriography has an advantage over other angiographic methods. Usually an abundance of tumor vessels is

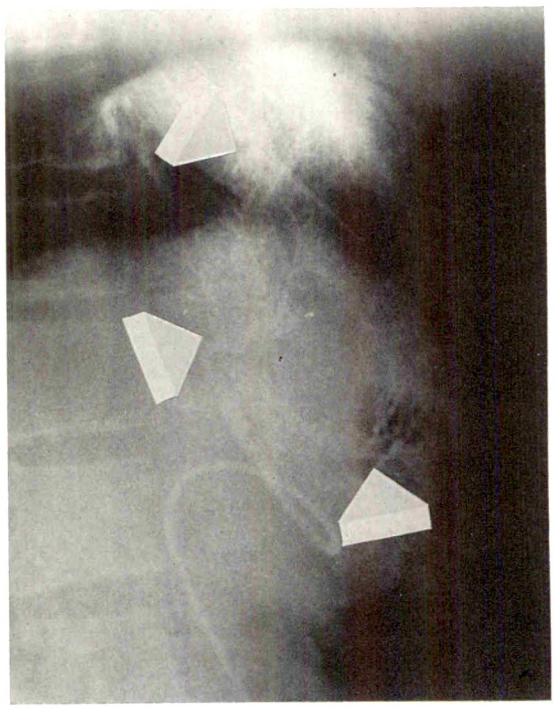


FIG. 7. Case III. Left hepatic angiogram (lateral view). The mass is located anteriorly in the left lobe of the liver (white markers).

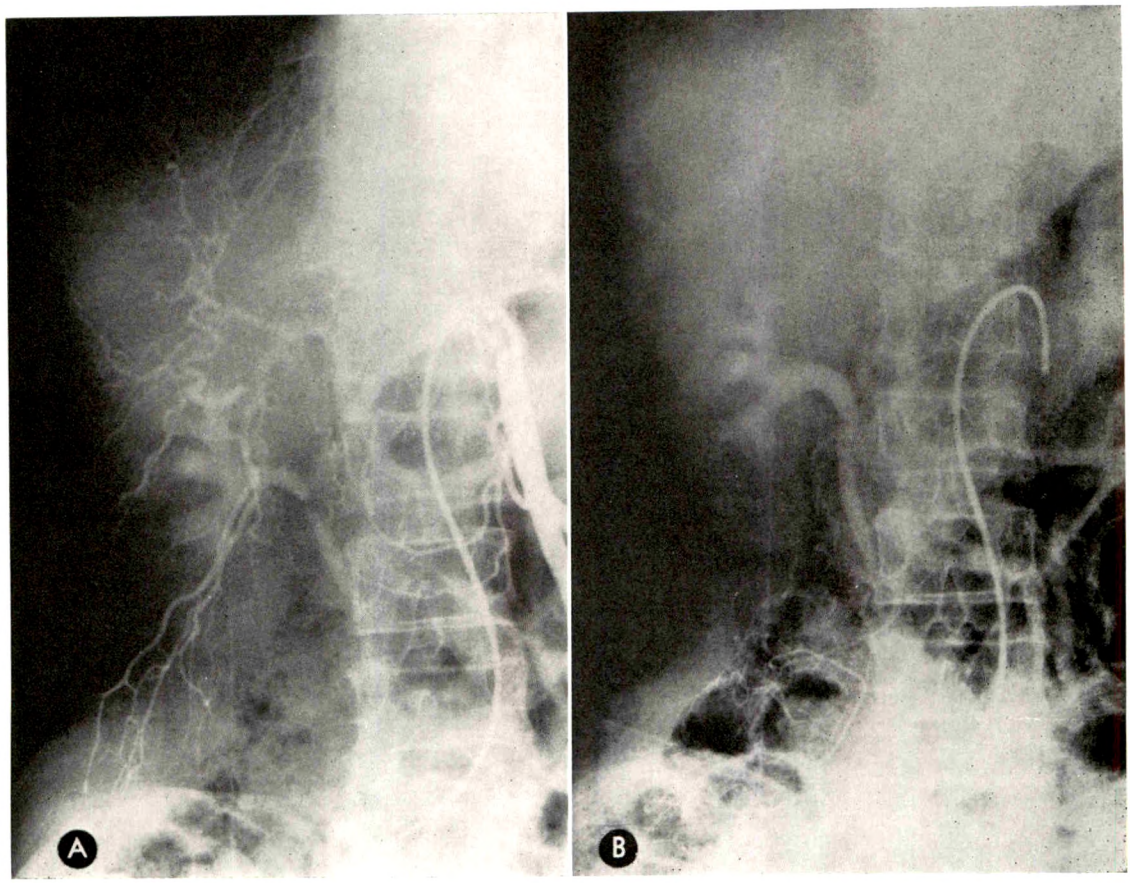


Fig. 8. Case IV. (*A*) Superior mesenteric angiogram. The right hepatic artery arises from the superior mesenteric artery. An enlargement of the right lobe is noticed. (*B*) Superior mesenteric angiogram. The capillary phase is negative.

seen by the selective examination of the celiac artery. However, when a large amount of contrast medium is deposited within organs other than the liver (*i.e.*, in the spleen), small lesions within the liver are not detected. Selective hepatic injection is helpful in such cases (Fig. 1–3). In addition to the obviously better filling of the tumor vessels, an advantage of the selective method is that only the hepatic vessels are filled.

The use of pharmacologic agents to enhance visualization of the tumor vessels is under study. Epinephrine has been shown to affect normal vessels but not diseased vessels. The use of this drug with renal angiography promises to enhance visualization of the diseased vessels of the kidney.[1] The pharmacologic angiography in the celiac arterial system, however, is not effective in enhancing the visualization of hepatic malignancy because the agent is not injected directly into the artery supplying the organ, unlike the renal artery injection. Kahn *et al.*[9] state that after the administration of epinephrine into the celiac artery, better visualization of the tumor vessels was obtained in less than half of the patients with hepatic tumors. Selective hepatic catheterization may be useful for pharmacologic angiography (Fig. 3).

The localization and extent of the hepatic tumor are of great importance in the surgical treatment.[2,7] Successful excision of a liver tumor requires careful angiographic analysis. The partial hepatectomy might be contraindicated when metastatic lesions in another area are detected. It is usually

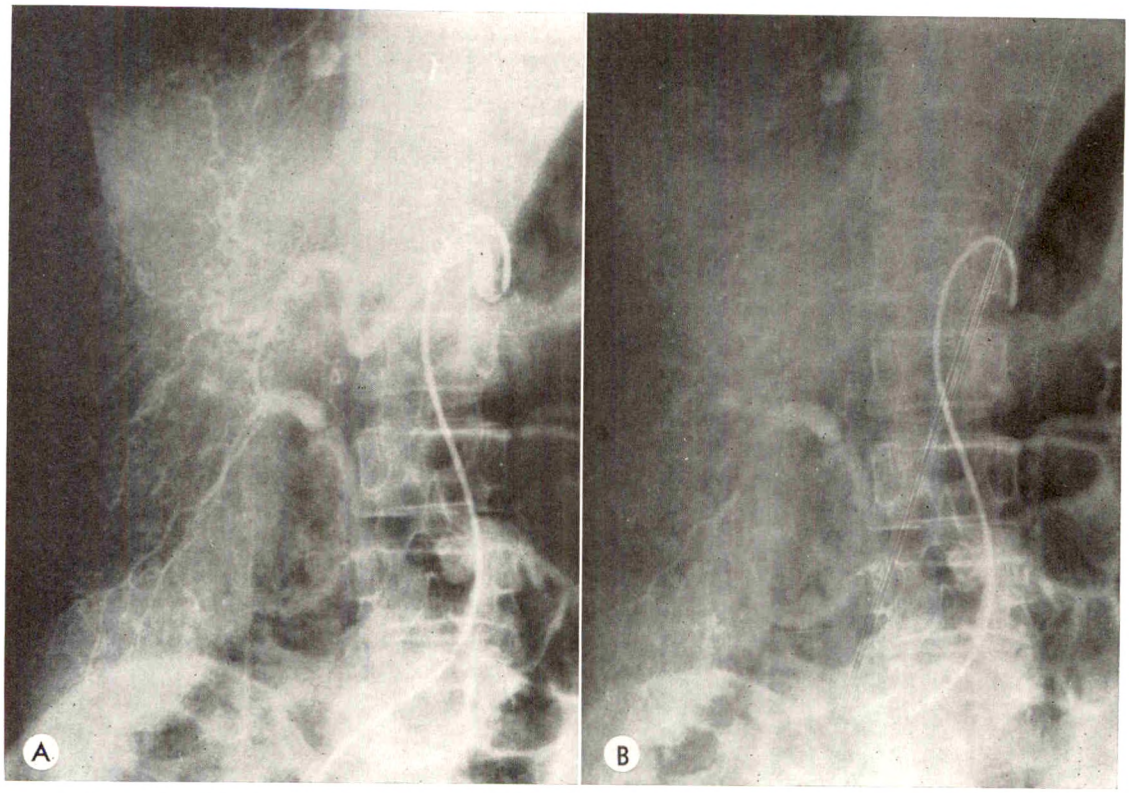


Fig. 9. Case iv. (*A*) Selective hepatic angiogram. Numerous hypervascular nodules are now seen throughout the right lobe. (*B*) Selective hepatic angiogram. The capillary phase is pronounced, as compared to Figure 8*B*.

more difficult to demonstrate the lesions in the left than in the right lobe of the liver.[2,5] At celiac angiography some area of the left lobe is obscured by the contrast-filled vessels of the spleen or stomach, and small vessels of the left lobe are not well visualized. These difficulties could be overcome by selective hepatic injections (Fig. 4; and 5). When surgical drainage for hepatic abscess is planned, it is important to assess the localization in order to determine the surgical approach. For the precise localization, the lateral arteriogram by selective hepatic injection is mandatory. Celiac arteriography does not produce the same good quality as lateral hepatic arteriograms, because of the overlap of other undesirable vessels, such as the splenic and gastric vessels (Fig. 6; and 7).

The arterial supply of the liver varies, and often partly originates from the superior mesenteric artery. In such cases, superior mesenteric angiography is not sufficient to detect the small lesions of the liver because nearly all of the contrast agent flows to the superior mesenteric system. The angiographic quality has to be excellent in order to demonstrate the small intrahepatic tumor. Selective hepatic catheterization beyond the trunk of the superior mesenteric artery definitely enhances the diagnosis of small hepatic pathologic lesions (Fig. 8; and 9).

The conventional methods of roentgen examination are disappointing for the diagnosis of malignant gallbladder tumors, although several authors express hope that the extremely poor diagnostic value may be improved by the frequent use of celiac angiography.[12,14] A comparison of the visualization of the cystic artery and the gallbladder wall was made using selective hepatic and celiac angiography. With celiac injections, the cystic artery was visualized in 72.3

per cent of 300 cases and the gallbladder wall in only 21.2 per cent of cases.[14] On the other hand, with selective hepatic injections, the cystic artery was visualized in 84.3 per cent, and the gallbladder in 51 per cent. Since good visualization of the gallbladder area in both the arterial and capillary phase is of the utmost importance for the diagnosis, the selective hepatic injection is a necessity in patients with suspected gallbladder tumor.

Recently, the direct serial magnification technique has been employed.[3,4] In order to achieve a full resolution by enlargement, filling of the smaller vessels and avoidance of overlapping of the superfluous vessels are essential. The angiographic diagnosis of hepatic diseases will be hopefully improved by the use of direct magnification technique, necessitating selective hepatic injection.

CONCLUSION

Hepatic angiography by the selective catheterization of the hepatic arteries is a useful procedure for the evaluation of not only the pathologic diagnosis, but also the extent and localization of the hepatic tumor.

This procedure is especially valuable in detecting lesions of the left lobe of the liver and in performing pharmacologic angiography.

The superselective study, however, should always be performed according to certain indications, following and supplementing conventional celiac angiography.

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REFERENCES

1. Abrams, H. L. Response of neoplastic renal vessels to epinephrine in man. *Radiology*, 1964, *82*, 217–224.
2. Bartley, O., Edlund, Y., and Helander, C. G. Angiography in primary hepatic carcinoma. *Acta radiol.* (Diag.), 1967, *6*, 81–90.
3. Baum, S. Hepatic arteriography. *Am. J. Gastroenterol.*, 1969, *51*, 151–153.
4. Baum, S., Nusbaum, M., Kuroda, K., and Blakemore, W. S. Direct serial magnification arteriography as adjuvant in diagnosis of surgical lesions in alimentary tract. *Am. J. Surg.*, 1969, *117*, 170–176.
5. Boijsen, E. Selective hepatic angiography in primary and secondary tumors of liver. *Rev. Int. Hepat.*, 1965, *15*, 385–395.
6. Boijsen, E. Selective pancreatic angiography. *Brit. J. Radiol.*, 1966, *39*, 481–487.
7. Bosniak, M. A., and Phanthamachinda, P. Value of arteriography in study of hepatic disease. *Am. J. Surg.*, 1966, *112*, 348–355.
8. Judkins, M. P., Billimoria, P. E., Green, G. S., and Dotter, C. T. Superselective visceral angiography. Book of abstracts from XII ICR meeting, Tokyo, 1969.
9. Kahn, P. C., Frates, W. J., and Paul, R. E. Epinephrine effect in angiography of gastrointestinal tract tumors. *Radiology*, 1967, *88*, 686–690.
10. Reuter, S. R. Superselective pancreatic angiography. *Radiology*, 1969, *92*, 74–85.
11. Rösch, J., and Grollman, J. H., Jr. Superselective arteriography in diagnosis of abdominal pathology. *Radiology*, 1969, *92*, 1008–1013.
12. Rösch, J., Grollman, J. H., Jr., and Stecke, R. J. Arteriography in diagnosis of gallbladder disease. *Radiology*, 1969, *92*, 1485–1491.
13. Takashima, T., Yamamoto, I., Mitani, I., and Shin, M. Transfemoral superselective celiac angiography. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *110*, 813–817.
14. Takashima, T., and Shin, M. Selective hepatic angiography in diagnosis of bile duct disease. *Naika* (Japan), 1971, *27*, 647–655.
15. Takashima, T., and Shin, M. Transfemoral superselective celiac catheterization: technical considerations. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1971, *113*, 280–282.

CARCINOMA OF THE GALLBLADDER: DIAGNOSIS AND EVALUATION OF REGIONAL SPREAD BY ANGIOGRAPHY*

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PREVIOUS reports have shown that carcinoma of the gallbladder may be diagnosed by means of celiac and superior mesenteric angiography.[1,5,15]

The present study demonstrates the role of angiography in evaluating local extension and regional metastases. Emphasis is placed upon venous and small artery abnormalities, especially in the lesser omentum and pancreatic region, which often indicate further extension of neoplasm than would otherwise be suspected.

REPORT OF CASES AND ANGIOGRAPHIC FINDINGS

CASE I. I.K., a 68 year old white female, was admitted following 3 weeks of abdominal pain and 1 week of anorexia. Jaundice developed 1 week prior to admission. Physical examination revealed a markedly icteric female. The abdomen was guarded. The liver was enlarged to 6 cm. below the right costal margin. A 5 cm. firm nodular mass, which was questionably adherent to the liver, was palpated in the right upper quadrant. The total bilirubin was 37 mg. per cent and the alkaline phosphatase was elevated. Routine abdominal roentgenograms showed a large soft tissue mass in the right upper quadrant with displacement of the hepatic flexure of the colon. A blastic metastasis was seen in the body of the 3rd lumbar vertebra. Upper gastrointestinal series revealed circumferential narrowing of the postbulbar duodenum. Liver scan showed a large liver with multiple defects in the right lobe consistent with metastatic disease.

Abdominal fluid, in the amount of 200 cc., was removed and the cytologic studies showed adenocarcinoma. No operation was performed. The patient's condition deteriorated rapidly. Autopsy revealed primary carcinoma of the gallbladder with extensive local spread as well as a distant metastasis to L3.

Angiography. Celiac and superior mesenteric arteriograms (Fig. 1, *A* and *B*; and 2, *A* and *B*) showed encasement of the hepatic and gastroduodenal arteries and of the pancreaticoduodenal arcade. The branches of the cystic artery were stretched. Neovascularity was noted in the gallbladder bed arising from the cystic artery, adjacent right hepatic arteries, and branches of the gastroduodenal and right gastroepiploic arteries. A large dense blush was noted in the parenchymal phase.

The pericholecystic veins were enlarged and there was early filling of the hepatic veins. The portal vein, splenic vein just before its junction with the superior mesenteric vein, and the superior mesenteric vein were occluded.

CASE II. M.N., a 61 year old white female, presented with a 2 week history of jaundice. Past history revealed attacks of "cholecystitis" several years previously. Upon physical examination the liver was enlarged to a finger breadth below the costal margin. Upper gastrointestinal series was negative. Following angiography the patient underwent surgical exploration and a carcinoma of the ampulla of the gallbladder was found with cystic and common duct obstruction. The gallbladder contained multiple calculi.

Histologic examination of the gallbladder showed an infiltrating adenocarcinoma with margins of resection free of tumor. The patient, however, had a stormy postoperative course, complicated by electrolyte imbalance and gram negative septicemia resulting in her demise. Autopsy was not performed.

Angiography. Celiac and superior mesenteric arteriograms (Fig. 3, *A–C*; and 4, *A* and *B*) showed enlargement of the cystic artery with stretching of the cystic branches. Encasement

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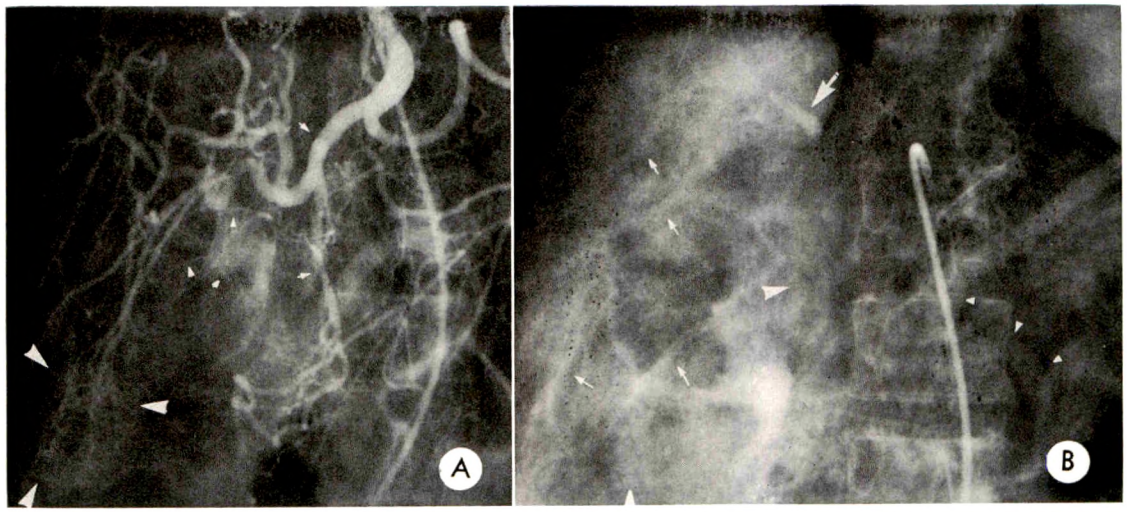


Fig. 1. Case 1. *Celiac arteriography.*

(*A*) Arterial phase shows encasement of the hepatic and gastroduodenal arteries (arrows). Tumor vessels and an early tumor blush are present in the right lobe of the liver (large arrowheads). The cystic branches are stretched (small arrowheads) with neovascularity arising from these arteries, the gastroduodenal and right gastroepiploic arteries. (*B*) Late capillary and venous phase shows large ill-defined mottled blush in the right upper quadrant involving the gallbladder bed and liver. Early draining of hepatic veins (small arrows) is noted. The pericholecystic veins (large arrowheads) are prominent. The portal vein is occluded with retrograde filling of the inferior mesenteric vein (small arrowheads). Large arrow points to filling of a branch of the portal vein beyond obstruction.

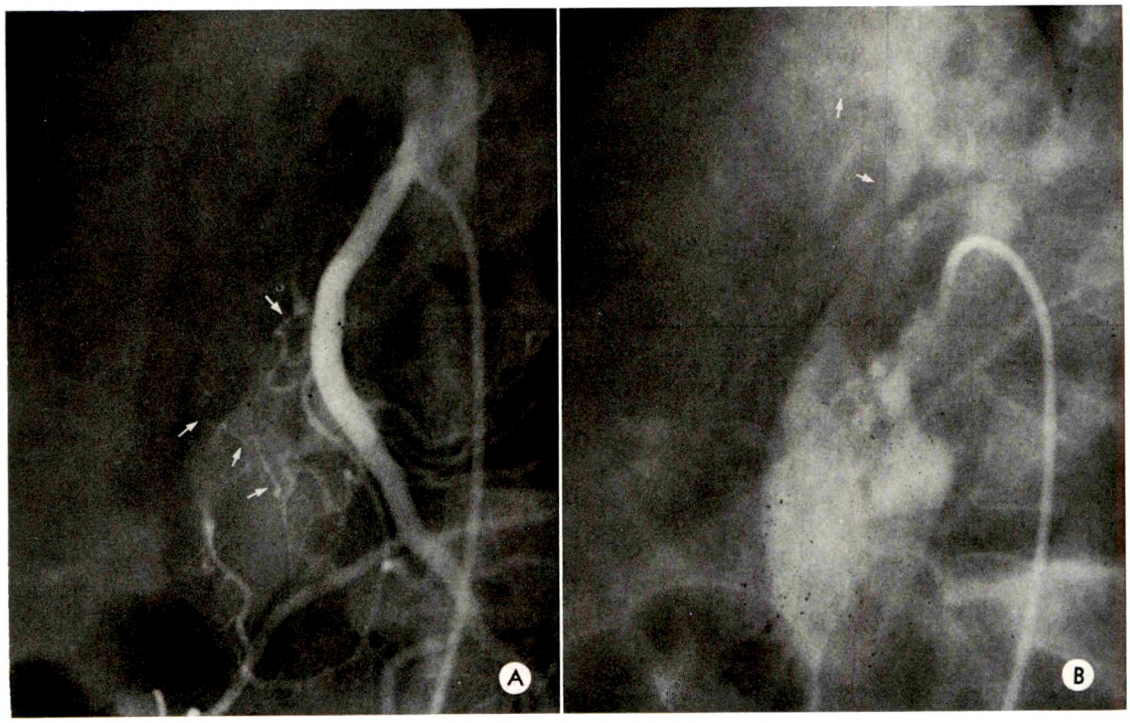


Fig. 2. Case 1. *Superior mesenteric arteriography.*

(*A*) Arterial phase shows encasement of pancreaticoduodenal arcade (arrows). (*B*) Venous phase shows nonfilling of the major superior mesenteric venous trunk with a large collateral vein (arrows) bypassing the obstructed portal vein.

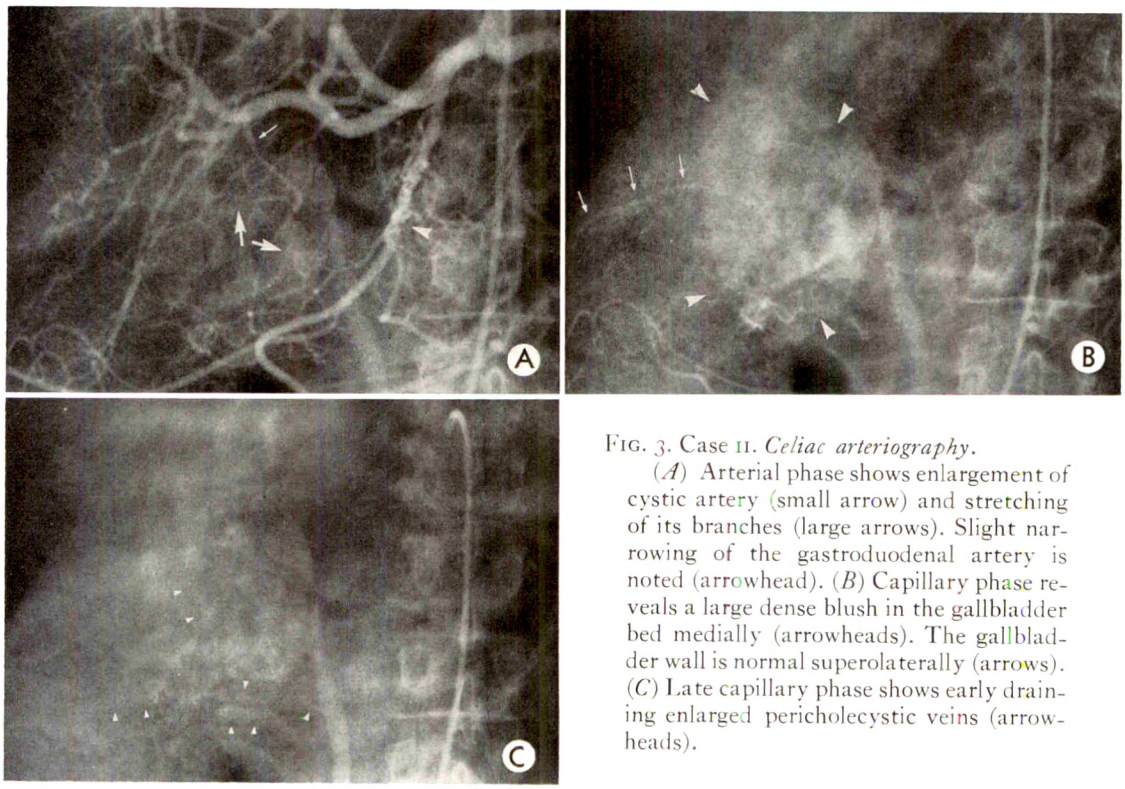


Fig. 3. Case II. *Celiac arteriography.*
(*A*) Arterial phase shows enlargement of cystic artery (small arrow) and stretching of its branches (large arrows). Slight narrowing of the gastroduodenal artery is noted (arrowhead). (*B*) Capillary phase reveals a large dense blush in the gallbladder bed medially (arrowheads). The gallbladder wall is normal superolaterally (arrows). (*C*) Late capillary phase shows early draining enlarged pericholecystic veins (arrowheads).

of the gastroduodenal and superior pancreaticoduodenal arteries was noted.

A large dense blush was present in the medial region of the gallbladder. The gallbladder wall was normal superolaterally.

Enlarged early draining veins arose from the gallbladder bed. The splenic and portal veins were normal. One of the major trunks of the superior mesenteric vein was narrowed.

Case III. L.G., a 54 year old white female, was in good health until a mass was noted in her right upper quadrant. Gallstones had been discovered 15 years previously as an incidental roentgenographic finding. Physical examination revealed a 15 cm. right upper quadrant mass which was irregular and firm. Liver chemistries were within normal limits. A plain roentgenogram of the abdomen demonstrated a right upper quadrant mass and several large calcified gallstones. Barium enema examination was normal.

At exploration, following angiography, carcinoma of the gallbladder was noted with direct invasion of the liver and spread to the regional lymph nodes. In addition, more distant lymph nodes in the periaortic area were enlarged. A resection was not performed, and following liver biopsy the abdomen was closed. Histologic examination revealed metastatic poorly differentiated adenocarcinoma. An autopsy was not performed following the patient's demise.

Angiography. Celiac arteriograms (Fig. 5, *A–C*) showed an enlarged cystic artery and neovascularity arising from the cystic artery, branches of the right hepatic artery in the lower portion of the right lobe of the liver, and from branches of the gastroduodenal artery. A large blush was present in the region of the gallbladder and lower portion of the liver.

Early filling of the hepatic veins and of prominent pericholecystic veins was seen. The portal and splenic veins were normal.

Case IV. E.J., an 82 year old female, was admitted with a 4 day history of epigastric pain and jaundice. An upper gastrointestinal series and barium enema examination were normal. At operation calculi were found in the gallbladder and common duct. A cholecystectomy was performed. No evidence of neoplasm was noted during the operation.

Histologic sections of the gallbladder revealed adenocarcinoma with extension of the

tumor to the serosal surface. The margins of resection were free of tumor. The patient had an uneventful recovery, but approximately 1 year later returned with progressive jaundice of 3 weeks' duration accompanied by a 20 lb. recent weight loss. The abdomen was distended and diffusely tender. Liver function tests showed findings consistent with obstructive jaundice. No operation was performed. The patient was discharged and died at home approximately 2 weeks after discharge. No autopsy was performed.

Angiography. On the first examination prior to cholecystectomy (Fig. 6, *A* and *B*; and 7, *A* and *B*) diffuse atherosclerosis of the visceral arteries was noted. The cystic artery was enlarged and divided into multiple small vessels with a blush in the region of the gallbladder. Prominent cholecystic veins were present. The superior mesenteric, portal and splenic veins were normal.

Angiography 1 year postcholecystectomy (Fig. 8, *A–C*; and 9) showed shortening of the hepatic artery as compared with the prior study. Localized constriction of the hepatic and gastroduodenal arteries was present, and was considered to represent tumor encasement superimposed upon atherosclerotic vessels. The superior pancreaticoduodenal arteries were also encased. No neovascularity or tumor blush was demonstrated. The splenic and portal veins were markedly narrowed. The superior mesenteric vein was normal.

Case v. C.C., a 54 year old white female, presented with a history of recent weight loss, chills, fever, and jaundice of 2 weeks' duration. Physical examination showed a jaundiced woman in no apparent distress. A hard irregular mass was palpated in the right upper quadrant extending from the right costal margin to the level of the umbilicus. The bilirubin was ele-

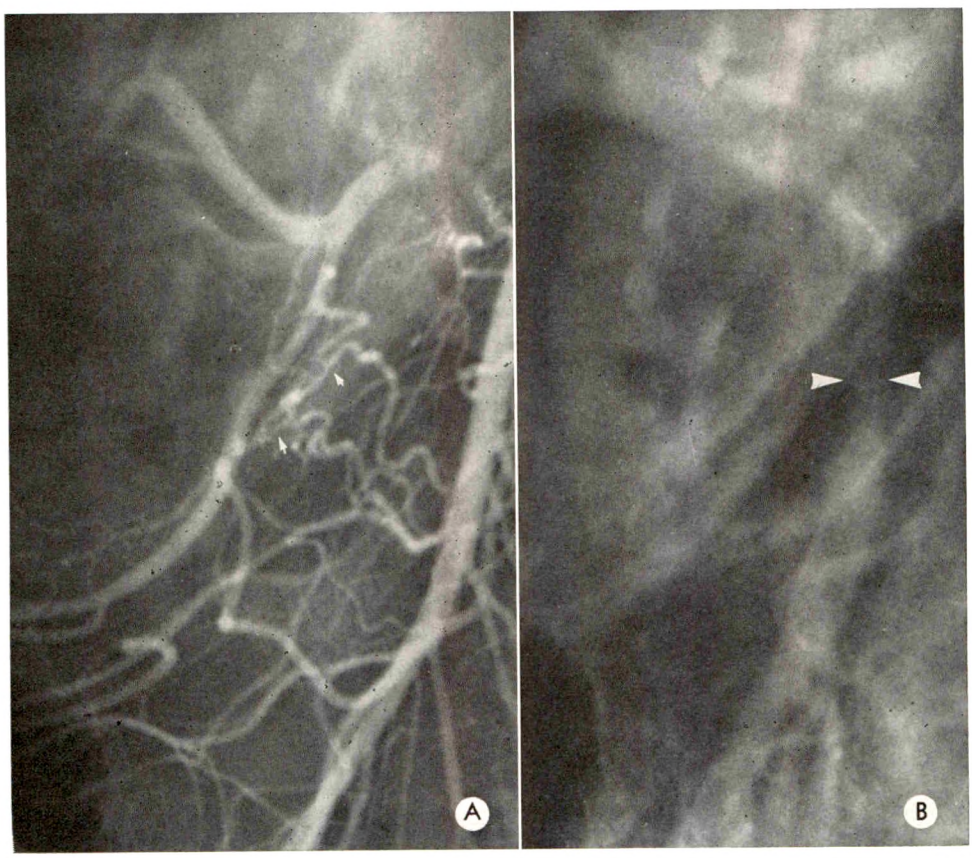


Fig. 4. Case 11. *Superior mesenteric arteriography*.
(*A*) Arterial phase shows encasement of superior pancreaticoduodenal arteries (arrows). (*B*) Venous phase shows narrowing of one of the major trunks of the superior mesenteric vein (arrowheads).

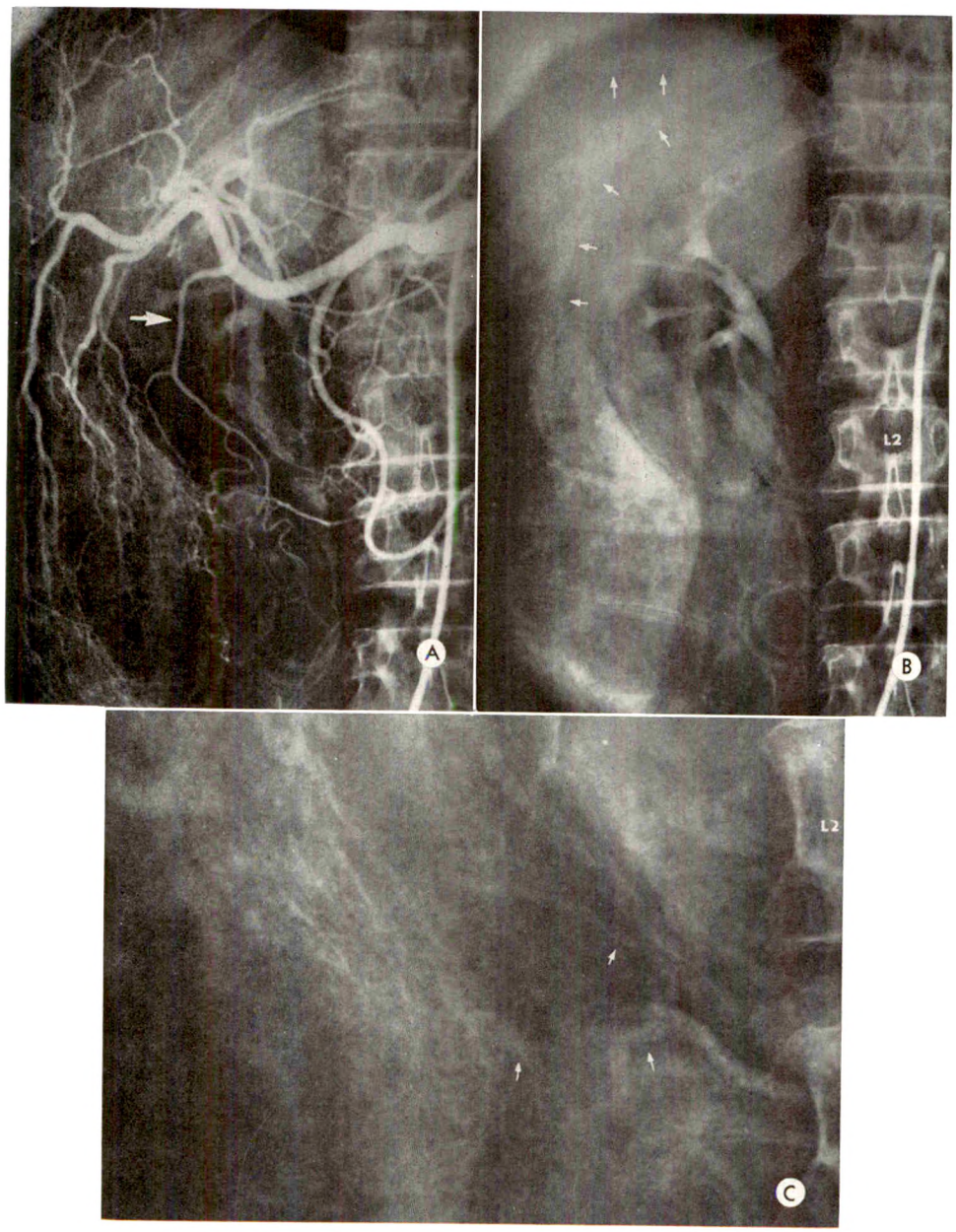


Fig. 5. Case III. *Celiac arteriography.*

(*A*) Arterial phase shows enlarged cystic artery (arrow) with neovascularity arising from cystic, right hepatic arteries and gastroduodenal branches. An early tumor blush is present involving the gallbladder and right lobe of the liver inferiorly. (*B*) Late capillary phase shows early visualization of hepatic veins (arrows) with large blush noted in the right upper quadrant involving the gallbladder bed and right lobe of the liver. (*C*) Late capillary phase from a second injection shows early draining of the enlarged pericholecystic veins (arrows).

vated and other liver chemistries were in keeping with a biliary obstructive process. Intravenous urography, upper gastrointestinal series and barium enema examination were negative.

Following angiography, exploration of the abdomen was performed. Carcinoma of the gallbladder was found with invasion of the liver, omentum, and mesentery of the large bowel, as

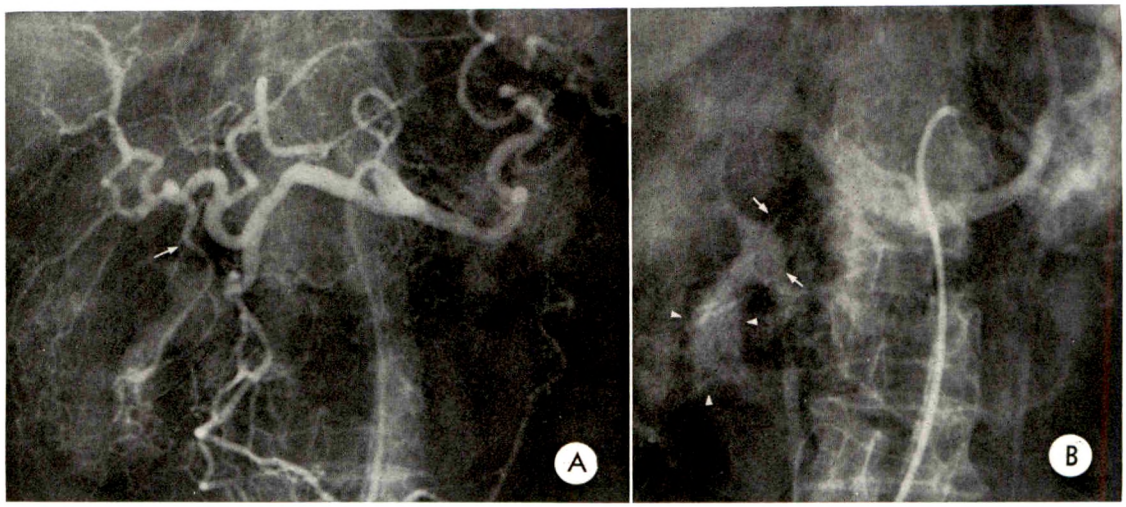


Fig. 6. Case IV. *Celiac arteriography prior to cholecystectomy.*
(*A*) Arterial phase shows diffuse atherosclerosis. The cystic artery is enlarged (arrow) and its branches are tortuous. (*B*) Venous phase demonstrates normal splenic and portal veins. A blush is present in the gallbladder region (arrowheads), and prominent cholecystic veins are noted (arrows).

well as peritoneal seeding. The gallbladder contained multiple calculi.

Angiography. Celiac (Fig. 10, *A* and *B*) and superior mesenteric arteriograms showed abrupt termination of the cystic artery. No neovascularity or tumor blush was present. An avascular defect was noted in the gallbladder bed and adjacent liver region in the capillary phase.

The portal vein was occluded with incomplete filling of the splenic vein just proximal to its junction with the portal vein. A venous phase of the superior mesenteric arteriogram was not obtained.

SUMMARY OF ANGIOGRAPHIC FINDINGS

Table 1 shows the results of angiography in the above 5 cases. Celiac and superior mesenteric arteriograms were obtained in all cases except Case III, in which a superior mesenteric arteriogram was not obtained.

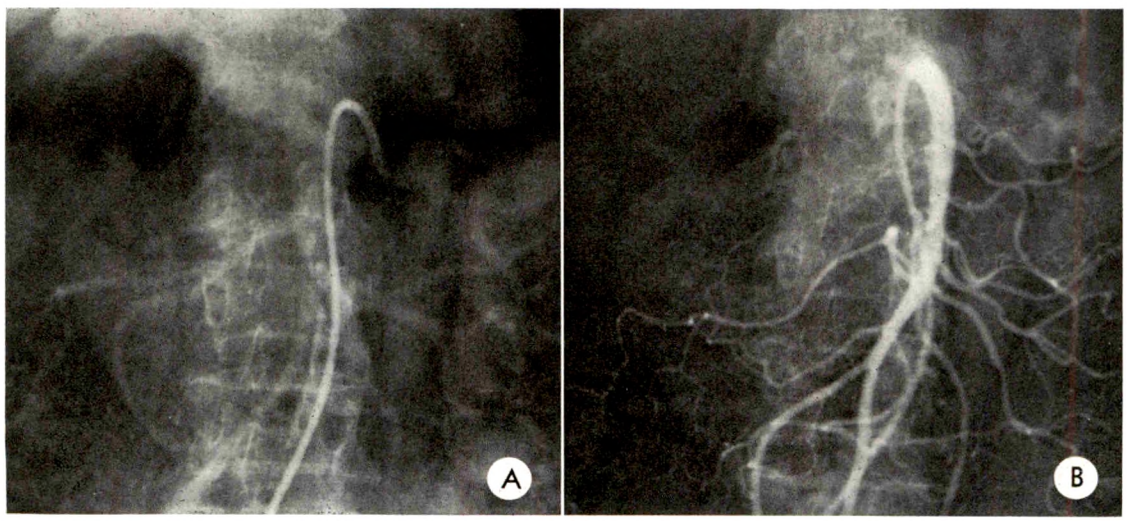


Fig. 7. Case IV. *Superior mesenteric arteriography prior to cholecystectomy.*
(*A*) Normal arterial phase. (*B*) Normal venous phase.

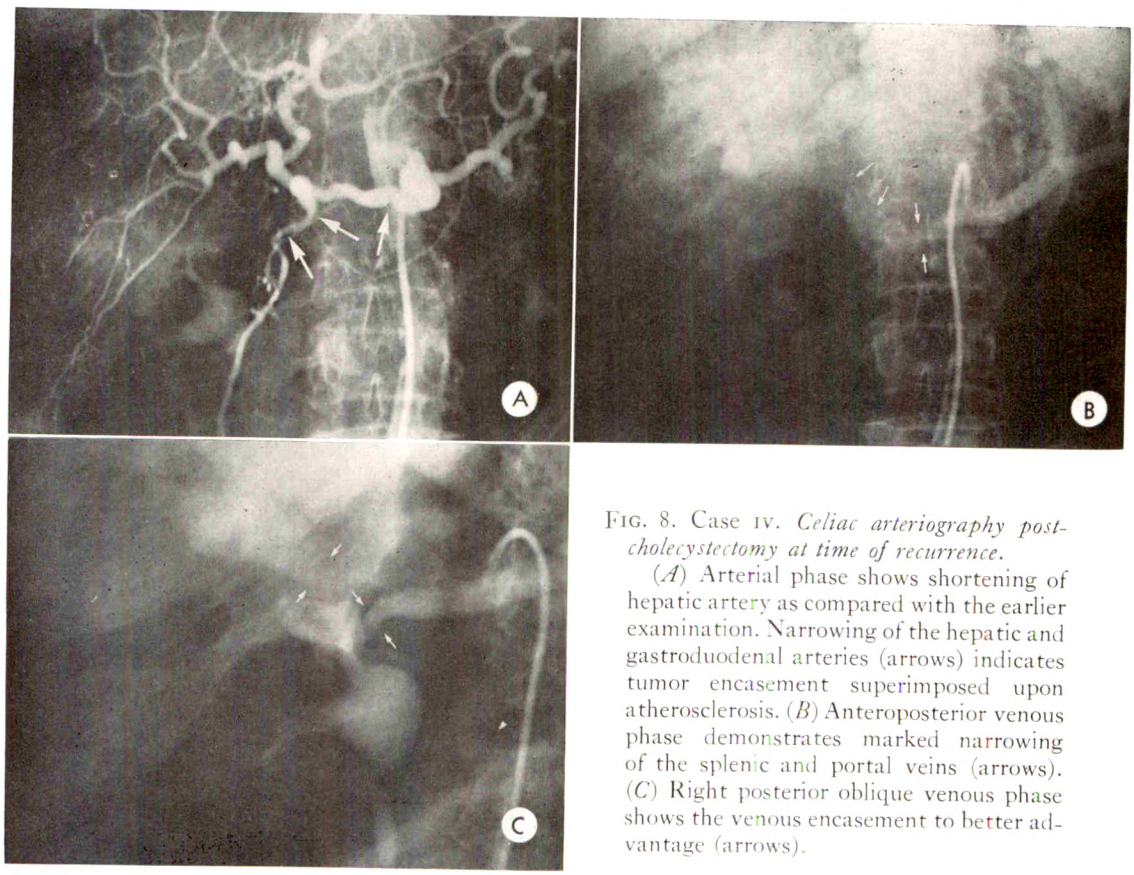


FIG. 8. Case IV. *Celiac arteriography postcholecystectomy at time of recurrence.*

(*A*) Arterial phase shows shortening of hepatic artery as compared with the earlier examination. Narrowing of the hepatic and gastroduodenal arteries (arrows) indicates tumor encasement superimposed upon atherosclerosis. (*B*) Anteroposterior venous phase demonstrates marked narrowing of the splenic and portal veins (arrows). (*C*) Right posterior oblique venous phase shows the venous encasement to better advantage (arrows).

A blush in the gallbladder region was identified in 4 cases. In 3 the blush was large and appeared to merge with the right lobe of the liver. The blush in 1 was confined to the gallbladder. Neovascularity was seen in 2 of the cases with blushes. The neovascularity was slight to moderate and not at all as prominent as that seen in the typical hepatoma. The feeding vessels were the cystic artery and branches of the right hepatic, gastroduodenal and right gastroepiploic arteries. The cystic artery was enlarged in 3 of the 4 cases with blushes.

Enlarged cholecystic or pericholecystic veins were present in all 4 patients with tumor blushes. These veins filled early in 2 cases. Early filling of the hepatic veins was also present in 2 cases. In both, neovascularity was demonstrated in the right lobe of the liver.

In 1 case the cystic artery was not enlarged and no neovascularity, tumor blush,

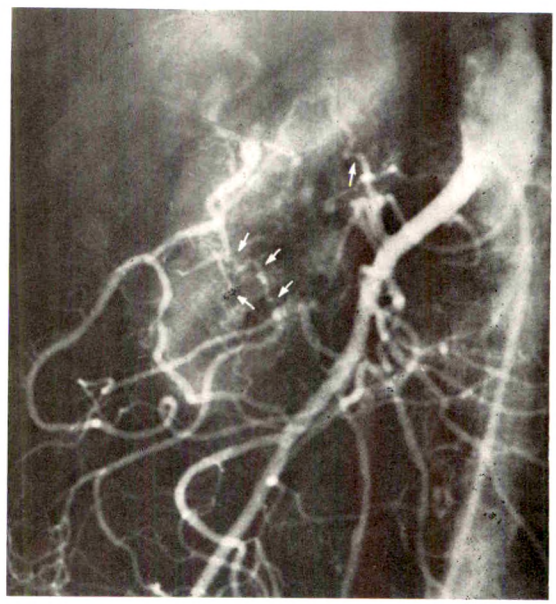


FIG. 9. Case IV. *Superior mesenteric arteriogram 1 year postcholecystectomy.*

Arterial phase shows encasement of the pancreaticoduodenal arteries (arrows).

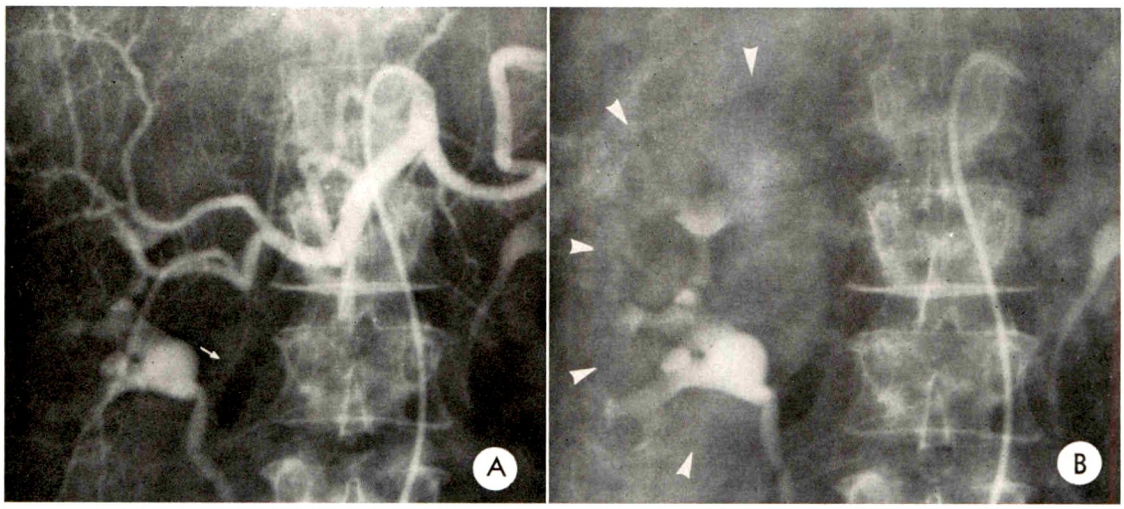


Fig. 10. Case v. *Celiac arteriography.*

(*A*) Arterial phase shows abrupt termination of cystic artery (arrow). No neovascularity is present. (*B*) Late capillary and venous phase shows an avascular defect in the gallbladder bed and medial portion of the liver (arrowheads). Neither the portal vein nor the splenic vein near the liver was visualized.

prominent cholecystic veins or early hepatic vein filling was demonstrated. A negative defect was present in the gallbladder region and adjacent hepatic parenchyma in the capillary phase.

Arterial encasement was present in 4 cases. The gastroduodenal artery was involved in 3 cases, the hepatic artery twice and the cystic artery once. The superior pancreaticoduodenal arteries were encased in 3 cases and the inferior pancreaticoduodenal arteries once. Neither superior mesenteric artery narrowing nor jejunal branch narrowing was noted.

The peripheral branches of the cystic artery were displaced around an enlarged gallbladder in 2 cases.

The portal vein was occluded in 2 cases and narrowed in 1. In these 3 cases, the superior mesenteric vein was occluded in 1, normal in 1, and not studied in 1; the splenic vein was occluded in 2 and narrowed in 1. In the 2 cases with normal portal veins the superior mesenteric vein was

Table I

SUMMARY OF ANGIOGRAPHIC FINDINGS

	Enlarged Cystic Artery	Stretched Cystic Branches	Tumor Encasement								Neovascularity	Blush	Early Hepatic Vein Filling	Prominent Cholecystic Pericholecystic Veins
			Arteries					Veins						
			Cy	H	GD	SPD	IPD	Sp	SM	P				
Case I		+		+	+	+	+	+	+	+	+	+	+	+
Case II	+	+			+	+			+			+		+
Case III	+								NS*		+	+	+	+
Case IV														
a. prior to cholecystectomy	+											+		+
b. 1 year post cholecystectomy				+	+	+		+		+				
Case V			+					+	NS*	+				

Cy = cystic; H = hepatic; GD = gastroduodenal; SPD = superior pancreaticoduodenal; IPD = inferior pancreaticoduodenal; Sp = splenic; SM = superior mesenteric; P = portal.

* Not Studied.

narrowed in 1 and not studied in the other; the splenic vein was normal in both. In the 3 cases with portal vein involvement, the hepatic artery was encased by tumor in 2 and normal in the third. No case had hepatic artery encasement with a normal portal vein.

Two cases showed involvement of the superior mesenteric vein (occluded in 1, narrowed in 1). The superior mesenteric artery was normal in these as it was in all other cases.

DISCUSSION

When carcinoma of the gallbladder spreads beyond the gallbladder, the liver and regional lymph nodes are most frequently involved.[2,6,8,12,14] Local extension to the stomach, duodenum, hepatic flexure of the colon, and abdominal wall may occur. Intraperitoneal seeding and widespread distant metastases occur only occasionally.

The incidence of liver involvement in surgical cases varies from 35 to 80 per cent.[3,6,7,8,10] Autopsy series have shown hepatic involvement in as high as 90 to 100 per cent of cases.[10,11] The liver is primarily involved by direct extension of tumor or by retrograde lymphatic spread. Intraductal and venous spread play a minor role in the spread of disease in surgical cases.[6] Disseminated liver metastases are uncommon, having been described in only 5 of 96 cases in 2 series with hepatic involvement.[4,6] Disseminated liver metastasis was not present in any of our cases.

Fahim and co-workers[6] carefully evaluated hepatic involvement in 9 patients with carcinoma of the gallbladder in whom a portion of the liver was resected at the time of surgery. In 5 of these the liver was involved by direct extension of the primary lesion, while in the other 4 cases malignant cells were entirely within the lymphatics in the portal spaces. This purely lymphatic extension of the liver involved only the right lobe. According to Rouvière,[16] and as shown in Figure 11, the lymphatics of the right lobe drain to the pericholedochal lymph nodes, which also receive the lymphatic drainage of the gallbladder. Massive involvement of these lymph nodes may result in retrograde spread of tumor along the lymph vessels to the right lobe of the liver. The lymph drainage of the left lobe (not shown in Fig. 11) is to lymph nodes along the hepatic pedicle in relation to the hepatic artery. These lymph nodes do not communicate with the lymphatics of the gallbladder.[3]

The angiographic findings associated with liver involvement in our series include neovascularity arising from the right hepatic arteries, early filling of hepatic veins, a blush in the right lobe of the liver or a defect in the right lobe of the liver in the hepatogram phase.

Two of the 3 cases with proved hepatic involvement had neovascularity in the right lobe of the liver and early filling of hepatic veins (Cases I and III).

In 3 patients a blush appeared to merge with the right lobe of the liver. In 2 cases (Cases I and III) direct invasion of the liver was present, while in the third (Case II) the tumor, although large, did not invade the liver. In Case IV the carcinoma was first detected upon histologic examination and no invasion of hepatic parenchyma was noted on the angiogram or at operation. This patient had a blush confined to the gallbladder. In Case V a defect was present in the hepatogram medially, which was correlated at operation with hepatic invasion.

Visualization of hepatic veins and of the cholecystic-pericholecystic veins may be explained by the venous drainage of the gallbladder. Cholecystic veins drain both to the hepatic veins via direct communications in the liver,[6] and to the portal vein.[9,13] Two cases showed early hepatic vein visualization. Both cases had large tumor blushes and also involved the right lobe of the liver (Cases I and III). Early hepatic vein visualization has not been previously described with carcinoma of the gallbladder. Enlarged and often early draining cholecystic and pericholecystic veins were present in 4 cases. All 4 were associated with a tumor

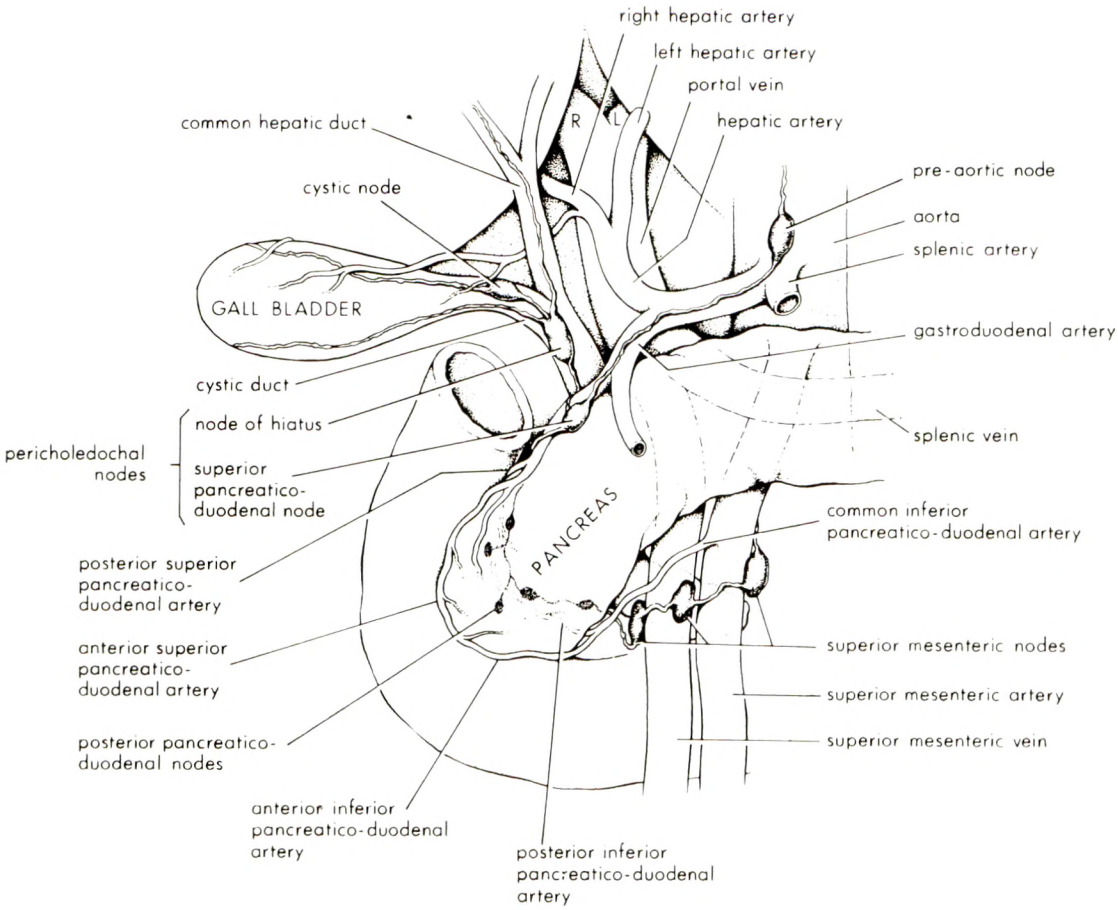


Fig. 11. Illustration of the relationship of the lymphatics draining the gallbladder to the regional arteries and veins.

blush of the gallbladder. Deutsch[5] also noted early draining pericholecystic veins in 1 of his 5 cases of carcinoma of the gallbladder.

The incidence of lymph node metastases with carcinoma of the gallbladder varies from 25 to 80 per cent.[2,6–8,12] This may represent the only type of metastasis in some cases. Fahim *et al.*[6] outlined Clermont's original work[3] which shows the lymph vessels draining the gallbladder following a course in relation to the bile ducts and vascular structures in the lesser omentum and peripancreatic region, ultimately reaching the lymph nodes about the celiac axis and superior mesenteric vessels (also see Fig. 11). The lymph drainage of the gallbladder is first to the cystic lymph node at the left side of the cystic duct and to the lymph nodes adjacent to the common bile duct. These lymph nodes, if large, may affect the hepatic artery and/or the portal vein which run with the common bile duct in the lesser omentum. The superior pancreaticoduodenal lymph node (one of the pericholedochal lymph nodes) is above the superior surface of the head of the pancreas between the first and second portions of the duodenum. It is to the right of the common bile duct and is closely applied to the gastroduodenal artery. From this lymph node the efferent lymphatics drain both to the preaortic lymph nodes and to the posterior pancreaticoduodenal lymph nodes which accompany the posterior pancreaticoduodenal vascular arcade. The latter lymph

nodes in turn drain directly to lymph nodes about the superior mesenteric artery and vein.

Arterial and venous encasement is best understood primarily in relation to the lymphatic drainage of the gallbladder, and secondarily by direct extension of tumor. Portal vein encasement was present in 3 of our 5 cases and in 2 of 7 cases reported by Rösch *et al.*[15] The portal vein may be narrowed in the absence of hepatic artery involvement, but hepatic arterial narrowing was not seen without portal vein involvement. This conforms to the concept that thin-walled veins are more readily affected by a neoplasm than adjacent arteries. The portal vein may be normal even with large tumors, as was the case in 2 of our patients. This may be explained by the facts that: (a) local extension has not reached the porta hepatis and lesser omentum; and (b) the lymph node metastases which are in relation to the common bile duct may be somewhat removed from the portal vein, which is posterior and medial to the duct. By virtue of its closer relationship to the primary tumor and to the proximally involved lymph nodes the portal vein is more readily involved than other major regional veins. However, in Case 11 the superior mesenteric vein was narrowed, while the portal vein was normal. We believe that this constriction of the superior mesenteric vein resulted from involvement of pancreaticoduodenal lymph nodes. This is an area not readily observed at the time of surgery.

Arterial encasement was present in 4 cases (Table 1). The cystic, hepatic, gastroduodenal and pancreaticoduodenal arcade all may show involvement. The superior mesenteric artery may also show encasement, but this was not present in our cases.

SUMMARY

Five cases of carcinoma of the gallbladder studied by selective visceral angiography are reported.

The value of angiography in detecting local extension to the liver and metastases to the regional lymph nodes is stressed.

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REFERENCES

1. Abrams, R. M., Meng, C. H., Firoognia, H., Beranbaum, E. R., and Epstein, H. Y. Angiographic demonstration of carcinoma of gallbladder. *Radiology*, 1970, *94*, 277–282.
2. Burdette, W. J. Carcinoma of gallbladder. *Ann. Surg.*, 1957, *145*, 832–844.
3. Clermont, D. Lymphatiques des voies biliares: Cancer de la vesicule (anatomie pathologique et traitement chirurgical). *Toulouse*, 1909, No. 814, 114.
4. Cooper, W. A. Carcinoma of gallbladder. *Arch. Surg.*, 1937, *35*, 431–448.
5. Deutsch, V. Cholecysto-angiography: visualization of gallbladder by selective celiac and mesenteric angiography. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *101*, 608–616.
6. Fahim, R. B., McDonald, J. R., Richards, J. C., and Ferris, D. O. Carcinoma of gallbladder: study of its modes of spread. *Ann. Surg.*, 1962, *156*, 114–124.
7. Glenn, F., and Hays, D. M. Scope of radical surgery in treatment of malignant tumors of extrahepatic biliary tract. *Surg., Gynec. & Obst.*, 1954, *99*, 529–541.
8. Gradisar, I. A., and Kelly, T. R. Primary carcinoma of gallbladder. *A.M.A. Arch. Surg.*, 1970, *100*, 232–235.
9. Gray's Anatomy of the Human Body. Twenty-seventh edition. Edited by C. M. Goss. Lea and Febiger, Philadelphia, 1959, pp. 765.
10. Illingworth, C. F. W. Carcinoma of gallbladder. *Brit. J. Surg.*, 1935, *23*, 4–18.
11. Kirshbaum, J. D., and Kozoll, D. D. Carcinoma of gallbladder and extrahepatic bile ducts: clinical and pathological study of 117 cases in 13,000 necropsies. *Surg., Gynec. & Obst.*, 1941, *73*, 740–754.
12. Litwin, M. S. Primary carcinoma of gallbladder: review of 78 patients. *A.M.A. Arch. Surg.*, 1967, *95*, 236–240.
13. Michels, N. A. Blood Supply and Anatomy of the Upper Abdominal Organs. J. B. Lippincott Company, Philadelphia, 1955, pp. 361.
14. Ram, M. D. Carcinoma of gallbladder. *Surg., Gynec. & Obst.*, 1971, *132*, 1044–1048.
15. Rösch, J., Grollman, J. H., Jr., and Steckel, R. J. Arteriography in diagnosis of gallbladder disease. *Radiology*, 1969, *92*, 1485–1491.
16. Rouvière, H. Anatomy of the Human Lymphatic System. Edward Brothers, Ann Arbor, 1938, pp. 318.

METASTATIC MELANOMA OF THE GALLBLADDER

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THE differential diagnosis of fixed filling defects in the gallbladder opacified by cholecystography includes adenomyomas, cholesterol polyps, fixed gallstones and adenomas of various histologic types.[2]

This report documents a rare and malignant cause for this finding. Substantiation of the true nature of the lesions was provided by hepatic arteriography.

REPORT OF A CASE

At the age of 53 years, this 59 year old white female had enucleation of the left eye for malignant melanoma of the choroid. She was well for 4 years thereafter. Several months prior to admission she experienced intermittent attacks of dull right upper quadrant pain and noticed a slowly enlarging cervical lymph node.

Physical examination disclosed several enlarged cervical lymph nodes; abdominal examination was negative. Chest roentgenograms and barium examination of the stomach, large and small bowel were negative. Multiple space occupying lesions were present on a liver scan. Biopsy of 1 of the enlarged cervical lymph nodes established the diagnosis of metastatic melanoma.

Oral cholecystography disclosed multiple fixed filling defects measuring 2–15 mm. in diameter (Fig. 1, *A* and *B*). Some of these were oval and others semilunar in shape. Hepatic arteriography disclosed multiple vascular liver metastases. The cystic arteries (main and accessory) supplied faint nodular stains approximately 1 cm. in diameter (Fig. 2). These corresponded to the nodules shown by cholecystography.

The patient was treated with various chemotherapeutic agents; however, she expired 7 months later due to cerebral metastases.

Autopsy disclosed widespread melanoma. Metastatic tumor replaced much of the liver. The gallbladder contained numerous polypoid mucosal melanomatous deposits (Fig. 3).

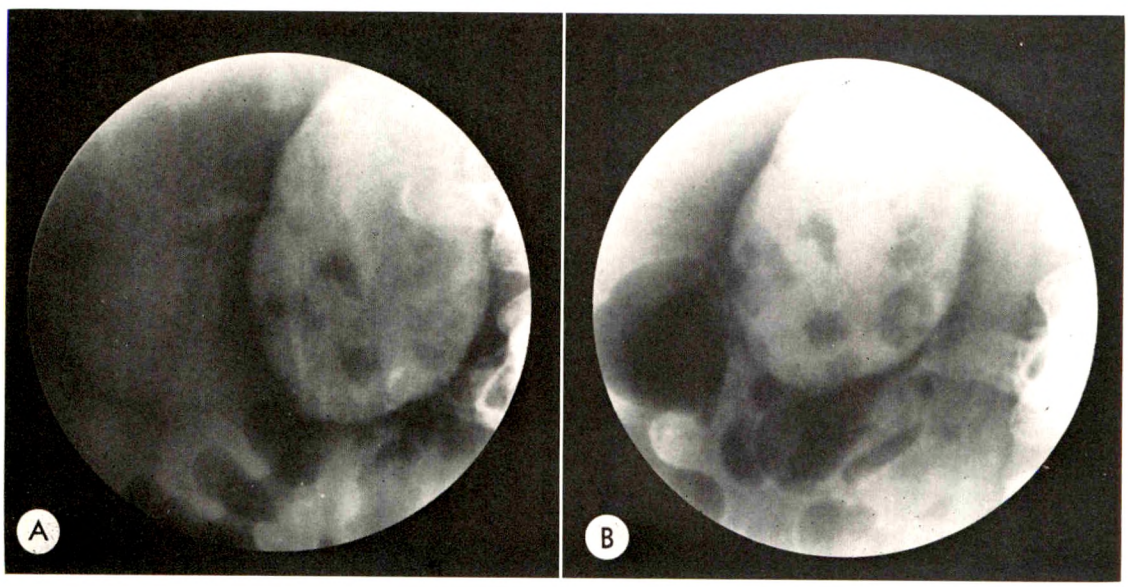


FIG. 1. (*A* and *B*) Oral cholecystograms, anteroposterior and 10° left posterior oblique views. Multiple fixed filling defects of varying shapes and sizes are present.

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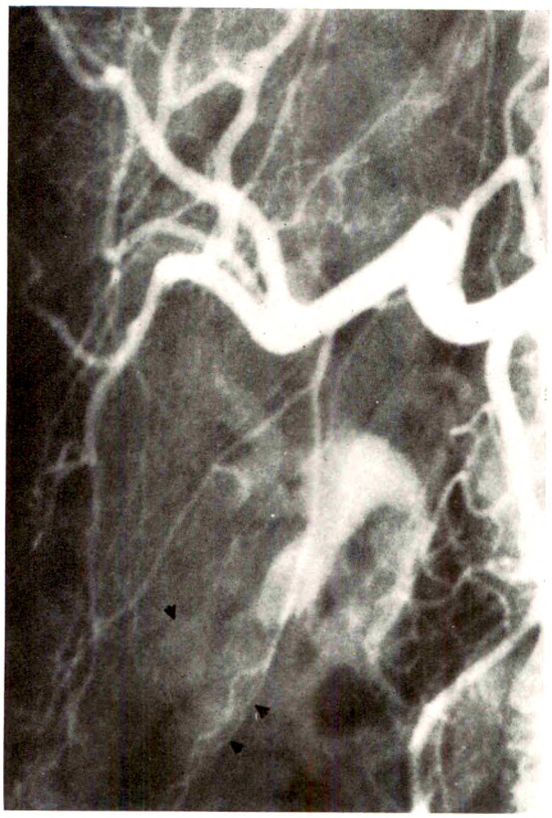


FIG. 2. Hepatic arteriogram. Faint nodular blushes supplied by the cystic arteries (arrows) are visualized.

DISCUSSION

In 1952 Willis[7] found only 21 reported cases of blood borne metastases to the mucosa of the gallbladder; 14 were due to melanoma. In a later autopsy series of 125 cases with metastatic melanoma, Das Gupta and Brasfield noted gallbladder metastases in 19 instances (15 per cent).[1] The most common form of metastatic involvement was the serosal implant. Seven cases had mucosal lesions and in only 2 of these were the lesions of such size to be described as polypoid by gross inspection. Melanomatous involvement of the gallbladder was accompanied by hepatic metastases in 18 of the 19 cases.

We reviewed the 78 cases of metastatic melanoma from our institution autopsied during the last 10 years,[4] and found gallbladder metastases in 11 (14 per cent). In our material mucosal lesions predominated, being present in 9 of 11 cases. In only 1 instance were the lesions limited to the serosa. Gallbladder involvement was accompanied by liver involvement in 10 of the 11 cases.

Appearing first as small, flat subepithelial nodules, mucosal deposits become polypoid and even pedunculated as they enlarge intraluminally. Craterform central ulceration, so characteristic of blood borne intestinal lesions, has been seldom encountered with melanoma of the gallbladder.[1,4,7]

Our case is the third reported with cholecystographic demonstration of metastatic melanoma of the gallbladder.[3,8] All 3 have appeared as multiple fixed filling defects. In each case the individual defects were of



FIG. 3. The gallbladder lumen is exposed. Numerous polypoid melanomatous deposits are shown.

varying size; in each the largest defect was greater than 14 mm. Differentiation from the more common benign causes of fixed filling defects may be suspected because, save for adenomyomas, benign lesions seldom exceed 7 mm.[2] Although experience with gallbladder angiography is limited, no angiographic findings have yet been reported with benign causes of fixed defects;[6] none would be expected with causes such as fixed stones or cholesterol polyps. In our case, the finding of nodular blushes supplied by the cystic artery suggested the malignant nature of the corresponding cholecystographic defects. Further angiographic substantiation was provided by documentation of liver metastases.

Interpretation of the cholecystographic findings demonstrated by this report may present a diagnostic dilemma, especially in a patient with right upper quadrant abdominal symptoms and apparent long-term cure after resection of a primary melanoma.[5] Although no signs or symptoms attributable to gallbladder involvement could be established in any of the 19 cases studied by Das Gupta and Brasfield,[1] the 3 reported cases with cholecystographic documentation of disease have been studied because of symptoms suggesting cholecystitis. If the cholecystogram reveals multiple radiolucent defects, their mucosal or intraluminal nature must be established; *i.e.*, whether they are fixed or freely movable. Herrington's case of metastatic melanoma was subjected to surgery because of interpretation of the cholecystogram as gallstones.[3]

Aside from the patient reported here, 1 other patient of our series of 11 with melanoma of the gallbladder had an oral cholecystography.[4] The patient had no abdominal symptoms or signs and his liver function tests were normal. The gallbladder was only barely opacified after a double dose study using teradax.

SUMMARY

Oral cholecystography in a patient with metastatic melanoma of the gallbladder demonstrated multiple fixed filling defects ranging in size from 2–15 mm. This appearance is similar to the only 2 previously reported cases.

Substantiation of the cholecystographic diagnosis was provided by the arteriographic demonstration of co-existing hepatic metastases and faint contrast staining of some of the gallbladder nodules.

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REFERENCES

1. Das Gupta, T., and Brasfield, R. Metastatic melanoma: clinicopathological study. *Cancer*, 1964, *17*, 1323–1339.
2. Grieco, R. V., Bartone, N. F., and Vasilas, A. Study of fixed filling defects in well opacified gallbladder and their evolution. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1963, *90*, 844–853.
3. Herrington, J. L., Jr. Metastatic malignant melanoma of gall bladder masquerading as cholelithiasis. *Am. J. Surg.*, 1965, *109*, 676–678.
4. Jaffe, E., and Soloway, M. S. Clinico-pathological review of the 78 autopsied cases of metastatic melanoma at the Clinical Center, National Institutes of Health, 1960–1970. Unpublished data.
5. Ronca, P. C. Benign and malignant disease: metastatic malignant melanoma, cholecystitis and cholelithiases. *Am. J. Gastroenterol.*, 1968, *50*, 307–314.
6. Rösch, J., Grollman, J. H., and Steckel, R. J. Arteriography in diagnosis of gall bladder disease. *Radiology*, 1969, *92*, 1485–1491.
7. Willis, R. A. The Spread of Tumors in the Human Body. Second edition. Butterworth & Co., Ltd., London, 1952, pp. 447.
8. Zemlyn, S. Metastatic melanoma of gallbladder. *Radiology*, 1966, *87*, 744–745.

UNFAMILIAR ROENTGEN FINDINGS IN PANCREATIC DISEASE*

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ACCORDING to the most recent statistics compiled by the American Cancer Society,[20] pancreatic cancer has now become the second most common malignancy of the gastrointestinal tract. It still ranks far below tumors of the colorectum, but its estimated incidence exceeds that for cancer of the esophagus, hepatobiliary system, and stomach. The necessity for radiologists to be thoroughly familiar with all the manifestations of pancreatic disease is, therefore, even more pressing than it has been in the past.

Since barium gastrointestinal examination is the initial radiologic procedure encountered by most patients ultimately found to have pancreatic cancer, it is fortunate that numerous signs demonstrable by this technique have been described. For example, almost any radiologist who identifies the "inverted '3' " sign of Frostberg is immediately alerted to the presence of pancreatic disease. This finding and certain others such as forward displacement of the stomach and a mass impression on the inner border of the descending duodenum are well known and specifically searched for by competent radiologists when examining patients clinically suspected of having pancreatic disease. There are, however, a number of other helpful roentgen signs of pancreatic pathology which are unfamiliar, misunderstood, or at least generally underutilized. Among these are: distorted duodenal diverticula; duodenal contour abnormalities consistent with gallbladder or common bile duct enlargement; abnormalities of the duodenojejunal junction; enlargement of the papilla of Vater; barium reflux into the common bile duct and/or pancreatic duct; and localized duodenal spasm.

The purpose of this paper is to emphasize the nature and importance of several of these unfamiliar radiologic indications of pancreatic pathology.

MATERIAL AND METHOD

The findings to be considered in this report deal with the appearance of the duodenum as demonstrated by barium meal examination. Their appreciation has been previously limited by a lack of information concerning certain anatomic features of the normal duodenum. In order to provide pertinent data on this subject 300 upper gastrointestinal barium studies were analyzed. Patients in whom there was clinical suspicion of biliary tract obstruction were excluded. Since early symptoms are so diffuse, pancreatic disease was at least a theoretic possibility in all these patients, but in any instance where such pathology was seriously entertained the examination was excluded from the series. Any patient in whom an elevated serum amylase or bilirubin had been detected was also excluded. Subject to these restrictions consecutive examinations were evaluated until a total of 300 had been accumulated. The average age of the patients was 47 years; there were 196 males and 104 females. In this series, however, only 19 duodenal diverticula were observed. Since this sample

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was judged too small, the next 31 duodenal diverticula encountered were analyzed separately. These 31 cases were otherwise completely unselected.

The observations recorded included the position of the duodenojejunal junction in relation to the spine and to the superior duodenal flexure (only anteroposterior roentgenograms were used for these particular determinations). When the duodenal papilla could be seen as a discrete filling defect, its size was determined. The presence of indentations suggestive of gallbladder or common bile duct enlargement was specifically searched for in each instance. Finally each diverticulum was assessed to determine its constancy of appearance (size and general contour, etc.) and also its orientation (*i.e.*, its angulation with reference to the main duodenal axis).

RESULTS

DUODENAL DIVERTICULA

The angle formed by the intersection of the central axis of the diverticulum with the long axis of the duodenum at its base varied between 29° and 122° (mean 76°). There was considerable observer disagreement about constancy of diverticular configuration, but all judges concurred that in at least some instances the appearance of a given diverticulum was essentially unchanged from roentgenogram to roentgenogram. Forty-six of the diverticula were rounded in contour, 1 was asymmetrically indented by a second diverticulum adjacent to it, and 3 were small perivaterian diverticula with flattened or even indented-appearing margins.

CONTOUR APPEARANCES CONSISTENT WITH GALLBLADDER OR COMMON BILE DUCT DILATATION

A smooth, rounded indentation on the outer margin of the upper descending duodenum typical of gallbladder enlargement was seen in only 1 of the 300 cases studied. A sharply defined, vertically oriented tubular indentation on the posterior aspect of the duodenal bulb or immediate postbulbar region suggests common bile duct enlargement. A smooth, rounded indentation on the superior aspect of the first portion of the duodenum can represent either enlargement of the neck of the gallbladder or common duct dilatation. One or both of these findings were present in 35 (12 per cent) of our 300 "normal" examinations.

DUODENAL PAPILLAE

An indentation defect consistent in appearance with the papilla of Vater was identified in 59 patients (20 per cent). The greatest dimension of these presumptive papillae ranged from 0.6 to 1.9 cm. (mean 0.9 cm.); in 3 instances (1 per cent) the greatest dimension exceeded 1.5 cm.

POSITION OF THE DUODENOJEJUNAL JUNCTION

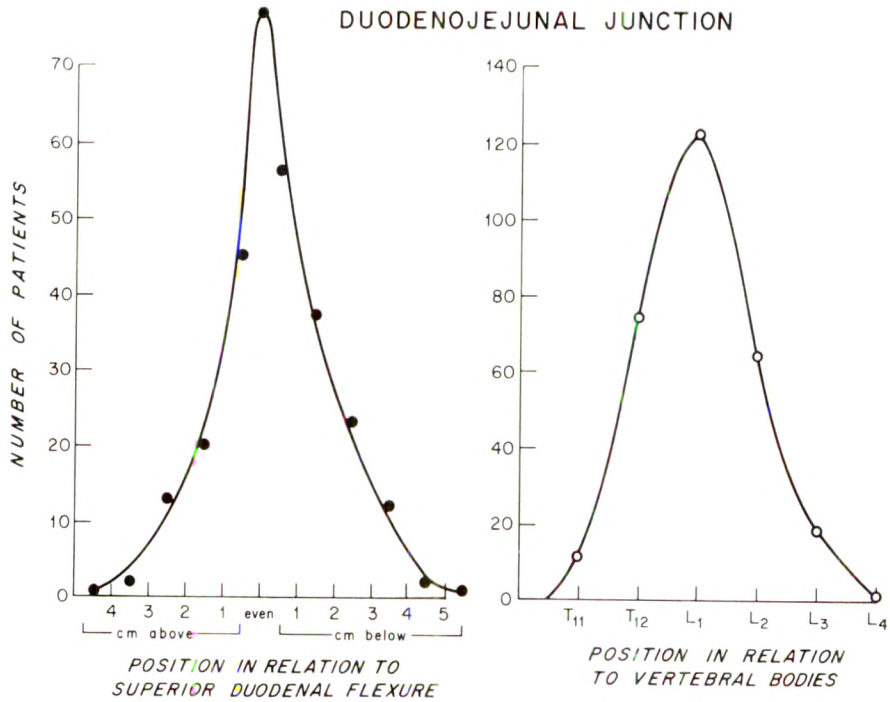
The position of the duodenojejunal flexure could be determined in 289 of the 300 patients. It varied widely, being found at any level from D 11 to L 4, and from 4 cm. above to 5 cm. below the level of the superior duodenal flexure (Graph 1). In 20 of the 289 patients the duodenojejunal junction was below the L 2–3 interspace.

DISCUSSION

DEFORMED DUODENAL DIVERTICULA

Distortion of a duodenal diverticulum as a result of pancreatic cancer has been sporadically reported.[1,14] To the authors' knowledge all cases illustrated have resulted from neoplastic disease, but theoretically, at least, chronic pancreatitis or pancreatic pseudocyst could produce a similar effect. In any event diverticular distortion, while an uncommon occurrence, has been amply validated as an indication of pancreatic pathology. Accordingly, when duodenal diverticula are present in a patient clinically suspected of having pancreatic disease, their appearance merits careful scrutiny.

Rotation, displacement, flattening, and fixation are the abnormalities most commonly mentioned as suggestive of underlying pancreatic pathology. In the individual case, however, determining whether or not these findings are present



Graph I.

can be difficult, particularly since the normal appearance of duodenal diverticula has seldom been described.

The results of our study indicate that rotation or displacement alone are completely unreliable indications of abnormality. The angulation of normal diverticula varies widely. Similarly, a constant or fixed appearance cannot be considered abnormal since some duodenal diverticula change little in appearance from roentgenogram to roentgenogram. The characteristic of duodenal diverticula most likely to be of aid in identifying adjacent pancreatic pathology is their shape. Since the convincingly abnormal diverticula illustrated in the literature have all shown flattening or some other contour distortion (Fig. 1), it is this parameter which should be most carefully assessed when pancreatic disease is a possibility. Small perivaterian diverticula represent an exception; these structures commonly have flattened or even concave sides suggesting extrinsic indentation (Fig. 2). Accordingly, unless gross irregularity can be identified their usefulness for diagnosis of pancreatic disease is minimal.

Indentations on the Duodenum Produced by the Biliary Tree

The superior duodenal flexure is partially surrounded by the gallbladder anterolaterally, the cystic and hepatic ducts superiorly, and the common bile duct posteriorly (Fig. 3). Because of this close proximity and because of the frequency with which pathologic processes involving the head of the pancreas produce secondary changes in the biliary tree, the relationship between these structures should be evaluated on any barium study where pancreaticoduodenal disease is a possibility and especially in patients with jaundice.

When a portion of the extrahepatic biliary system is dilated, a characteristic indentation on the barium-filled duodenum is commonly produced. Unfortunately, somewhat similar false indentations occur in certain patients where no biliary tract pathology is present. This fact complicates evaluation of the duodenum on barium

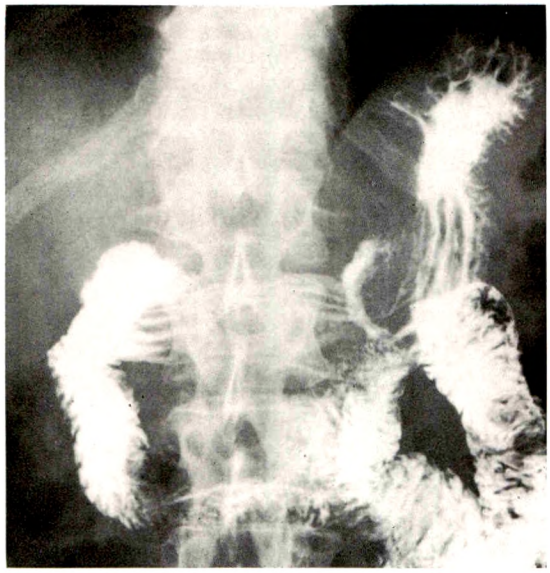


FIG. 1. Diverticulum arising from the fourth portion of duodenum and distorted by cancer of the body of pancreas. (Courtesy of Justin L. Williams, M.D., San Francisco.)

studies, especially since no figures concerning the incidence of such false positive findings have been reported.

Dilatation of the gallbladder typically produces a smooth, rounded indentation

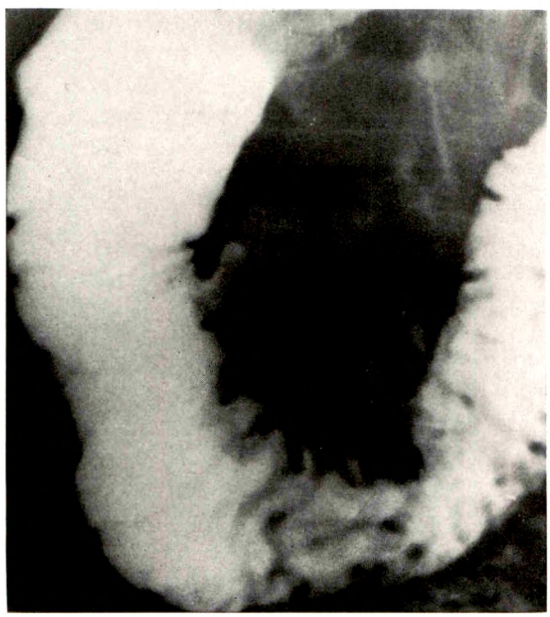


FIG. 2. Small perivaterian diverticulum. Note slight concavity of inferomedial aspect. No pancreatic disease was suspected.

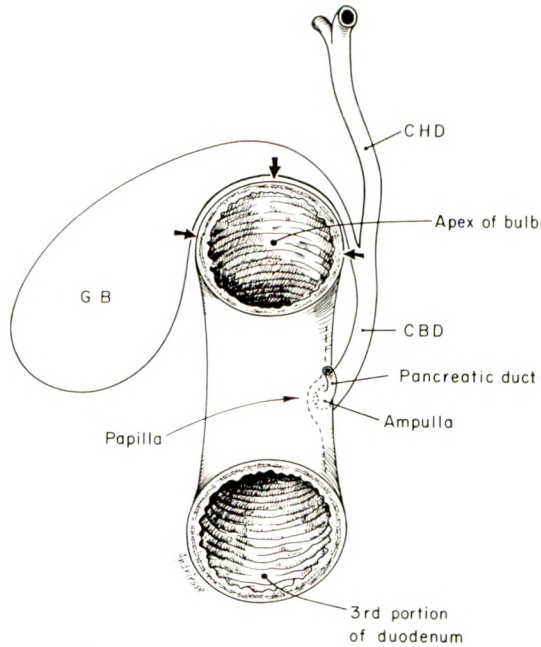


FIG. 3. Relationships between duodenum and biliary tree as seen in thick sagittal section. GB = gallbladder; CHD = common hepatic ducts; CBD = common biliary duct.

on the outer aspect of the upper descending duodenum (Fig. 4). When present, this is a very reliable sign. In Eyler's series 71 per cent (27 of 38) of enlarged gallbladders

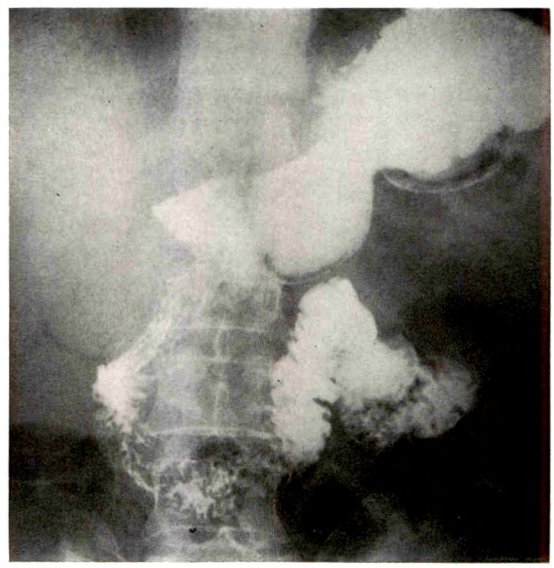


FIG. 4. Courvoisier gallbladder indenting the outer aspect of the duodenum in a patient with cancer of the ampulla of Vater.

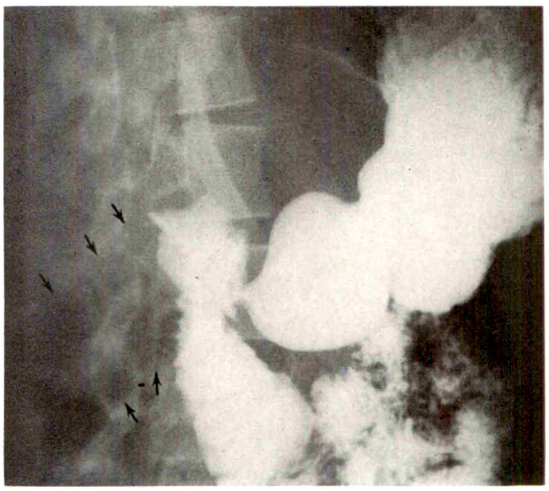


Fig. 5. Duodenocolic apposition—indentation on the outer duodenal border produced by the hepatic flexure of the colon. (Arrows indicate gas in colon.)

were demonstrated by barium meal examination and there were no false positives.[6]

A smooth indentation defect on the outer duodenal border may be produced by several different pathologic conditions. In the presence of obstructive jaundice a Courvoisier gallbladder is the likeliest cause. In tumors of the papilla of Vater and/or head of the pancreas, gallbladder enlargement is present in from 82 to 87 per cent at surgery, although the gallbladder is palpable in only 38 to 67 per cent.[9] In the absence of jaundice, empyema and hydrops of the gallbladder are possibilities, as are masses arising from the right kidney and liver. A normal condition which can produce indentation of the lateral duodenal border is duodenocolic apposition; this caused the one such finding in the 300 patients evaluated in this report (Fig. 5). Poppel[16] originally described the roentgen features of this condition which results from unusually close approximation of the hepatic flexure and duodenum. He felt that the colon indents the lateral duodenal margin in as many as 3 per cent of all examinations, but in the current series only 1 convincing instance occurred.

An enlarged common bile duct can indent the immediate postbulbar portion of the duodenum. The value of this observation has been stressed by several authors as a sign of extrahepatic biliary obstruction;[4,10] in 1 series of 27 patients with resectable tumors of the pancreaticoduodenal region this was the second most frequently noted sign, being radiologically demonstrable in 12 of the 21 cases where the common duct was found to be dilated at surgery.[15]

From a descriptive standpoint there are 2 components to this radiologic sign. First, there may be a rather sharply defined, vertically oriented tubular indentation on the posterior aspect of the apex of the duodenal bulb or on the immediate postbulbar region (Fig. 6). Secondly, there may be a smooth, rounded indentation on the superior aspect of the duodenum in the same area (Fig. 7). Most published examples of this finding incorporate both components to a greater or lesser degree.

In 35 of 300 patients without clinical evidence of biliary abnormality one or both of these components were noted. Angulation at the superior duodenal flexure, peristaltic activity, incomplete filling, the

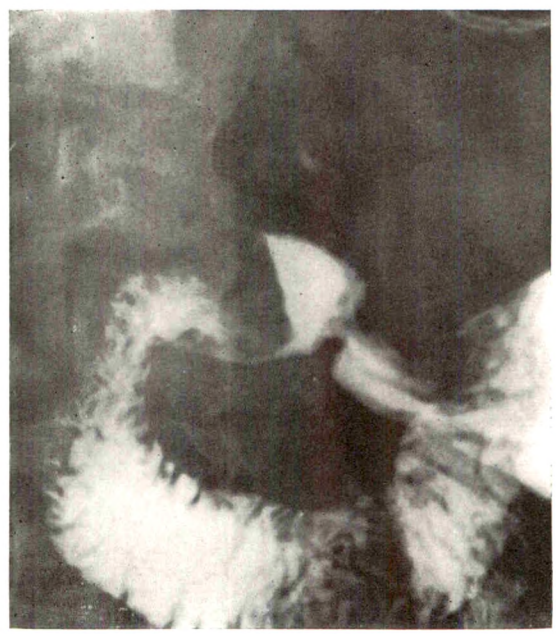


Fig. 6. Dilatation of the common bile duct produces indentation on the apex of the duodenal bulb. Choledocholithiasis was the underlying cause.

cystogastrocolic ligament (a broad band-like peritoneal reflection present in a minority of the population)[18] and a nondistended, nonpathologic gallbladder are among the various causes which can simulate common duct and/or gallbladder enlargement. Furthermore there is an extensive diagnostic differential to be considered when these signs are present. Duodenal varices, an enlarged quadrate lobe of the liver, a dilated hepatic artery, masses in the porta hepatis, and duodenal ulcer disease are all possibilities.

The 12 per cent incidence of presumptive false positives and the broad differential requiring consideration abate the confidence with which a radiographic diagnosis of common bile duct enlargement can be based on barium gastrointestinal examination. On the other hand, when findings suggestive of a dilated common duct are noted in patients with jaundice they should not be disregarded. They may best be considered as an indication for more refined radiologic examination by angiography, isotopic imaging, or percutaneous cholangiography.

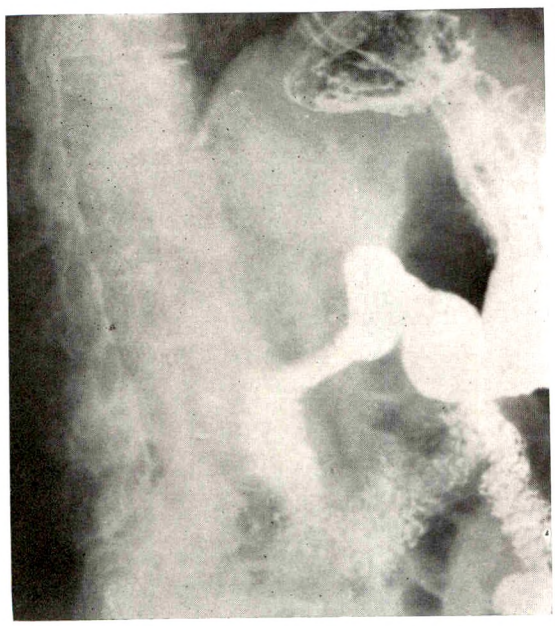


Fig. 7. Hydrops of gallbladder (resulting from impaction of calculus in the neck of the gallbladder) produces indentation on the superior aspect of the duodenal bulb.



Fig. 8. Acute pancreatitis with enlargement of the papilla of Vater (Poppel's sign). Note associated edema of duodenal folds. (Courtesy of James G. Moore, M.D., and Lincoln Lum, M.D., Daly City, California.)

Enlargement of the Papilla of Vater

Although the duodenal papilla is an intrinsic component of the duodenum itself, enlargement or undue prominence of the structure most often reflects either pancreatic or biliary tract pathology. Edema of the papilla resulting from choledocholithiasis or from acute pancreatitis (Poppel's sign) (Fig. 8) is a well known phenomenon, although differentiation from perivaterian neoplasm may be difficult on strictly roentgenographic grounds. While most radiologists are alerted to the significance of papillary enlargement, there is a lack of agreement about what measurements should be considered the upper limits for a normal papilla. Poppel and his

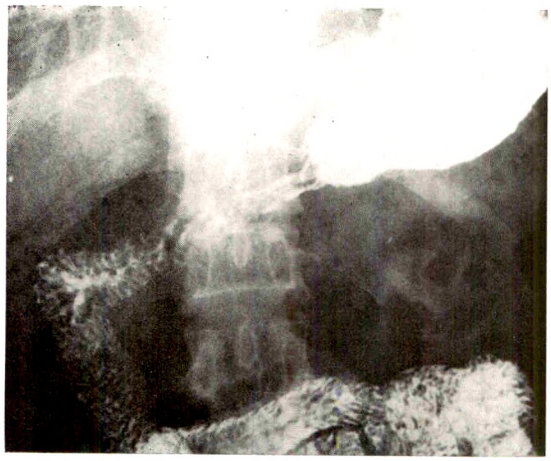


FIG. 9. Low duodenojejunal junction in patient subsequently explored (for cholelithiasis) and found to have no evidence of pancreatic pathology. Note the normal duodenal folds.

co-workers roentgenographed autopsy specimens and demonstrated that papillae up to 3 cm. in greatest dimension could be normal.[17] More recently, however, several reports[3,8] have suggested that a papilla greater than 1.5 cm. in size (as demonstrated by barium examination in living patients) should be viewed with considerable suspicion.

The results obtained from our series of 300 normal patients suggest that in only about 1 per cent of apparently normal patients can a duodenal papilla greater than 1.5 cm. in size be found. Accordingly, when large papillae are shown by barium meal examination, it seems reasonable to consider them pathologic until proven otherwise.

Differential diagnosis for papillary enlargement must include numerous conditions. Both an impacted distal common duct calculus[5] and acute pancreatitis are frequent causes in the authors' experience. In the latter condition, associated duodenal changes such as fold edema and atony are often present together with enlargement of the papilla, and these features help to distinguish pancreatitis from choledocholithiasis (Fig. 8). Perivaterian malignancy can produce a smooth, rounded enlargement of the duodenal papilla similar to that seen in benign conditions. This can result either from the tumor mass itself or from malignant lymphatic obstruction with secondary papillary edema. Benign perivaterian tumors including ectopic pancreas can also simulate enlargement of the papilla. Finally, papillary enlargement in association with acute duodenal ulceration has been described. This has been an unusual cause in the authors' experience; when present, a bulbar ulcer crater has invariably been shown as well. This long list of radiologic possibilities dictates that correlation with clinical manifestations is necessary for accurate diagnosis, but duodenal papillae greater than 1.5 cm. in maximum dimension should be considered radiologically abnormal and worthy of further investigation.

APPEARANCE OF THE DUODENOJEJUNAL FLEXURE

Depression of the duodenojejunal junction (*i.e.*, at the ligament of Treitz) has long been considered a sign of mass lesions in the body or tail of the pancreas. The radiologist attempting to make use of this sign in clinical practice, however, must decide just what constitutes significant depression. In one study[19] the investigators considered duodenojejunal junctions below the L 2-3 interspace to be depressed; in 200 consecutive upper gastrointestinal barium examinations the junction was below this level in only 1 instance, except in an unspecified number of patients with "very ptotic stomachs."

The observations resulting from analysis of our 300 patient series, however, suggest that "depression" of the ligament of Treitz is a treacherous indicator of pancreatic disease since the normal location of the area is so variable (Fig. 9). On the other hand, these results by no means diminish the requirement for careful evaluation of the distal duodenum in patients where a lesion of the body or tail of the pancreas is clinically suspected. The significant abnormalities, however, are not positional, but rather structural such as indentation, mucosal splaying, elongation, and fixation of folds

or frank ulceration. Such changes can be noted in up to 75 per cent of patients with cancer of the body or tail of the pancreas.[13] Similar changes (except for ulceration) can occur in pancreatitis or pancreatic pseudocyst, although their frequency is less well documented. Such abnormalities vary from the obvious to the subtle (Fig. 10), and accordingly, when questionable changes are demonstrated on conventional barium examination, follow-up study by hypotonic duodenography to confirm or exclude their presence seems logical.

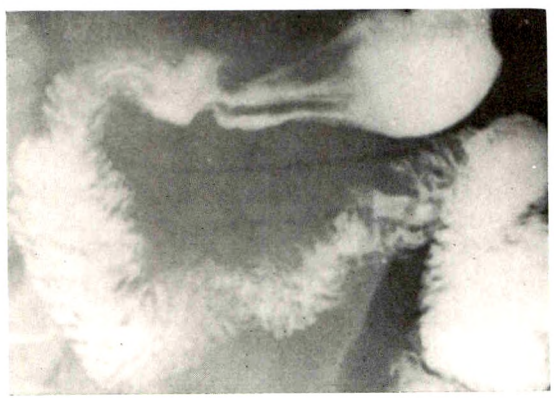


Fig. 10. Mucosal changes along inner border of the third portion of the duodenum produced by carcinoma of the body of pancreas. Both spiculation and ulceration are present.

BARIUM REFLUX INTO THE COMMON BILE DUCT

Filling of the common bile duct and/or main pancreatic duct during the course of barium duodenal examination is a spectacular roentgen finding[7] (Fig. 11). This is not infrequent when there has been a previous sphincterotomy and, of course, spontaneous (or surgical) fistulae between the biliary and gastrointestinal tracts must be excluded. During the past 3 years, however, the authors have observed this phenomenon on 5 occasions in intact patients. Of these, 2 had pancreatitis, 2 had pancreatic cancer, and in 1 the common duct entered a duodenal diverticulum. Strongyloidiasis,[12] regional enteritis,[11] and recent passage of a common duct stone are also occasionally associated with this finding, which is by no means, therefore, specific; however, when

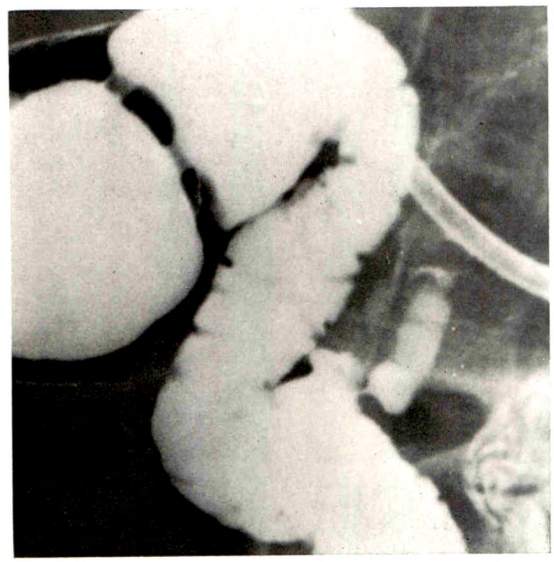


Fig. 11. Spontaneous reflux of barium into the pancreatic duct during hypotonic duodenography. There was no previous surgery; reflux resulted from pancreatitis.

barium fills the common duct of an intact patient during upper gastrointestinal examination, some form of pancreaticoduodenal pathology can be confidently predicted.

Such reflux is said to occur in normal patients being treated with anticholinergics due to relaxation of the sphincter of Oddi, but the authors have not noted it during the course of more than 300 hypotonic duodenographies.

LOCALIZED DUODENAL SPASM

One previously undescribed roentgenologic sign has been noted by the authors in 7 patients with pancreatic cancer. The finding must be demonstrated by hypotonic duodenography and consists of a localized area of persistent duodenal contraction in a duodenum which is otherwise flaccidly distended. The constricted area is variable in appearance (*i.e.*, not constant or fixed), suggesting that it results from excessive local irritability rather than from tumor invasion. The outer aspect of the duodenum has been involved in all cases; sometimes the appearance has resembled an incisura similar to that observed in the

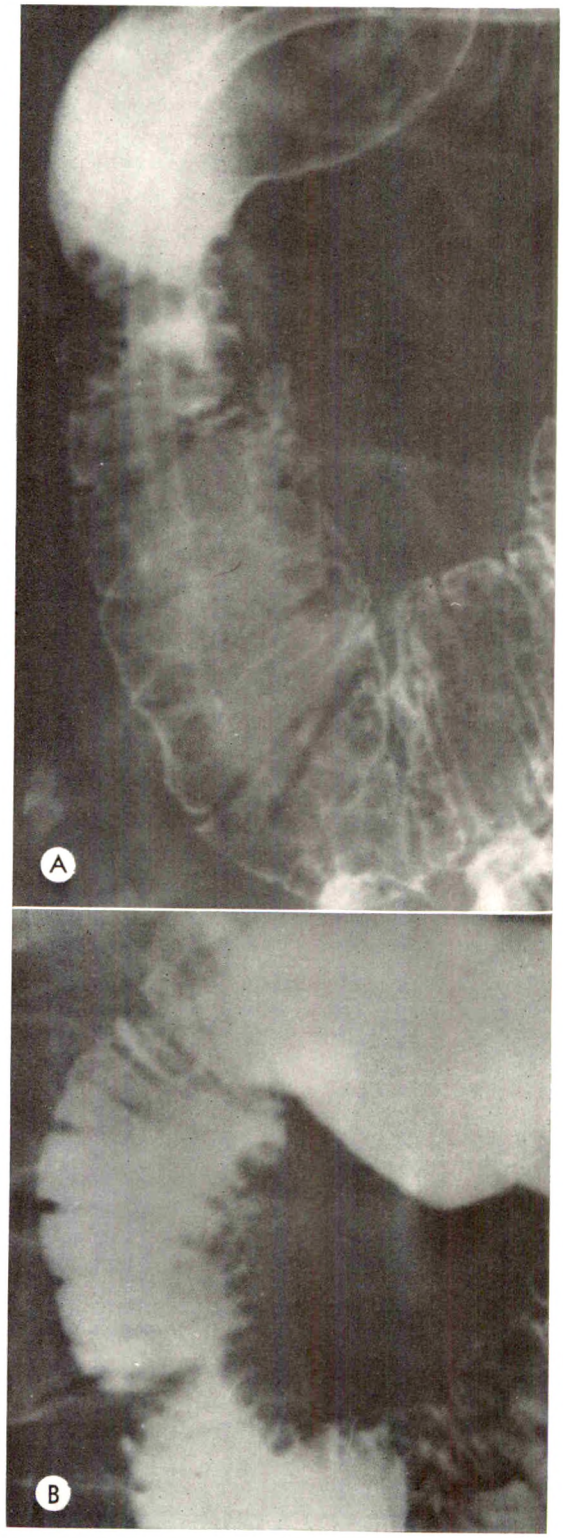


FIG. 12. (*A* and *B*) Hypotonic duodenography showing localized duodenal spasm in 2 patients with pancreaticoduodenal cancer. Note incisura-like appearance in *B*.

stomach opposite an active ulcer crater (Fig. 12, *A* and *B*). The inner border at the level of the constriction may appear frankly malignant, nonspecifically abnormal, or even essentially normal. The authors theorize that this segmental failure of relaxation results from focal neuromuscular irritability arising from the relatively localized zone of inflammation which surrounds most pancreatic cancers. If this be so, then the sign should be seen more commonly in malignant disease since in pancreatitis (without associated neoplasia) inflammation usually involves the entire organ. When focal pancreatitis occurs, however, a region of localized duodenal constriction may be demonstrable.[2]

SUMMARY

Three hundred normal upper gastrointestinal examinations as well as 50 studies demonstrating duodenal diverticula were analyzed to better characterize the aspects of normal duodenal anatomy pertinent to diagnosis of pancreatic disease.

The results are applied to distorted duodenal diverticula, depression of the duodenojejunal junction, duodenal indentations resulting from gallbladder or common bile duct enlargement and enlargement of the duodenal papilla.

Common misconceptions concerning these signs are clarified.

In addition barium reflux into the common bile duct and localized duodenal spasm on hypotonic duodenography are discussed.

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REFERENCES

1. Beranbaum, S. L. Carcinoma of pancreas: bidirectional roentgen approach. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1966, *96*, 447–467.
2. Bilbao, M. K., Frische, L. H., Dotter, C. T., and Rösch, J. Hypotonic duodenography. *Radiology*, 1967, *89*, 438–443.
3. Bilbao, M. K., Rösch, J., Frische, L. H., and Dotter, C. T. Hypotonic duodenography in

diagnosis of pancreatic disease. *Seminars Roentgenol.*, 1968, *3*, 280–287.

4. BROWN, S., and HARPER, F. G. New roentgen sign in extrahepatic biliary tract disease. *Radiology*, 1946, *47*, 239–248.
5. EATON, S. B., FERRUCCI, J. T., BENEDICT, K. T., and MARGULIS, A. R. Diagnosis of choledocholithiasis by barium duodenal examination. *Radiology*, 1972, *102*, 267–273.
6. EYLER, W. R. Evaluation of roentgen signs of pancreatic enlargement. *J.A.M.A.*, 1962, *181*, 967–971.
7. FAUST, D. B., and MUDGETT, C. S. Visualization of biliary tree with air and barium following barium meal. *Ann. Int. Med.*, 1943, *19*, 356–367.
8. FERRUCCI, J. T., BENEDICT, K. T., PAGE, D. L., FLEISCHLI, D. J., and EATON, S. B. Radiographic features of normal hypotonic duodenogram. *Radiology*, 1970, *96*, 401–408.
9. HESS, W. Surgery of the Biliary Passages and the Pancreas. D. Van Nostrand Company, Inc., Princeton, N. J., 1965.
10. HODES, P. J., PENDERGRASS, E. P., and WINSTON, N. J. Pancreatic, ductal, and vaterian neoplasms: their roentgen manifestations. *Radiology*, 1954, *62*, 1–15.
11. LEGGE, D. A., CARLSON, H. C., and HOFFMAN, H. N. Roentgenologic sign of regional enteritis of duodenum. *Radiology*, 1971, *100*, 37–39.
12. LOUISY, C. L., and BARTON, C. J. Radiological diagnosis of *Strongyloides stercoralis* enteritis. *Radiology*, 1971, *98*, 535–541.
13. MANI, J. R., ZBORALSKE, F. F., and MARGULIS, A. R. Carcinoma of body and tail of pancreas. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *96*, 429–446.
14. OCHSNER, H. C. Roentgenologic diagnosis of periduodenal and extraduodenal lesions. AM. J. ROENTGENOL. & RAD. THERAPY, 1941, *45*, 718–723.
15. PEREZ, C. A., POWERS, W. E., HOLTZ, S., and SPJUT, H. S. Roentgenologic-pathologic correlation of resectable carcinoma of pancreatico-duodenal region. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1965, *94*, 438–448.
16. POPPEL, M. H. Duodenocolic apposition. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1960, *83*, 851–856.
17. POPPEL, M. H., JACOBSON, H. G., and SMITH, R. W. The Roentgen Aspects of the Papilla and Ampulla of Vater. Charles C Thomas, Publisher, Springfield, Ill., 1953, pp. 96.
18. SAMUEL, E. Cystogastrocolic band: radiologic considerations. *Brit. J. Radiol.*, 1952, *25*, 19–21.
19. SIFRE, R. A., STENBERG, E. S., and GRAYBILL, L. D. Roentgen configuration of duodenojejunal area in carcinoma of pancreas. *Geriatrics*, 1953, *8*, 486–493.
20. SILVERBERG, E., and HOLLEB, A. I. Cancer statistics, 1971. *Ca*, 1971, *21*, 13–31.

SONOGRAPHIC DIAGNOSIS OF PANCREATIC LESIONS*

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DISEASE processes involving the pancreas continue to be difficult to localize and diagnose. A retrospective review of upper gastrointestinal series in patients with proven carcinoma of the pancreas or pancreatitis yielded a diagnostic accuracy of about 50 per cent.[4] Frequently, numerous repeat barium studies are performed on the same patient with discouraging results. Pancreatic arteriography usually requires superselective techniques and carries some degree of patient risk. The diagnostic accuracy varies from 70 to 90 per cent depending on whether false positives are included in the statistics.[1,10] Isotope studies of the pancreas are reported to be accurate in 90 per cent of normal studies.[8,11] However, if the pancreatic isotope scan is abnormal, its diagnostic accuracy falls to 50 per cent.

The value of diagnostic ultrasound has received scant attention in review articles dealing with the diagnosis of pancreatic lesions.[2] A few references have appeared in the recent literature emphasizing the usefulness of this modality.[3,5,7]

Our experience with this technique has been very rewarding. In some cases diagnostic ultrasound has correctly diagnosed the pancreatic abnormality when all other studies have been normal or inconclusive. In other patients valuable supportive evidence has been accumulated, and the size and extent of the process has been documented.

MATERIAL AND METHOD

In reviewing 250 sonograms obtained in the last 8 months, 31 cases were selected in which the presence or absence of pancreatic disease was diagnosed by ultrasound techniques. Clinical records, roentgenographic studies and operative reports were correlated with sonographic studies. Unfortunately, not all of the patients underwent abdominal exploration; however, all the pancreatic carcinomas, 2 cases of pancreatitis, and 3 cases of pseudocyst were verified by pathologic studies. Interpretation of the sonograms was made independently by 2 of us.

Compound contact scanning with an ultrasonic laminagram was accomplished in all cases. A 2.5 MHz transducer and B-mode display were used. Images built up on a persistence scope were photographed with a Polaroid camera to obtain a permanent record.[13]

An abdominal roentgenogram is obtained prior to the sonogram to confirm the absence of barium in the gastrointestinal tract. This also indicates the amount of bowel gas present. Sufficient quantities of gas or barium can prevent through transmission of the ultrasonic beam.[9] Lead markers are placed on the umbilicus and xiphoid process prior to roentgenography. This occasionally provides useful correlative information when a mass is identified on the sonogram. The studies are performed with the patient in the supine position. Transverse and longitudinal scans are made routinely at 2 cm. intervals. A diagonal scan extending from the right mid-abdomen towards the left upper quadrant oriented to the suspected anatomic position of the pancreas is also usually made.

REPORT OF CASES

A. CARCINOMA OF THE PANCREAS

CASE 1. This patient had a parathyroid

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adenoma removed 4 months prior to his admission to the Kansas University Medical Center (K.U.M.C.). He continued to lose weight following the procedure. Two upper gastrointestinal series were interpreted as normal. A pancreatic scan was unremarkable. The sonogram showed a large mass in the region of the tail of the pancreas (Fig. 1). At surgery a large tumor originating from the body and tail of the pancreas was discovered.

Case II. A 64 year old white male entered the K.U.M.C. for evaluation of weight loss. Six months previously an aortic aneurysm had been resected at another hospital. A complete evaluation at the original hospital failed to disclose an etiology for the weight loss. An upper gastrointestinal series at K.U.M.C. was normal. An intravenous pyelogram suggested the possibility of a mass in the left kidney. A prone transverse sonogram of the left kidney showed no evidence of a renal mass. Supine transverse scans identified a large mass in the pancreas (Fig. 2). Subsequently, a pancreatic isotope scan was positive. Surgical exploration confirmed the presence of carcinoma of the pancreas.

Case III. This patient was admitted to K.U.M.C. with obstructive jaundice. An upper gastrointestinal series revealed a malignant antral ulcer. A sonogram disclosed a large pancreatic mass and a dilated gallbladder (Fig. 3, *A* and *B*). At surgery a large carcinoma of the head of the pancreas obstructing the common duct and extending into the antrum of the stomach was found.

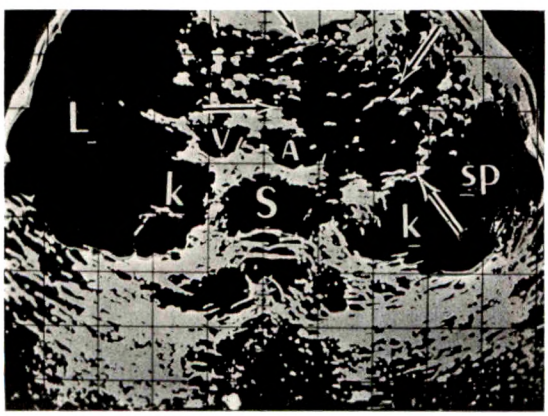


Fig. 1. Case I. Carcinoma involving the body and tail of the pancreas (arrows). Other normal structures are: (k) kidneys, (L) liver, (S) spine, (sp) spleen, (v) vena cava, (a) aorta.

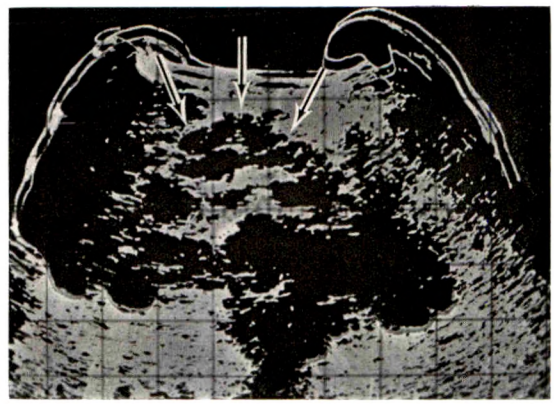


Fig. 2. Case II. Carcinoma involving the head and body of the pancreas (arrows). Note the internal echoes at a routine scanning sensitivity.

Comment. Seven of the 8 carcinomas of the pancreas have had similar ultrasonic characteristics. The only variation has been size. The lesions have irregular borders which are often lobulated. Internal echoes are usually noted at routine scanning sensitivities. As the transducer sensitivity is increased the internal echoes become more pronounced and irregular. These echoes may be due to necrosis within the tumor or to residual normal pancreatic tissue. Sometimes there is an ill-defined component to the tumor with a different echo structure characterized by very indistinct borders. This portion of the tumor may be mistaken for normal tissue when it actually represents infiltration of adjacent structures.

Most of the carcinomas have been relatively large at the time of diagnosis. However, since enlarged retroperitoneal lymph nodes can be detected, it would appear that tumors measuring 2 or 3 cm. can be identified by diagnostic ultrasound.

B. Pancreatitis

Case IV. This patient was admitted to the psychiatric service at the K.U.M.C. because of recurrent abdominal pain. An upper gastrointestinal series showed a retrogastric mass. A sonogram revealed pancreatitis and cholelithiasis (Fig. 4, *A* and *B*). A selective celiac

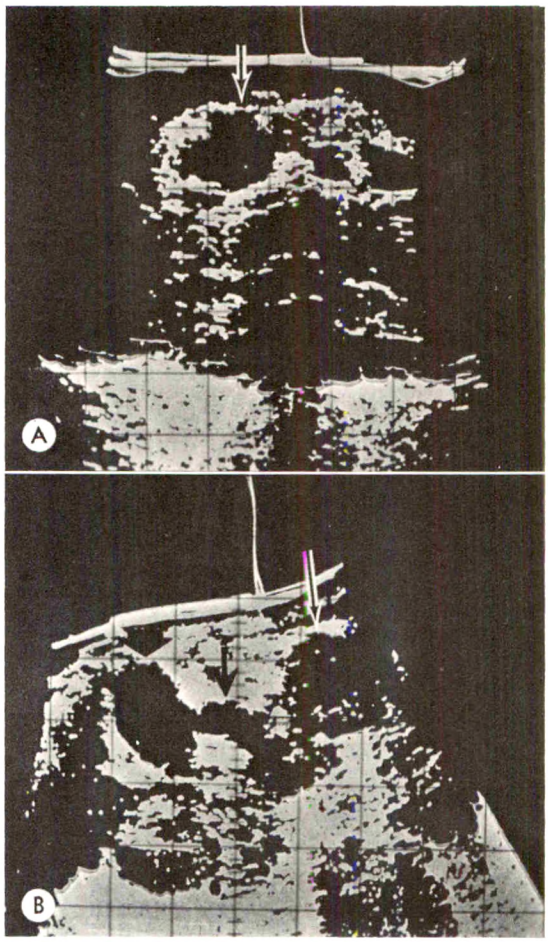


Fig. 3. Case III. Carcinoma of the pancreas. (*A*) Mass in the head of the pancreas demonstrating a grossly irregular inner border (arrow). (*B*) Oblique subcostal view showing the carcinoma of the head of the pancreas (arrow), distended gallbladder (black arrowhead), and dilated common duct (black arrow).

arteriogram was normal. At surgery pancreatitis and cholelithiasis were confirmed.

Case V. A 45 year old female entered K.U.M.C. for evaluation of recurring episodes of abdominal pain. An upper gastrointestinal series showed abnormalities in the descending duodenum. A sonogram demonstrated the changes of pancreatitis (Fig. 5).

Case VI. This female patient presented with symptoms of cholelithiasis. Because of repeated elevated amylases, pancreatitis was suspected. An upper gastrointestinal series and pancreatic isotope scan were normal. Sonography was compatible with pancreatitis (Fig. 6).

Comment. Pancreatitis produces an elongated, transonic area anterior to the aorta and vena cava at normal scanning sensitivities. The image usually conforms to the normal shape of the pancreas as seen on gross inspection. This feature is important in differentiating pancreatitis from tumors. The thickness of the pancreatic sonographic image is apparently related to the amount of organ edema present.[5] The borders are usually smooth and distinct, but occasionally an irregular outline is seen. At increasing transducer sensitivities, scattered internal echoes develop.

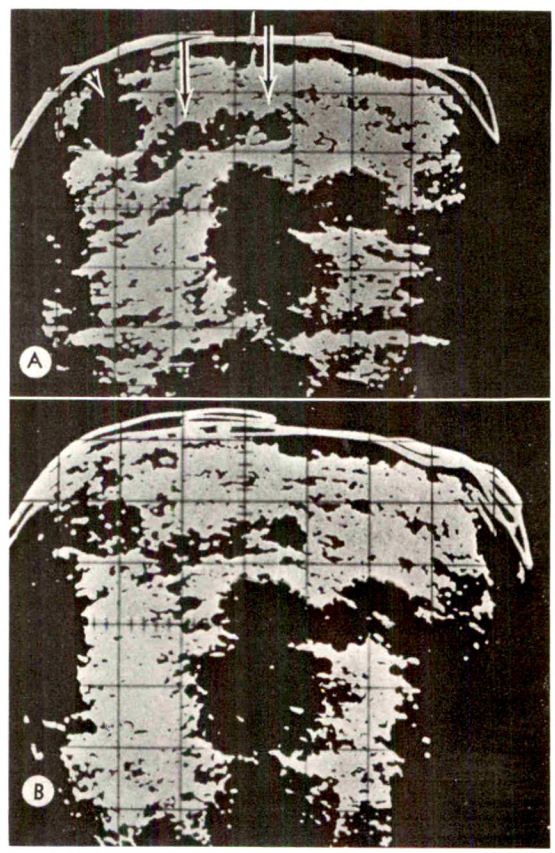


Fig. 4. Case IV. Chronic pancreatitis. (*A*) At a routine scanning sensitivity an elongated mass conforming to the general configuration of the pancreas is identified (arrows). The gallbladder is also noted (arrowhead). (*B*) At an increased sensitivity gallstones are now evident in the gallbladder.

C. PSEUDOCYST OF THE PANCREAS, CYSTADENOMA, AND CHOLEDOCHAL CYST

Case VII. A 28 year old female was admitted to K.U.M.C. following an automobile accident. At laparotomy a lacerated pancreas was found. Postoperatively the patient complained of persistent abdominal pain. Physical examination was handicapped by marked tenderness and guarding. A sonogram showed a large transonic mass characteristic of a cyst (Fig. 7*A*). A diagnosis of traumatic pseudocyst of the pancreas was made. A progress study in 1 week demonstrated an increase in the size of the lesion (Fig. 7*B*). Following marsupialization of the cyst, sonography documented a decrease in its size (Fig. 7*C*).

Case VIII. This patient entered K.U.M.C. for evaluation of abdominal pain. A plain film of the abdomen showed pancreatic calcification. An upper gastrointestinal series depicted classical changes of a mass in the head of the pancreas. A pancreatic isotope scan was abnormal. The sonogram was interpreted as a solid mass in the head of the pancreas because of the presence of marked internal echo formation with increasing sensitivity (Fig. 8, *A* and *B*). A cystadenoma of the pancreas was found at surgery.

Case IX. This 10 year old jaundiced white female presented with a large abdominal mass. Physical examination demonstrated a large right-sided abdominal mass which was thought to be a solid tumor. An excretory urogram was normal. An ultrasonic study outlined a large cystic structure (Fig. 9*A*) which appeared to be continuous with a dilated common bile duct (Fig. 9*B*). A preoperative diagnosis of choledochocyst was made on the basis of the ultrasonic study and was confirmed at surgery.

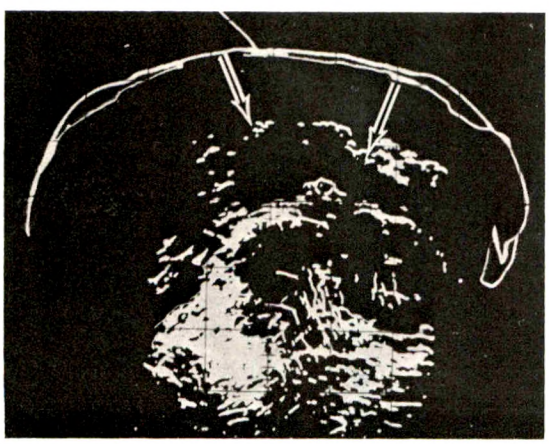


Fig. 5. Case V. Chronic pancreatitis depicting enlargement of the entire pancreas (arrows) particularly in the region of the pancreatic head.

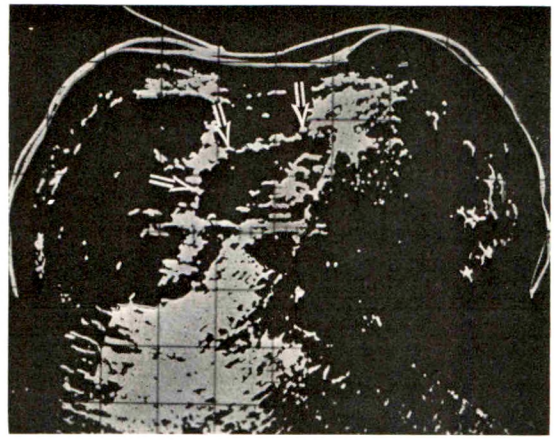


Fig. 6. Case VI. Chronic pancreatitis with the sonographic image conforming to the general shape of the pancreas (arrows).

Comment. Pseudocysts are generally round to oval transonic masses at normal sensitivity settings. In some cases internal echoes may be seen at higher sensitivity settings because of inhomogeneous cystic contents. However, the diagnosis of pseudocyst is usually not difficult. Erroneous interpretations may be avoided if plain film correlation is sought. In one of our cases the mass filled in at higher sensitivity settings because of the presence of abundant calcification. We have had no further experience with choledochocysts. Nevertheless, delineation of a cystic structure in a jaundiced child should be very suggestive of a choledochocyst.[12]

DISCUSSION

Diagnostic ultrasound is an innocuous means of examining a difficult region of the abdomen; namely, the pancreas. No untoward effects of ultrasound have been reported with energy levels used in clinical practice. Thorough familiarity with the equipment is essential, since no standardization exists. Adherence to published

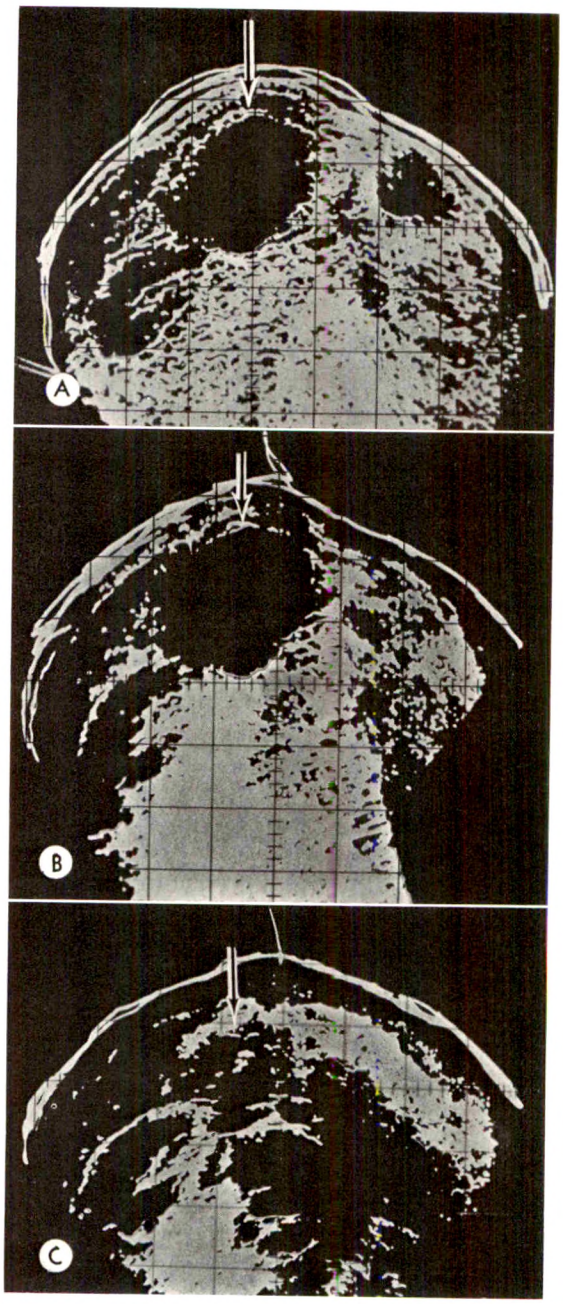


FIG. 7. Case VII. Traumatic pseudocyst of the pancreas. (*A*) Even at a high sensitivity the mass remains free from internal echoes indicating homogeneous contents. The size of the cyst can be measured; each square on the print equals 3 cm. (*B*) One week later the cyst has enlarged. (*C*) Following marsupialization, the cyst is significantly decreased in size.

diagnostic criteria will help prevent diagnostic errors.[6,13]

Filley and Freimanis[5] reported the sonographic appearance of the isolated pancreas in a waterbath. Our clinical experience supports their contention that the normal pancreas cannot be outlined as a distinct organ. Multiple disorganized echoes are present in the region of the normal pancreas. Occasionally, small irregular transonic areas appear in the region of the head of the pancreas. Repeat scans at a slightly increased transducer sensitivity usually resolve the problem since the transonic areas disappear.

The differential diagnosis between pancreatitis, carcinoma and pseudocysts has already been outlined. Metastatic disease to the peripancreatic region may simulate carcinoma of the pancreas. Scans at different levels in the abdomen provide helpful differential information. Direct extension by other tumors into the pancreas can usually be detected by other roentgenographic studies. Retroperitoneal lymph node enlargement may occasionally be confused with carcinoma of the pancreas. However,

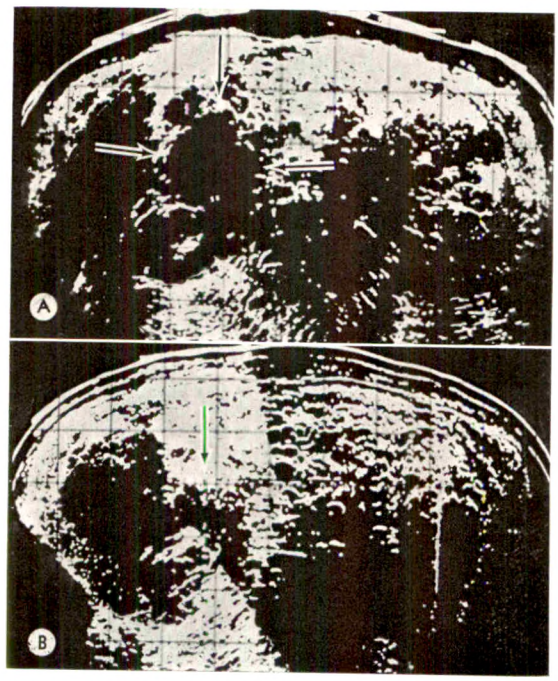


FIG. 8. Case VIII. Calcified pseudocyst of pancreas. (*A*) At routine sensitivity the mass (arrows) has the characteristics of a cyst. (*B*) With increased sensitivity the mass has filled in simulating a solid tumor. The internal echoes in this case are felt to be secondary to the extensive calcification.

in the former, other periaortic lymph nodes are usually enlarged and the lymph nodes themselves tend to be round with smooth borders. Aortic aneurysms should be easily distinguished from a pancreatic lesion by multiple transverse and longitudinal scans.

It is difficult to assess diagnostic accuracy in a small series. However, we have been impressed with the frequency of positive information obtained by sonography. Of 18 operative cases, the sonographic diagnosis proved to be correct on 16 occasions.

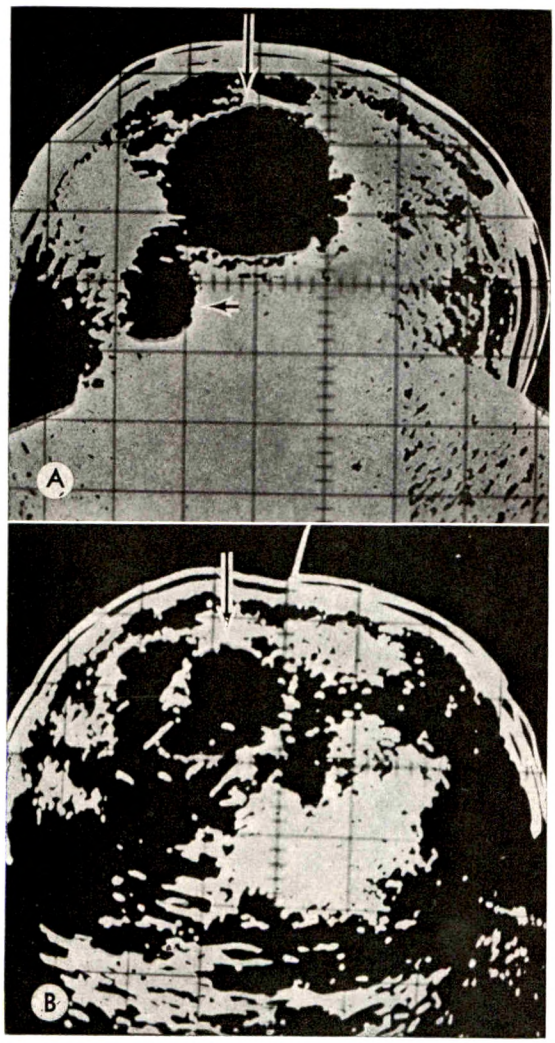


FIG. 9. Case IX. Choledochal cyst. (*A*) A large cystic mass is outlined at a level 4 cm. above the umbilicus. A diverticular outpouching is noted (arrowhead). (*B*) Consecutive cephalad scans revealed the mass to be continuous with a segment of markedly dilated common duct (arrow) at a level 11 cm. above the umbilicus.

One error represented a false negative. In this case a carcinoma of the cardia had extended into the tail of the pancreas. In this small series a diagnostic accuracy of 84 per cent has been achieved. If one considers only primary carcinomas of the pancreas, a correct diagnosis was made by ultrasound in 7 of 8 cases. The 1 error was a false negative examination in a very obese patient.

In the 13 nonoperated patients there was 1 apparent false positive. This patient had back pain and gastrointestinal symptoms. The initial sonogram revealed a mass in the tail of the pancreas. A celiac arteriogram, isotope scan and upper gastrointestinal series were all normal. Two repeat sonograms were completely normal. A review of the original sonogram showed that the basic diagnostic requirement of through transmission[13] had not been satisfied.

Serial studies are completely safe. They frequently provide important information regarding the development of a lesion or changes in the size of a tumor or cyst following therapy.

CONCLUSION

Since it is noninvasive and has a relatively high diagnostic yield, sonography should be the initial procedure in evaluating patients with suspected pancreatic disease.

Continued use of the modality and improvement in resolution and electronic display will increase the diagnostic accuracy of sonography and expand its usefulness.

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REFERENCES

1. BOOKSTEIN, J. J., REUTER, S. R., and MARTEL, W. Angiographic evaluation of pancreatic carcinoma. *Radiology*, 1969, *93*, 757–764.
2. EATON, S. B., FERRUCCI, J. T., and BENEDICT, R. T. Comprehensive evaluation of radiologic exploration of pancreas. *Crit. Rev. Radiol. Sc.*, 1970, *1*, 165–205.
3. ENGELHART, G., and BLAUENSTEIN, U. W. Ultrasound in diagnosis of malignant pancreatic tumors. *Gut*, 1970, *11*, 433–449.
4. EYLER, W. R., CLARK, M. D., and RIAN, R. T.

Evaluation of roentgen signs of pancreatic enlargement. *J.A.M.A.*, 1962, *181*, 967–972.

5. Filley, R. A., and Freimanis, A. K. Echographic diagnosis of pancreatic lesions. *Radiology*, 1970, *96*, 575–582.
6. Freimanis, A. K., and Asher, W. M. Development of diagnostic criteria in echographic study of abdominal lesions. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *108*, 747–755.,
7. Holm, H. H. Ultrasonic scanning in diagnosis of space occupying lesions of upper abdomen. *Brit. J. Radiol.*, 1971, *44*, 24–36.
8. Landman, S., Polcyn, R., and Gottschalk, A. Pancreas imaging—is it worth it? *Radiology*, 1971, *100*, 631–636.
9. Leopold, G. R., and Asher, W. M. Deleterious effects of gastrointestinal contrast material on abdominal echography. *Radiology*, 1971, *98*, 637–640.
10. Reuter, S. R., Redman, H. C., and Bookstein, J. J. Differential problems in angiographic diagnosis of carcinoma of pancreas. *Radiology*, 1970, *96*, 93–99.
11. Rodriquez, A., Alfidi, R. J., and Gill, W. M. Pancreas scanning. *Crit. Rev. Radiol. Sc.*, 1970, *1*, 25–44.
12. Suruga, K., Hirai, Y., Nagashima, K., Wagai, T., and Ihui, M. Ultrasonic echo examination as aid in diagnosis of congenital bile duct lesions. *J. Pediat. Surg.*, 1969, *4*, 452–456.
13. Templeton, A. W., and Stuber, J. L. Abdominal and retroperitoneal sonography. To be published.

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SPLENIC TRAUMA: ASSESSMENT OF PROBLEMS IN DIAGNOSIS*

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SPLENIC injury occurs frequently in patients with blunt abdominal trauma. These lesions are often clinically obvious because of hemoperitoneum and left upper quadrant injury. Such patients undergo exploratory celiotomy. There is, however, a group of patients who have blunt abdominal trauma in whom there is less convincing evidence of intraabdominal injury. These patients may have a minimally positive lavage for intraperitoneal blood or may be seen some days after an injury with vague left upper quadrant symptoms or a mass. Angiography has been used rather

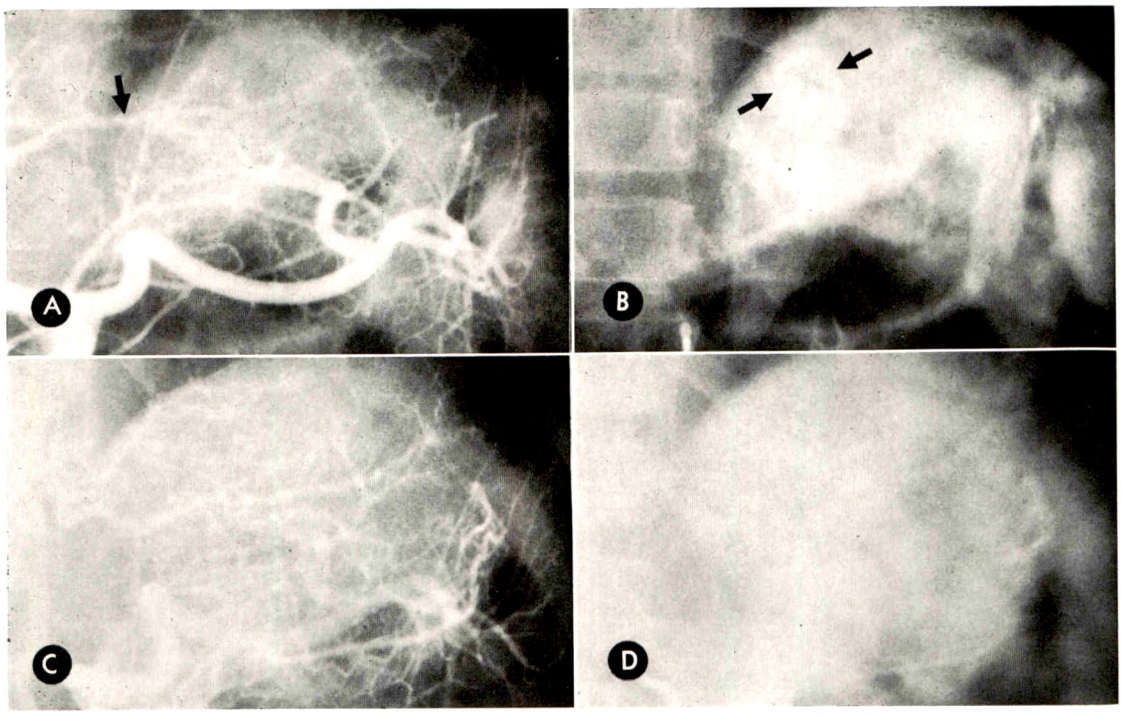


FIG. 1. Normal spleen following automobile accident. Parenchymal stain in liver or gastric fundus suggesting splenic rupture.

(*A*) Celiac injection on a 14 year old girl after an automobile accident. Anteroposterior projection (arrow indicates left hepatic artery). (*B*) The parenchymal phase shows dense contrast stain in the gastric fundus overlying the superomedial aspect of the spleen. A small area of punctate contrast material accumulation is present (arrows), but there is no way of telling whether it is in spleen, stomach or left lobe of liver.

(*C*) Celiac arteriogram in the left posterior oblique projection. (*D*) Parenchymal phase. The area of punctate contrast material accumulation is no longer seen. The upper pole of the spleen still cannot be identified however. A selective splenic arteriography was not performed. (Courtesy of J. J. Bookstein, Ann Arbor.)

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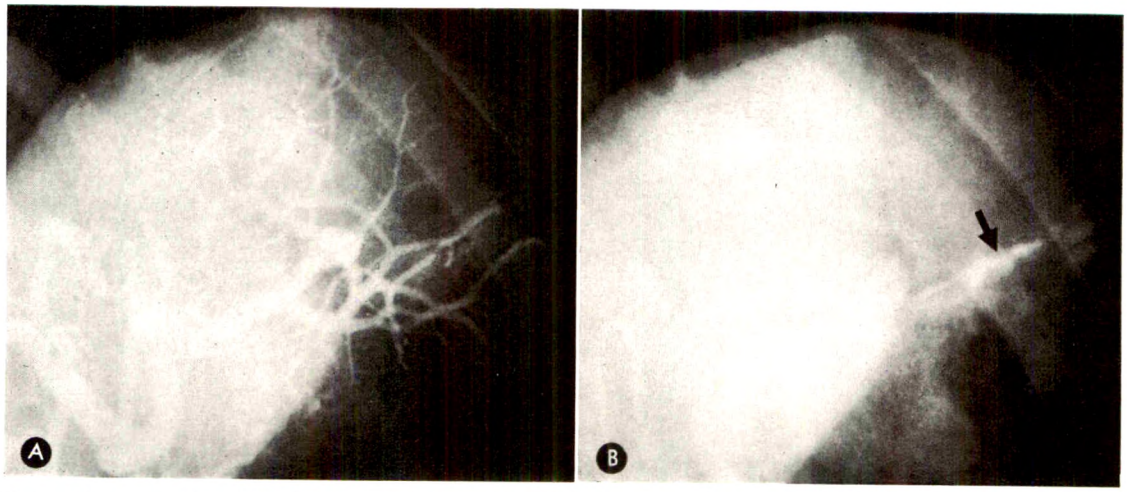


FIG. 2. Normal spleen following automobile accident. Contrast material accumulation in mesentery adherent to spleen simulating splenic rupture.

(*A*) Celiac arteriogram in left posterior oblique projection. The splenic arteries are all normal without stretching or displacement. (*B*) Parenchymal phase, showing dense linear accumulation of contrast material (arrow) corresponding to midportion of the spleen. Note that, because this was a celiac artery injection, the splenic contours are not well defined. No selective splenic arteriography was performed.

extensively in the evaluation of these patients.[1–7] False negative diagnoses have been consistently avoided. However, we have encountered a number of patients in whom various angiographic findings suggested splenic abnormality. These false positive findings can be distinguished from true injury in most patients. Several such pitfalls are discussed here.

MATERIAL AND METHOD

Over the past 4 years 83 patients with blunt abdominal trauma have undergone abdominal angiography at Wayne County General Hospital. Screening angiograms were obtained in patients with a weakly positive peritoneal lavage in order to determine proper management. These studies generally included lumbar aortography and celiac and superior mesenteric angiography. If splenic injury was specifically suspected, selective splenic arteriography may also have been performed in the anteroposterior and left or right posterior oblique projections.

RESULTS

There were 8 splenic lacerations and 3 subcapsular hematomas in this group of patients. Five patients had minor abnormalities, interpreted as insignificant splenic contusion. In addition, there were 12 patients in whom the early vascular studies suggested serious splenic injury. Subsequent injections of the celiac or splenic arteries in the same or different projections or following air insufflation into the stomach, indicated that the spleen was normal in 11. In 1 patient a false positive diagnosis of splenic rupture was made. In all 12 patients normal spleens were found at surgery or autopsy or strongly suggested by uneventful clinical courses.

DISCUSSION

The angiographic features of splenic rupture are several. They include: arterial leakage of contrast material into a laceration; early venous filling; arterial displacement; mottled or irregular contrast medium accumulation; and areas of absent contrast medium accumulation due to laceration. Subcapsular hematomas of the spleen may also displace extrasplenic arteries and veins and compress the splenic parenchyma medially into a crescent shape. Such spleens are enlarged, while those with

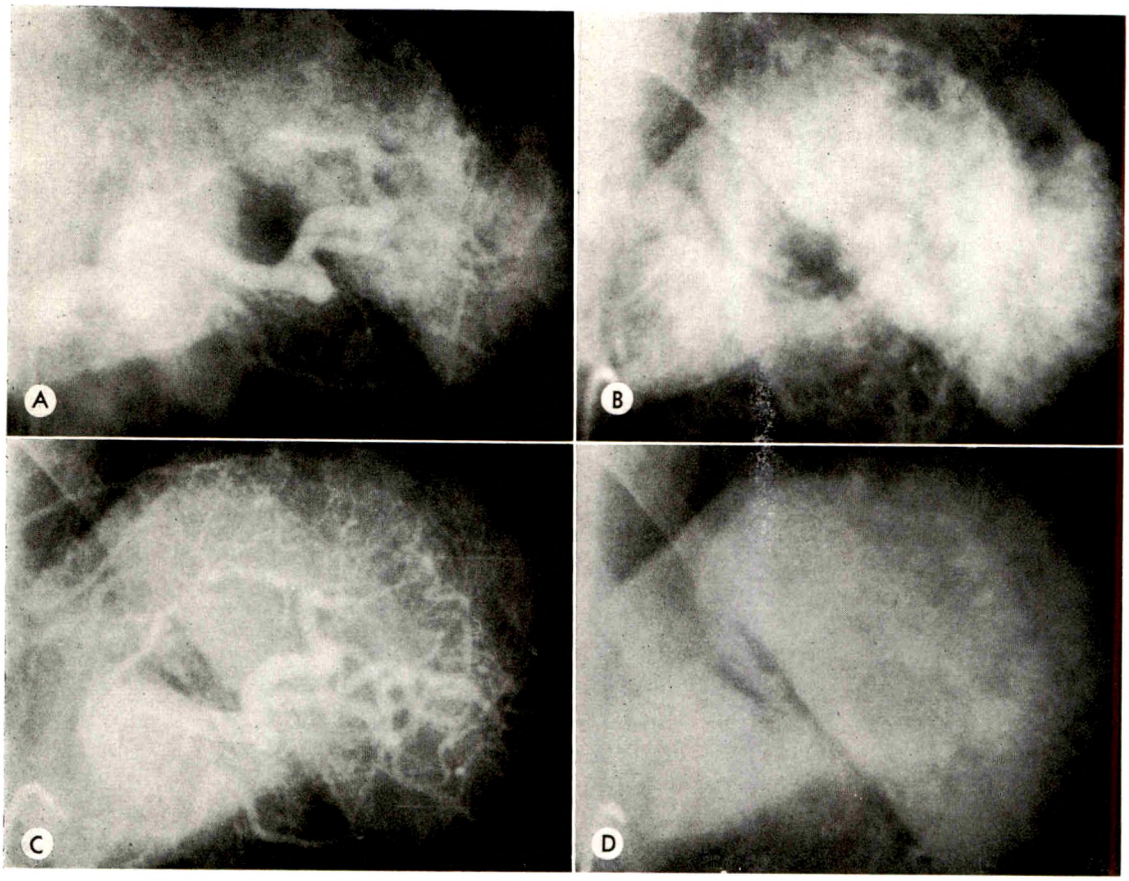


Fig. 3. Normal spleen following automobile accident. The splenic contours are obscured in the anteroposterior projection of celiac arteriography, but well demonstrated in the left posterior oblique projection. Selective splenic arteriography, therefore, was not required.

(*A*) Anteroposterior celiac arteriogram. Arterial phase. (*B*) Parenchymal phase. The upper pole of the spleen cannot be identified due to overlying of the gastric fundus.

(*C*) Celiac arteriogram in left posterior oblique position. Arterial phase. (*D*) Parenchymal phase. The gastric fundus can still be identified, but does not interfere with the splenic contour visualization. Note also the irregularities in the splenic contour along the superior margin. This degree of irregularity is frequently seen and is normal.

rupture may be normal in size. Each of these abnormalities is very definite and is always seen in every projection.

Problems in diagnosis have occurred most frequently in patients with anatomic variations. The spleen often lies behind the colon or stomach, and the left lobe of the liver may be superimposed on the spleen. The tail of the pancreas sometimes is adjacent to the spleen. The added parenchymal accumulation of contrast material in the liver, stomach, colon or pancreas can make the interpretation of splenic angiograms difficult (Fig. 1, *A–D*). Adherent omentum can cause a similar problem (Fig. 2, *A* and *B*). The overlying contrast material makes the splenic accumulation appear mottled or irregular. A second projection or varying the amount of air in the stomach or colon often will separate the structures (Fig. 3, *A–D*; and 4, *A* and *B*), so that the area in question lies outside the splenic contour or overlies a different part of the spleen. Superselective splenic arterial injection diminishes the problems in interpretation, since fewer overlying arteries are opacified (Fig. 5, *A–D*).

The spleen frequently does not appear

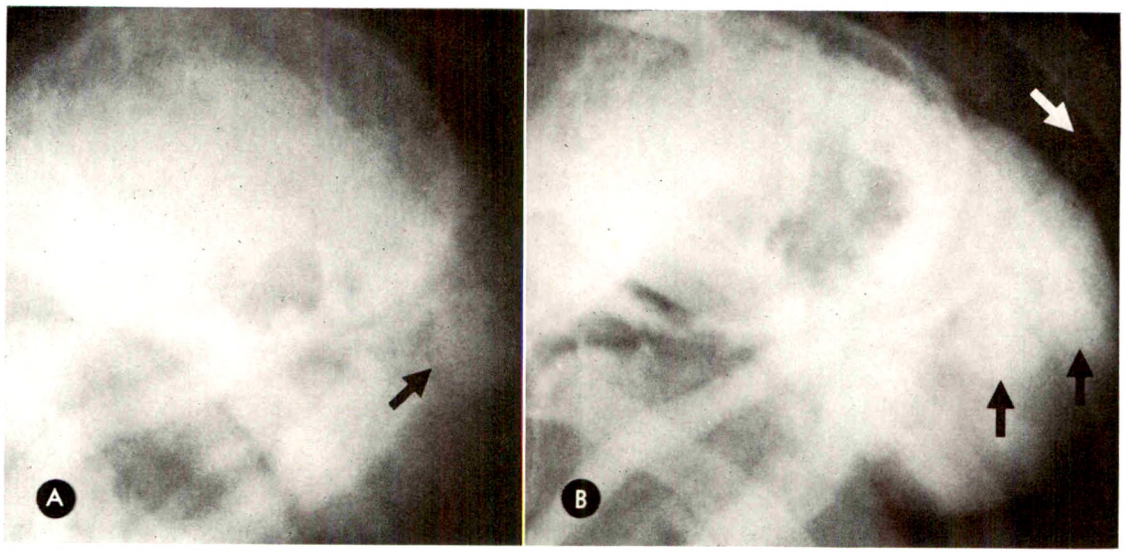
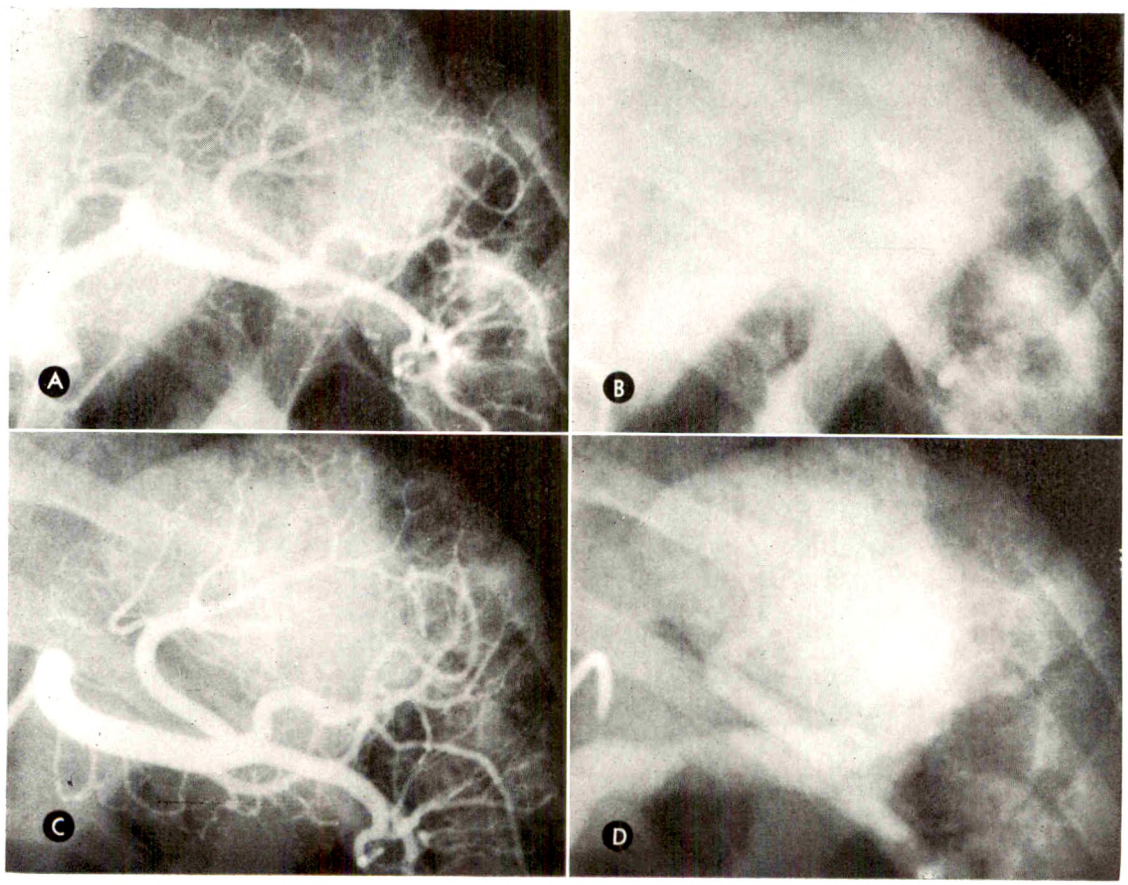


FIG. 4. Normal spleen following automobile accident. Gas insufflation into stomach permits visualization of splenic contour through the gastric fundus.

(*A*) Anteroposterior projection of venous phase of celiac arteriogram. The superior margin of the spleen is not adequately visualized. Only one accessory spleen is seen (arrow).

(*B*) Slight right posterior oblique projection (estimated at approximately 15°) of venous phase of celiac arteriogram. The gastric body and fundus are better outlined by gas. The superior margin of the spleen is better visualized. Now 3 accessory spleens are visible (arrows), each drained by a separate vein.



to lie against the diaphragm superiorly and the superior contour is often less distinct than the remainder of its outline, although it is sometimes better defined in the left posterior oblique projection. Indistinctness of the superior margin cannot be considered abnormal and, in the absence of other findings of splenic rupture, this cannot be considered diagnostic of splenic injury (Fig. 3, *A–D*; and 4, *A* and *B*).

Fetal lobulations commonly occur on the lateral margin of the spleen. Lobulations result from incomplete fusion of the splenic anlage. Such lobulations indent the splenic contour in 1 or more places (Fig. 6). These indentations may appear indistinct, especially in the oblique projections. Generally, however, the anteroposterior projection will demonstrate these lobulations clearly as sharply defined defects without any associated arterial lesions. Lacerations of the spleen do not have distinct, sharp, smooth margins. Often lacerations are associated with occluded or displaced arteries or irregular contrast medium accumulations adjacent to the laceration itself. Such abnormalities are never seen with fetal lobulations alone. Lobulations can be quite deep, extending nearly to the splenic hilum. Indeed, some patients have several spleens due to failure of fusion.

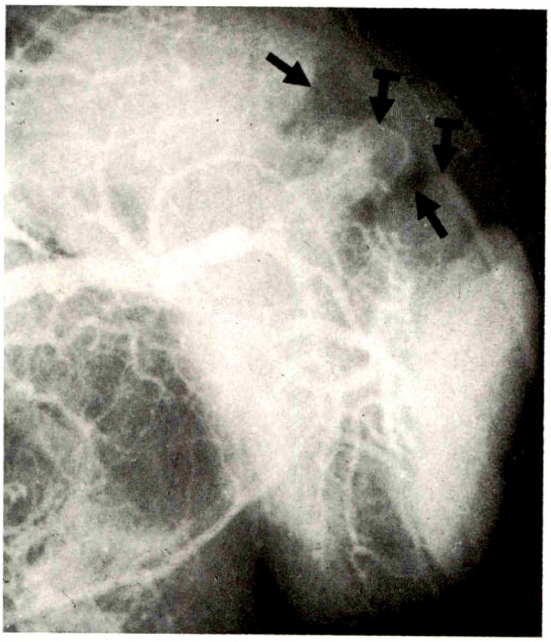


Fig. 6. Fetal lobulations in a normal spleen following automobile accident. The parenchymal defects have smooth sharp borders (simple arrows). The lobulations have rounded margins (crossed arrows). No other signs of splenic rupture are evident.

Contrast medium accumulation in the spleen is generally uniform and homogeneous. Density varies only with the thickness of the splenic parenchyma. Abnormal contrast accumulation due to contusion usually involves only a portion of the spleen and is often associated with arterial displacement or early venous filling. We have encountered several patients, who have had a small area of uneven contrast medium accumulation in an isolated part of the spleen without any accompanying abnormality. These have been called minor contusion and have been treated conservatively without any delayed complications.

Accessory spleens can simulate an area of increased contrast medium accumulation, but these should not be confused with splenic injury (Fig. 4, *A* and *B*). They are discrete and often have a definite feeding artery and draining vein. Frequently an oblique view will move the accessory spleen or the spleen itself, should differentiation be difficult.

←

Fig. 5. Normal spleen following automobile accident. Value of superselective splenic arteriography.

(*A*) Celiac arteriogram in anteroposterior projection. Arterial phase. (*B*) Parenchymal phase. The splenic contours are well seen, except for the superomedial portion. This part is obscured by parenchymal stain in the gastric fundus.

(*C*) Superselective anteroposterior splenic arteriogram. Arterial phase. (*D*) Venous phase. The superomedial aspect of the spleen is well visualized. A portion of the gastric fundus is still stained by contrast material accumulation, but interferes less with splenic visualization than on the celiac arteriogram.

SUMMARY

Review of our experience with splenic arteriography following blunt abdominal trauma revealed no instance of missed splenic injury and only 1 case of false positive diagnosis of splenic injury. However, 12 patients in our series of 83 examinations presented some difficulty in establishing a diagnosis of no splenic injury and required more than a single anteroposterior celiac arteriogram.

Techniques employed to arrive at a correct diagnosis in 11 of the 12 patients included: changing the amount of gas in the stomach; right and left posterior oblique filming projections; superselective splenic arteriography; and recognition of normal variants in splenic anatomy.

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REFERENCES

1. Aakhus, T., and Enge, I. Angiography in traumatic rupture of spleen. *Brit. J. Radiol.*, 1967, *40*, 855–861.
2. Berk, R. N., and Wholey, M. H. Application of splenic arteriography in diagnosis of rupture of spleen. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *104*, 662–667.
3. Lepasoon, J., and Olin, T. Angiographic diagnosis of splenic lesions following blunt abdominal trauma. *Acta radiol.* (Diag.), 1971, *11*, 257–273.
4. Love, L., Greenfield, G. B. Braun, T. W., Moncada, R., Freeark, R. J., and Baker, R. J. Angiography of splenic trauma. *Radiology*, 1968, *91*, 96–102.
5. Lundstrom, B. Angiographic demonstrations of rupture of spleen. *Acta radiol.* (Diag.), 1970, *10*, 145–150.
6. Stein, H. L. Diagnosis of traumatic laceration of spleen by selective arteriography: direct serial magnification angiography, and intra-arterial epinephrine. *Radiology*, 1969, *93*, 367–372.
7. Thompson, D. P., Shultz, E. H., and Benfield, J. R. Celiac angiography in management of splenic trauma. *A.M.A. Arch. Surg.*, 1969, *99*, 494–497.

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HAMARTOMA OF THE SPLEEN*

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A NUMBER of papers have described benign tumors of the spleen with varying histology that have been given such descriptive names as hamartoma, splenoma, splenadenoma, hemangioma and nodular hyperplasia of the spleen. These lesions are relatively rare and are usually diagnosed at the time of laparotomy and histologic study.

Histologically the splenic hamartoma may be composed of abnormal mixtures of normal splenic elements. Basically 2 main types are described: lymphoid; and pulposal (red pulp).

This report describes an additional splenic hamartoma of the pulposal type, and the arteriographic and scintigraphic characteristics that make preoperative diagnosis possible.

REPORT OF A CASE

A 71 year old white female with hematuria was evaluated at the University of Virginia Hospital.

Routine blood studies and pyelograms were normal.

An abdominal aortogram and selective renal and splenic arteriograms showed a circumscribed mass superimposed on the lower pole of the spleen. The major blood supply was the splenic artery and the gastroepiploic artery. The lesion appeared to contain neovascularity and displaced adjacent splenic vessels. Since

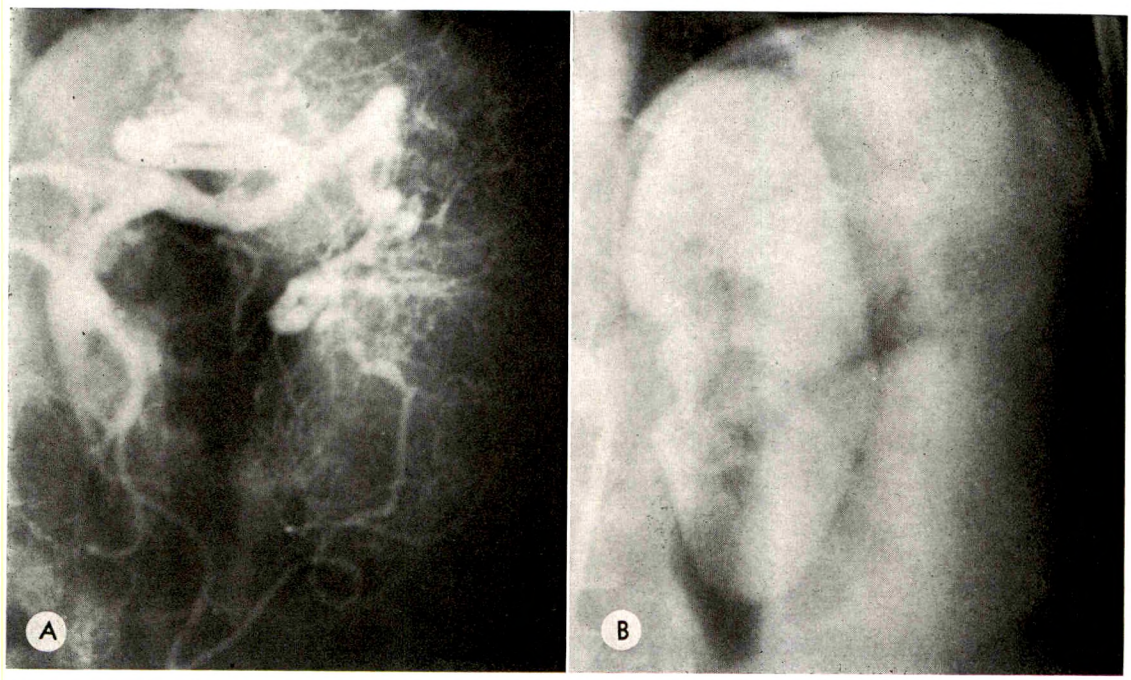


FIG. 1. (*A*) Arterial phase of splenic arteriography which shows stretching and upward displacement of intrasplenic arterial branches with early venous filling, both in the spleen and in the mass. The vascular pattern resembles neovascularity. (*B*) Late phase of aortography showing a dense, homogeneous parenchymal blush in the mass as contrasted to the less homogeneous blush of the remainder of the spleen. The left nephrogram appears normal.

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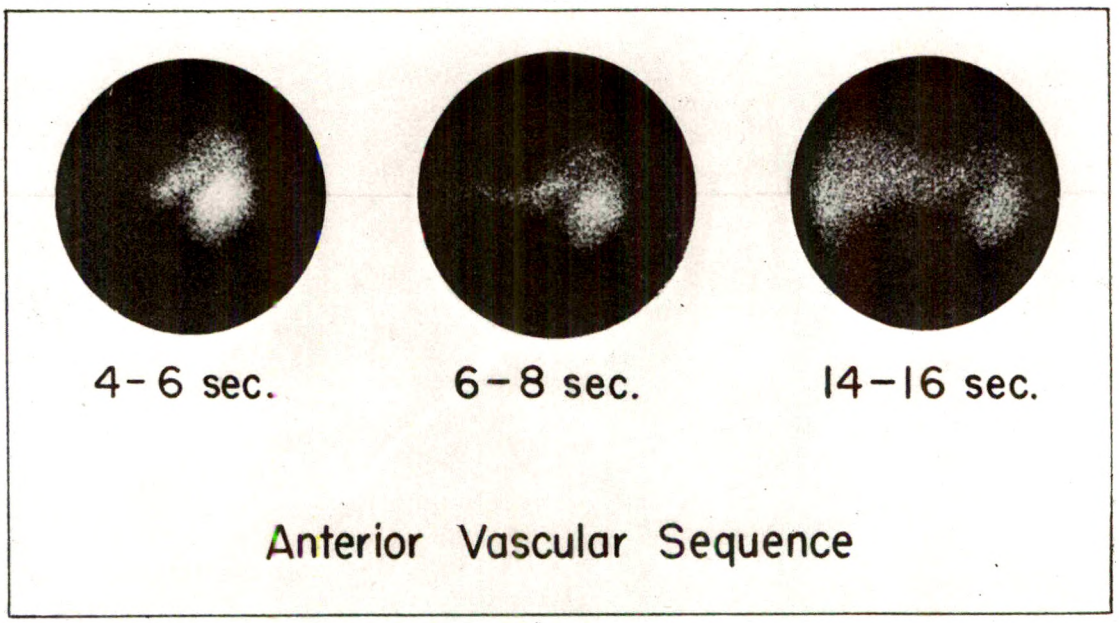


FIG. 2. Scintiphotographs of the spleen after the selective injection of 6 mc Tc^{99m} pertechnetate in the splenic artery. The mass at the lower pole of the spleen has a greater blood pool than the normal spleen and the activity persists after much of the radionuclide is washed into the splenic vein and liver.

the left kidney appeared uninvolved, it seemed that the lesion arose from the spleen, but involvement the of greater curvature of the stomach could not be completely ruled out (Fig. 1, *A* and *B*).

The selective splenic artery catheter was used to inject 6 mc Tc^{99m} pertechnetate while rapid sequence views were obtained with the gamma camera. This sequence demonstrated increased blood flow and blood pooling in the lower pole of the spleen, which persisted somewhat after the radionuclide cleared from the rest of the spleen (Fig. 2).

The patient subsequently had a spleen

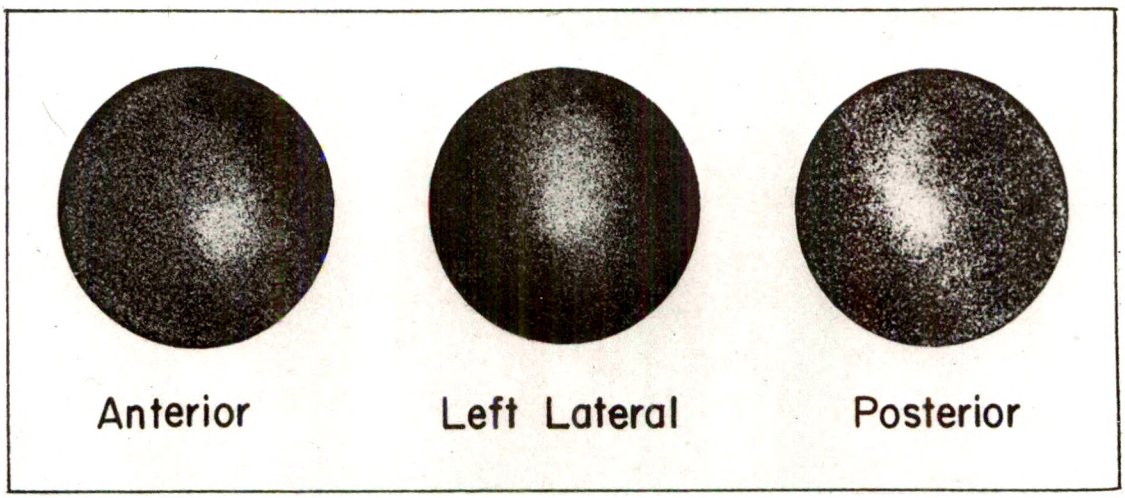


FIG. 3. Scintiphotographs of the spleen several hours after administering Cr^{51} labeled damaged red blood cells. The lesion appears to have greater activity than the normal upper pole of the spleen.

scintigraphy with heat-treated, Cr^{51} labeled red blood cells. Gamma camera and rectilinear images showed that the lesion sequestered red cells, and, in fact, the count rate over the lesion was higher than over the rest of the spleen (Fig. 3).

Surgery was delayed 2 weeks during which time the hematuria and urinary tract infection had cleared. At exploratory laparotomy the lower pole of the spleen was noted to be enlarged. The spleen was removed and the postoperative course was uneventful.

Pathologic examination showed the spleen to weigh 163 grams, measuring $13 \times 5\frac{1}{2} \times 3$ cm. The lower pole contained a nodule measuring $5\frac{1}{2}$ cm. in diameter which was moderately well circumscribed and not markedly different in appearance from the rest of the spleen. The nodule was for the most part splenic pulp tissue with a marked reduction in number of trabeculae and follicles. The descriptive label most suitable for this lesion was a nodular hyperplasia or hamartoma, predominantly red pulp type (Fig. 4, *A* and *B*).

DISCUSSION

Since the histology of benign spleen tumors varies somewhat, it is not surprising that the angiographic and scintigraphic patterns vary. A previous communication from this hospital described a benign tumor that failed to sequester heat-treated red blood cells.[1] That tumor was composed of vascular spaces, fibrin, and areas of necrosis, being termed a hemangioma. Angiographic studies of that tumor were not obtained. The lesion reported herein was composed largely of splenic pulp tissue that sequestered damaged red blood cells readily. We did not obtain tissue count rates, but the tumor count rate appeared to exceed the normal spleen on scintigraphy. This would indicate that the tumor was composed of functional splenic pulp. Sequestration of heat-treated red blood cells is not surprising, since splenic hamartomas have been known to cause hypersplenism when of large size.[4,6–8]

Previous reports of the angiographic appearance of splenic hamartomas are quite similar to that observed in the present case. Wexler and Abrams[9] noted stretching and displacement of adjacent vessels. Vessels

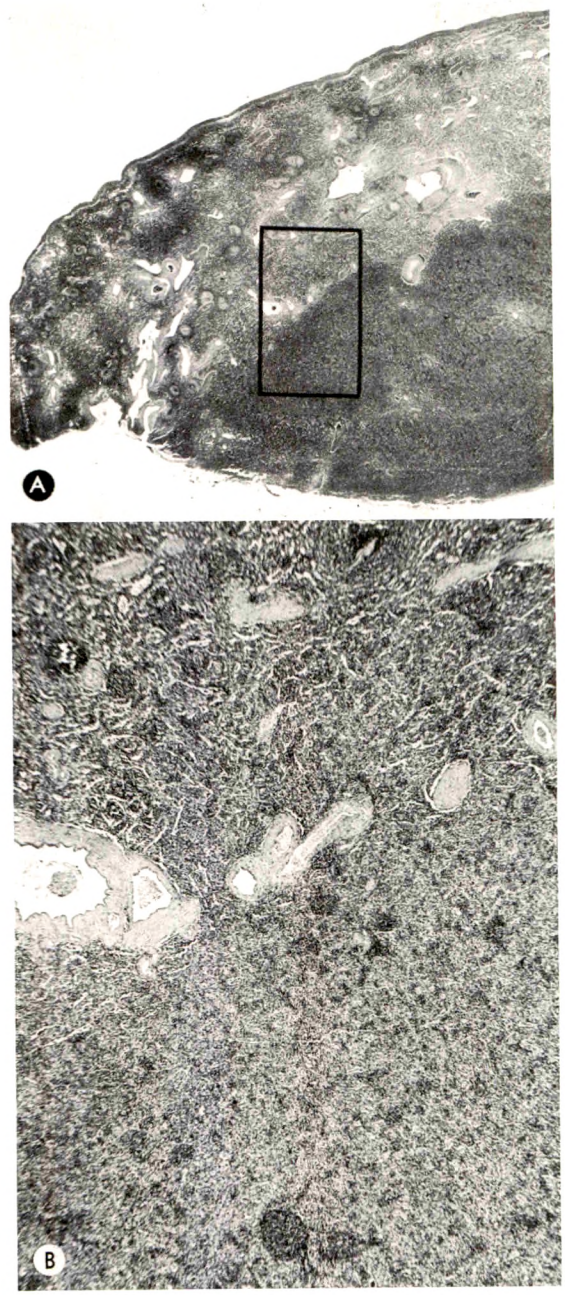


Fig. 4. (*A*) Low power section of the spleen showing normal spleen above and splenic hamartoma (pulposal-red pulp type) below ($\times 6\frac{1}{2}$). (*B*) Higher power view of the insert shows the hamartoma to be composed for the most part of red pulp ($\times 35$).

within the lesion were suggestive of malignant neovascularity with tortuosity, irregularity, venous lakes and arteriovenous shunting. The pattern in the lesion described by Graham *et al.*[3] is similar even though the histology was different, resembling a hemangioma.

The roentgenographic and scintigraphic studies reported herein demonstrated a solid, vascular tumor in the spleen which functioned like normal or "supernormal" spleen in regard to sequestering damaged red blood cells. This combination would appear to rule out all solid or cystic masses in the spleen except nodular hyperplasia or hamartoma. Spleen scintigrams were not obtained with radioactive colloids, but presumably the result would be similar to that with damaged red blood cells. Many of the reported benign splenic tumors have histologic features similar to those reported in the present case, and a nonoperative diagnosis would seem possible if the proper studies were obtained.

SUMMARY

A case of hamartoma of the spleen is reported.

Arteriographic and scintigraphic studies demonstrated a solid, vascular splenic tumor which sequestered damaged red blood cells.

This pattern may allow nonoperative diagnosis of the hamartoma composed predominantly of splenic pulp.

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REFERENCES

1. BENJAMIN, B. I., MOHLER, D. N., and SANDUSKY, W. R. Hemangioma of spleen. *A.M.A. Arch. Int. Med.*, 1965, *115*, 280–284.
2. COE, J. I., and VON DRASHEK, S. C. Hamartoma of spleen: report of four cases. *Am. J. Path.*, 1952, *28*, 663–671.
3. GRAHAM, J. C., JR., WEIDNER, W. A., and VINIK, M. Angiographic features of organizing splenic hematoma ?hamartoma. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1969, *107*, 430–433.
4. HARDMEIER, T. Hypersplenismus bei einem Hamartom der Milz; Splenom. *Schweiz. med. Wchnschr.*, 1962, *92*, 1270–1274.
5. KIRKLAND, W. G., and MCDONALD, J. R. Hamartoma of spleen: report of three surgical cases. *Arch. Path.*, 1948, *45*, 371–379.
6. SCHRIJVER, H., and VERDONK, G. J. Hamartoma of spleen with inhibition of bone marrow. *Acta med. scandinav.*, 1957, *158*, 235–237.
7. SEN GUPTA, P. C., and MUKERJEE, A. M. Hamartoma of spleen. *J. Indian M. A.*, 1963, *41*, 610.
8. VIDEBAEK, A. Hypersplenism associated with hamartomas of spleen. *Acta med. scandinav.*, 1953, *146*, 276–280.
9. WEXLER, L., and ABRAMS, H. L. Hamartoma of spleen: angiographic observations. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1964, *92*, 1150–1155.

ACCORDION-LIKE COMPRESSION OF A CALCIFIED SPLENIC ARTERY: A PLAIN FILM ROENTGENOGRAPHIC SIGN OF SPLENIC ENLARGEMENT*

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PLAIN film roentgenographic diagnosis of splenic enlargement may depend on a large number of indirect, nonspecific findings apart from enlargement of the organ itself. These include: soft tissue density in the left upper quadrant; medial displacement of the stomach gas bubble or serration of its greater curvature; downward displacement of the splenic flexure; and medial and downward displacement of the left kidney. Elevation or restricted motion of the left hemidiaphragm, atelectasis in the left lower lung field, and fractured ribs may be seen with trauma or an acute process such as intrasplenic hemorrhage or infarction. If rupture has occurred, free intraperitoneal fluid may be present. Barium studies may be helpful in assessing organ enlargement. Aortography and selective angiography are often required to establish a diagnosis of laceration, hemorrhage, rupture or infarction.

The purpose of this paper is to describe an additional plain film roentgenographic finding of splenic enlargement: accordion-like compression and medial displacement of the calcified splenic artery. When seen in the appropriate clinical context, it may suggest an actute process such as hemorrhage or rupture.

REPORT OF A CASE

A 58 year old white female was admitted to the hospital complaining of severe back pain of 3 days' duration, which became progressively more severe and radiated to the left shoulder, neck, upper abdomen and buttock. She fainted twice just prior to admission. There was no history of trauma. Bowel movements had been normal. Past history included a hospital admission 8 months previously for investigation of severe hypertension (blood pressure ranging from 200–220 mm. Hg systolic/110–120 mm. Hg diastolic). The patient was treated with Ismelin, Thiazides and Meprobamate.

On the present admission, the blood pressure was 100 mm. Hg systolic/60 mm. Hg diastolic. The pulse was 92 per minute and regular. There was fullness in the left upper quadrant. All pulses were present and symmetric bilaterally. The chest was clear and the heart sounds were normal.

Laboratory findings were as follows: hemoglobin 7.6 gm. per cent; white blood cell count 7,050 per cu. mm.; post cibum sugar 166 mg. per cent; blood urea nitrogen 59 mg. per cent. Serum electrolytes and amylase were normal. Electrocardiogram showed left axis deviation and an old anteroseptal infarct. Stools were negative for occult blood.

Plain film roentgenograms of the abdomen were obtained and the diagnosis of splenic rupture was made on the basis of a left upper quadrant mass and free intraperitoneal fluid. Marked accordion-like compression with medial displacement of the calcified splenic artery was noted.

At laparotomy, the spleen had a large rent on its anteromedial surface and the peritoneal cavity was filled with blood. A splenectomy was performed. The spleen measured 11×8×6.5 cm. and a large subcapsular hematoma was present. Microscopic studies revealed moderate amyloidosis.

ROENTGEN FINDINGS

Figure 1 is a plain film roentgenogram before intravenous pyelography done on April 18th, 1968 (1st admission). There is marked calcification in the wall of the tortuous splenic artery.

Figure 2 is a supine roentgenogram of the abdomen at the time of the present admission. Note the accordion-like com-

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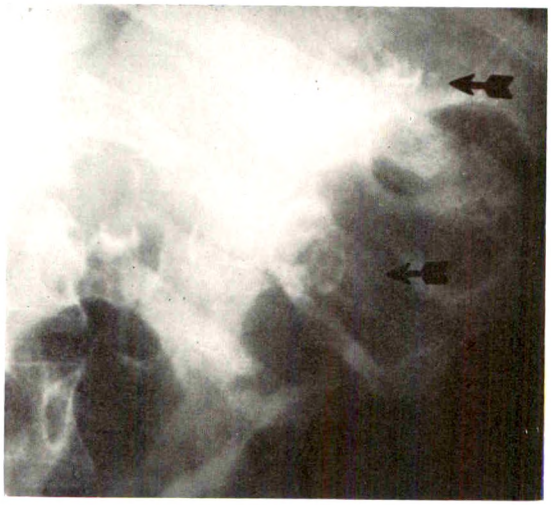


FIG. 1. *April 18, 1968.* There is marked calcification in the wall of the splenic artery.

pression of the calcified splenic artery by the left upper quadrant mass. The gas-filled stomach is displaced medially.

Figure 3 is a roentgenogram taken following splenectomy demonstrating a return to the original normal position of the splenic artery remnant.

DISCUSSION

Accordion-like compression of the splenic artery has been noted on arteriography in 2 of 17 cases or splenic rupture reported by Love *et al.*[3] To our knowledge, this is the first time that this finding has been described on plain film roentgenograms of the abdomen.

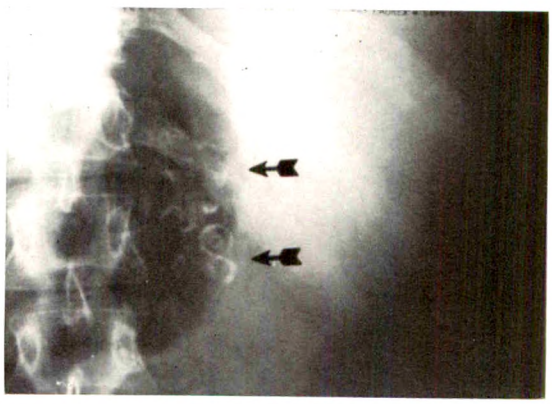


FIG. 2. *December 31, 1968.* Note the "accordion-like" compression and medial displacement of the splenic artery by the left upper quadrant mass.

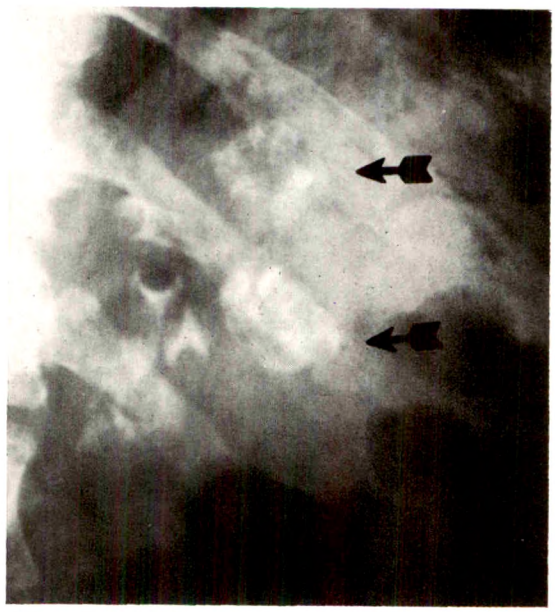


FIG. 3. *August 18, 1969.* The splenic artery remnant has returned to its normal position.

The mechanism would appear to be medial displacement of the splenic artery by the enlarging "mass" due to subcapsular or perisplenic hemorrhage.

Since the splenic artery is not infrequently calcified, especially in older patients, accordion-like deformity of this structure should be looked for as an additional sign when the diagnosis of splenic trauma is being considered. A change from previous roentgenograms, when these are available, would reinforce this finding.

It is true that any cause of splenic enlargement could lead to a similar displacement. However, when noted in the proper clinical context, this sign may be an important clue suggesting the diagnosis of subcapsular hemorrhage and/or rupture. Arteriography might then be performed if clinically indicated.

SUMMARY

A case of spontaneous splenic rupture is reported in which accordion-like compres-

sion of a calcified splenic artery is demonstrated roentgenologically, representing an additional sign in the diagnosis of splenic enlargement on plain film roentgenograms of the abdomen.

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REFERENCES

1. Berk, R. N., and Wholey, M. H. Application of splenic arteriography in diagnosis of rupture of spleen. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *104*, 662–667.
2. Berk, R. N. Changing concepts in plain film diagnosis of ruptured spleen. *J. Canad. A. Radiologists*, 1970, *21*, 67–70.
3. Love, L., Greenfield, G. B., Braun, T. W., Morcada, R., Freeark, R. J., and Baker, R. J. Arteriography of splenic trauma. *Radiology*, 1968, *91*, 96–102.
4. McCort, J. J. Radiologic examination in blunt abdominal trauma. *Radiol. Clin. North America*, 1964, *2*, 121–143.
5. Meschan, I. Roentgen Signs in Clinical Practice. W. B. Saunders Company, Philadelphia, 1966, pp. 1239–1241.
6. Taveras, J. M., and Golden, R. In: Golden's Diagnostic Radiology. Williams & Wilkins Company, Baltimore, 1968, pp. 14–124.
7. Zatzkin, H. R. The Roentgen Diagnosis of Trauma. Year Book Medical Publishers, Chicago, 1965, pp. 214–217.

HIGH INCIDENCE OF CELIAC AXIS NARROWING IN ASYMPTOMATIC INDIVIDUALS*

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ATTENTION was first called to the clinical implications of the celiac compression or median arcuate ligament syndrome by Dunbar *et al.*,[6] who described a group of patients with intermittent abdominal pain, usually in the postprandial period. Diarrhea, nausea and vomiting, weight loss and malabsorption were also present occasionally. The chief physical finding was a midepigastric abdominal bruit. The symptoms have been attributed to compression of the proximal celiac axis from above by either the median arcuate ligament of the diaphragm or the celiac plexus or both.[7] Reuter[8] has shown that this effect is accentuated by expiration, when the retroperitoneal contents (including the aorta and its branches) move in a cephalad direction with respect to the diaphragmatic ligament which is relatively fixed in position at the aortic hiatus. Other less frequent causes of celiac axis narrowing include atherosclerosis, fibromuscular hyperplasia, neoplasms, emboli, periarteritis nodosa, retroperitoneal fibrosis and congenital hypoplasia.

The original group of patients almost all benefited from surgical division of the median arcuate ligament and the adjacent cords of the celiac plexus, although it was not entirely clear whether this was due to release of compression of the artery or disruption of the neural pathways. More recently it has been pointed out that not all patients are helped by the operation.[2] Some authors have even questioned whether isolated narrowing of the celiac axis can actually produce clinical symptoms.[5,7] At the present time, then, there is lack of agreement as to the true significance of celiac compression, the mechanism of production of symptoms and the nature of therapy to be undertaken.

We have been impressed with the fact that this lesion is encountered from time to time in patients referred for clinically unrelated abdominal angiographic procedures and have wondered as to its incidence in a totally asymptomatic population. If an anatomic abnormality is thought to be pathologic, one of the first things to be determined is, if, or how often, it occurs in relatively healthy asymptomatic individuals. Bron and Redman[1] found a 12.5 per cent incidence of celiac axis narrowing among 713 abdominal aortograms. The degree of severity of narrowing was unspecified. Cornell[3] noted approximately a 1 per cent incidence of severe celiac axis narrowing among 1,500 abdominal aortograms. However, neither of these retrospective studies were designed to ascertain the incidence of the lesion among randomly selected symptom-free patients.

Elucidation of this point is the purpose of the present study.

CASE MATERIAL AND RESULTS

Fifty patients undergoing abdominal angiography at the New York Hospital—Cornell Medical Center participated in this study. They were selected at random with the only requirement being complete absence of abdominal pain or any other symptoms referable to the gastrointestinal tract. Twenty-six were potential renal donors, 11 had renal masses seen on intravenous urograms and 6 were being evaluated for hypertension. The remaining 7 were being studied for miscellaneous genitourinary tract problems. There were 20 males and 30 females. The age range was 21 to 77 years. After determining that one additional contrast injection would not be harmful, 40 cc. of hypaque 75M* was in-

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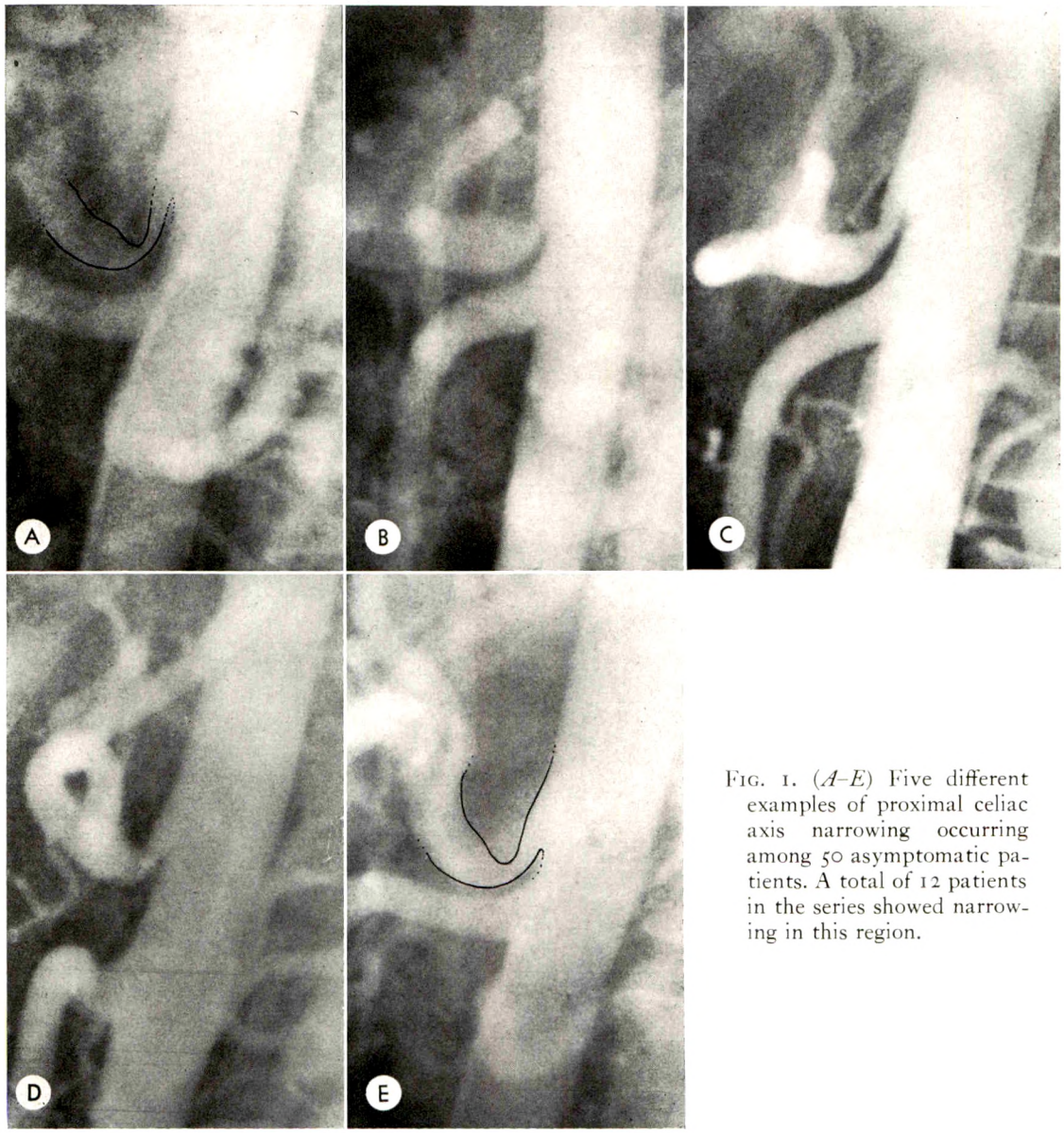


Fig. 1. (*A–E*) Five different examples of proximal celiac axis narrowing occurring among 50 asymptomatic patients. A total of 12 patients in the series showed narrowing in this region.

jected over a 2 second period with the catheter tip placed in the aorta a short distance above the celiac axis. Two films per second were exposed for 3 seconds with respiration suspended at full expiration. The study was considered positive only if more than 50 per cent narrowing of the proximal celiac axis was present.

Among the 50 subjects, 12 were noted to have celiac axis narrowing of 50 per cent or more. Representative examples of this positive group are shown in Figure 1, *A–E*. Only 1 of the 12 had an audible bruit. An important sign of celiac axis narrowing on the frontal aortogram or superior mesenteric arteriogram is collateral circulation from the superior mesenteric artery to the celiac axis through enlarged pancreaticoduodenal vessels. Only 3 of the 12 showed such collateral connections.

The proximal celiac axis on most of the positive lateral aortograms had a U-shaped configuration with compression along the superior aspect. This is characteristic of impingement by the median arcuate ligament and/or celiac plexus.

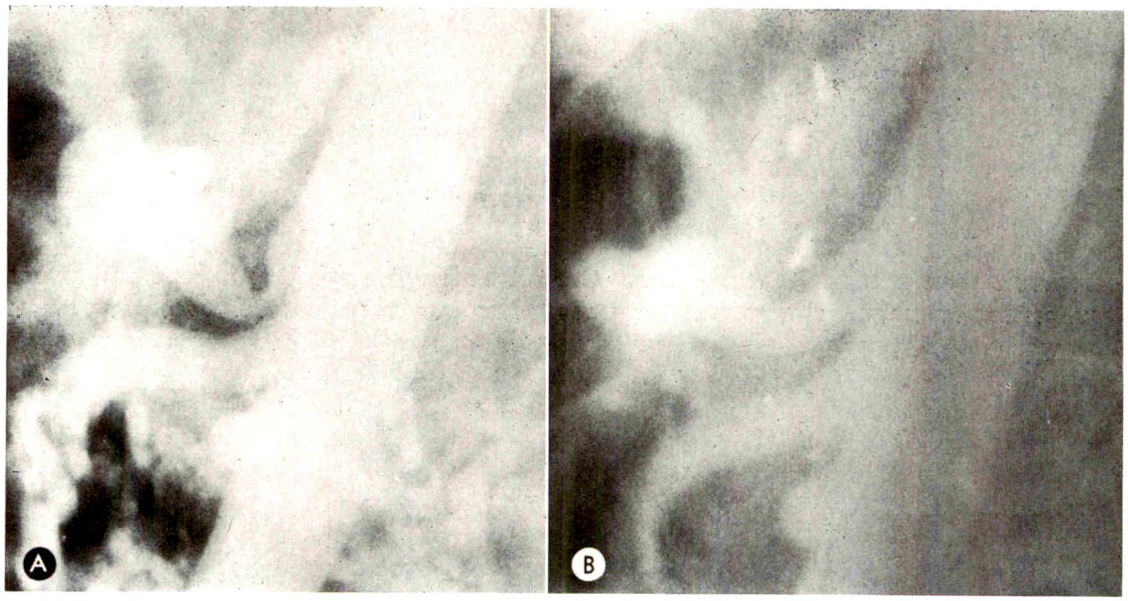


FIG. 2. (*A*) Lateral aortogram in a 51 year old male with vague epigastric pain, primarily at night and after meals. Postprandial nausea and vomiting also occurred frequently. No abdominal bruit was heard. Because of the symptoms, the finding of significant narrowing of the proximal celiac axis, and the configuration of this narrowing, a diagnosis of median arcuate ligament syndrome was made. This was confirmed at surgery and division of the ligament was carried out. (*B*) Postoperative lateral aortogram showing restoration of normal celiac axis anatomy. The operation resulted in complete disappearance of the patient's symptoms.

DISCUSSION

The discovery of a 24 per cent incidence of celiac axis narrowing among a group of asymptomatic patients is indeed surprising. Obviously this lesion is present in a significant percentage of the general population without producing clinically apparent disease.

Bron and Redman,[1] and Cornell[3] found a substantially lower incidence of this lesion than we have, but the reason for the difference seems obvious. The earlier studies were retrospective and presumably the large majority of patients did not have lateral aortograms, since this is not a standard part of abdominal angiographic procedures. Although the authors did not specify their protocols in this respect, it seems likely that lateral aortograms were obtained only when the frontal aortogram or superior mesenteric arteriogram suggested celiac obstruction by showing collateral circulation. If so, many cases of celiac axis narrowing were undoubtedly missed. As has been noted above, the majority of positive cases in our study failed to show these collaterals on the frontal roentgenograms.

In spite of the findings of this study, there is no doubt that celiac compression by the median arcuate ligament or celiac plexus can in some individuals cause serious symptoms. The case illustrated in Figure 2, *A* and *B*, is an example of classical median arcuate ligament syndrome with complete clinical cure and angiographic evidence of restoration of normal celiac axis anatomy following surgical division of the ligament.

Thus, in a patient with the often vague clinical complaints of this syndrome and positive angiographic findings, the dilemma arises as to whether the celiac axis compression is the true cause or merely an incidental discovery. Clinicians must exercise considerable caution in recommending surgery for these patients. Laparotomy should be performed only when the symptoms are serious and unrelenting and only after care-

ful investigation has failed to reveal any other possible etiology. The surgeon would be well advised to adopt a skeptical attitude and thoroughly explore the abdomen for other types of pathology before finally proceeding to incise the median arcuate ligament and adjacent cords of the celiac plexus.

SUMMARY

Lateral aortograms were obtained in 50 patients who had no abdominal pain or other gastrointestinal tract symptoms. Twenty-four per cent of the patients had significant narrowing of the proximal portion of the celiac axis.

Most of these vessels showed a configuration indicating compression from above by the median arcuate ligament of the diaphragm and/or the celiac plexus.

The surprisingly high frequency of occurrence of this lesion among asymptomatic individuals necessitates considerable caution in making the diagnosis of celiac compression or median arcuate ligament syndrome.

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REFERENCES

1. Bron, K. M., and Redman, H. C. Splanchnic artery stenosis and occlusion. *Radiology*, 1969, *92*, 323–328.
2. Carey, J. P., Stemmer, E. A., and Connolly, J. E. Median arcuate ligament syndrome: experimental and clinical observations. *A.M.A. Arch. Surg.*, 1969, *99*, 441–446.
3. Cornell, S. H. Severe stenosis of celiac axis: analysis of patients with and without symptoms. *Radiology*, 1971, *99*, 311–316.
4. Deutsch, V. Compression of coeliac trunk and angiographic evaluation of its hemodynamic significance. *Clin. Radiol.*, 1968, *19*, 309–314.
5. Drapanas, T., and Bron, K. M. Stenosis of celiac artery. *Ann. Surg.*, 1966, *164*, 1085–1088.
6. Dunbar, J. D., Molnar, W., Beman, F. F., and Marable, S. A. Compression of celiac trunk and abdominal angina: preliminary report of 15 cases. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1965, *95*, 731–744.
7. Lindner, H. H., and Kemprud, E. Clinicoanatomical study of arcuate ligament of diaphragm. *A.M.A. Arch., Surg.*, 1971, *103*, 600–605.
8. Reuter, S. R. Accentuation of celiac compression by median arcuate ligament of diaphragm during deep expiration. *Radiology*, 1971, *98*, 561–564.
9. Reuter, S. R., and Olin, T. Stenosis of celiac artery. *Radiology*, 1965, *85*, 617–627.
10. Stoney, R. J., and Wylie, E. J. Recognition and surgical management of visceral ischemic syndrome. *Ann. Surg.*, 1966, *164*, 714–727.

BUDD-CHIARI SYNDROME*

STUDY OF ANGIOGRAPHIC FINDINGS AND REMARKS ON ETIOLOGY

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THE Budd-Chiari syndrome consists of thrombotic occlusion of the hepatic veins causing hepatomegaly, ascites and abdominal pain. Occasionally, jaundice and hematemesis due to postsinusoidal portal hypertension also occur. The etiologic factors causing occlusion of the hepatic veins may be divided into a smaller group in which thrombosis is a primary condition, and into a larger group in which thrombosis is secondary to some other disease. In the primary form the etiology of the thrombosis, in most of the cases, is unknown. Secondary occlusion of the hepatic veins is principally due to intra or perihepatic tumorous processes. Renal carcinoma, retroperitoneal sarcoma extending into the inferior vena cava, and leiomyosarcoma of the inferior vena cava occluding the entrance of the hepatic veins with secondary thrombosis of the latter veins have been reported.[1,17,22]

In other cases, thrombophlebitis migrans, polycythemia and other hematologic conditions such as sickle cell anemia were described as primary causes of hepatic vein thrombosis.[21,22] Recently, obstruction of the inferior vena cava by a membrane above the entrance of the hepatic veins was found causing occlusion of the hepatic veins.[28]

The clinical picture and the laboratory findings in this disease are not pathognomonic. Liver biopsy does not reveal information as to the site of the lesion, and may also confuse the clinical picture. Parker[22] emphasized that patients who survived sufficiently long and developed hepatic parenchymal necrosis and fibrosis could have a biopsy resembling cirrhosis. Erlic *et al.*[8] operated on a young woman and performed a successful porta-caval shunt. Four months later, biopsy showed centrilobular and portal fibrosis, resembling postnecrotic cirrhosis. It is therefore necessary to examine the importance of the roentgenologic contribution to the diagnosis of the Budd-Chiari syndrome. Conventional roentgen examinations are not contributory except in cases complicated by portal hypertension causing esophageal varices. Inferior venacavography and, more recently, selective hepatic arteriography and selective hepatic venography were found to be useful diagnostic methods.[2,16,17,24]

The purpose of this paper is twofold: (1) to report on our experience with these latter diagnostic methods in 9 cases of hepatic vein occlusion (8 had primary thromboses of different etiology and 1 had secondary thrombosis due to leiomyosarcoma of the inferior vena cava); and (2) to investigate the various possible etiologic factors present in our 8 cases with primary thrombosis of the hepatic veins.

MATERIAL

Clinically, all 9 patients showed the classical features of the Budd-Chiari syndrome. All had marked hepatomegaly, ascites and abdominal pains. Their ages ranged between 3 and 50 years. Five were females and 4 were males. The suspected cause of the occlusion of the hepatic veins was as follows: in 2 patients there was a clear-cut trauma of the upper abdomen; in 1 of these the clinical signs of the hepatic

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vein thrombosis developed rapidly and the patient succumbed in a few days, due to extension of the thrombus into the right atrium, right ventricle and the main pulmonary arteries. In the other patient with trauma of the upper abdomen, the clinical course of occlusion of the hepatic segment of the inferior vena cava including the site of entrance of the hepatic veins was prolonged.

In 2 patients the symptomatology began shortly post partum. Another young woman took oral contraceptives for a few months prior to the onset of her symptoms. In 1 case, leiomyosarcoma of the inferior vena cava occluded the hepatic veins. Finally, in 3 patients no relevant history was available which could explain the cause of the Budd-Chiari syndrome. It may be of interest that 2 of these 3 patients were Arabs—an adult and a 3 year old child.

Four patients died and postmortem examination confirmed the diagnosis of Budd-Chiari syndrome in all. One additional patient had the diagnosis confirmed by needle biopsy of the liver.

METHOD

Inferior venacavography was performed by the percutaneous method introducing a large sized polyethylene catheter through the femoral vein into the iliac vein. Approximately 1 cc. per kg. of 60 per cent urografin (Schering) was injected by a Gidlund automatic injector at 6 kg. pressure. A large film size semiautomatic film changer (80/35 cm. films) was used in order to include the whole inferior vena cava and the chest in the exposed area. Hepatic phlebography was performed by the percutaneous introduction of a green end hole Kifa catheter through a femoral or antecubital vein. Under fluoroscopic television guidance, the catheter was directed into a hepatic vein and advanced as far as possible.

After pressure determinations, about 8–10 cc. of 75 per cent urografin (Schering) was injected in approximately 3–4 seconds. The roentgenographic recording was made by an Elema Schönander rollfilm changer at the rate of 2 films per second for the first 4 seconds, and 1 film per second for the succeeding 8 seconds.

Selective celiac angiography was performed percutaneously by the femoral approach according to the method of Ödman using a green Kifa catheter. About 50 cc. of 75 per cent urografin (Schering) was injected by a Gidlund automatic injector at 3–4 kg. pressure. The roentgenographic recording was done by the Elema Schönander automatic rollfilm changer at a rate of 2 films per second for 4 seconds, 1 film per second for the next 4 seconds and 1 film every third second for the succeeding 30 seconds.

ROENTGEN FINDINGS

Inferior venacavography was performed in all patients, selective hepatic venography in 2, and selective celiac and mesenteric arteriography in 3 patients.

INFERIOR VENACAVOGRAPHY

The inferior venacavograms showed 4 distinct patterns. In 3 of the patients there was a smooth long compression of the hepatic segment of the inferior vena cava, more marked from side-to-side than from back-to-front (Fig. 1*A*). In 1 case the compression was so marked that it caused almost complete occlusion, and the larger portion of the injected contrast material was shunted into the azygos system (Fig. 1*C*). In another patient with compression of the hepatic segment of the inferior vena cava, a repeated venogram 1 year later showed complete occlusion of the upper segment of the inferior vena cava (Fig. 1*B*).

The second pattern, noted in 1 case, was an irregular circular intrinsic filling defect at the upper end of the inferior vena cava at the level of entry of the hepatic veins (Fig. 1*D*). This was interpreted as representing extension of the thrombus from the hepatic veins into the vena cava.

The third pattern was noted in 1 case in which the inferior vena cava was completely occluded except for the hepatic seg-

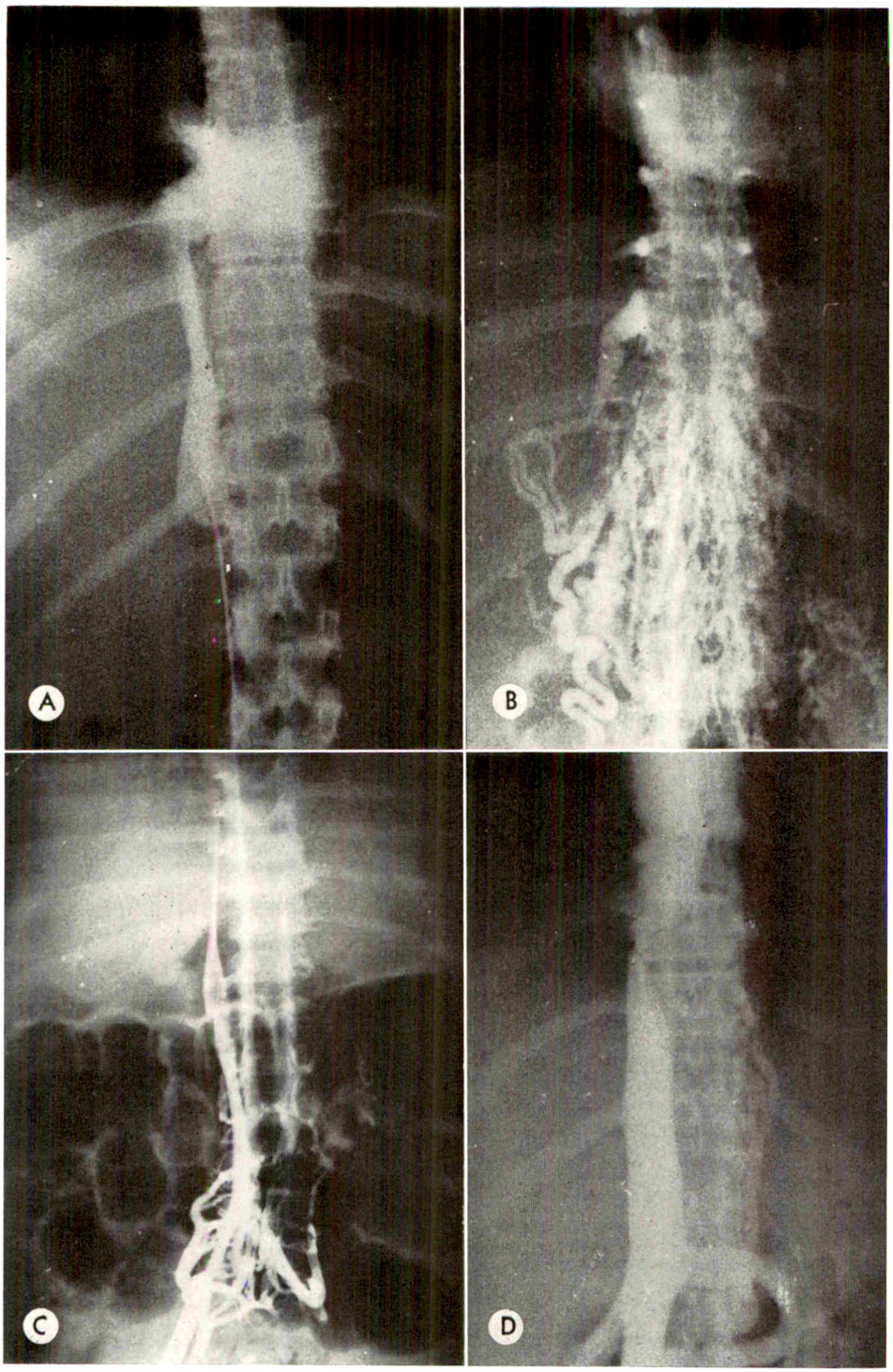


FIG. 1. Inferior venacavograms in different patients with the Budd-Chiari syndrome. (*A*) Compression of the hepatic segment of the inferior vena cava. (*B*) The same patient as in *A* investigated 1 year later shows complete occlusion in the vena cava. (*C*) Extreme compression of the hepatic segment of the inferior vena cava with shunting of the contrast material into the azygos system. (*D*) The venacavogram shows a localized intrinsic filling defect at the level of entry of the hepatic veins.

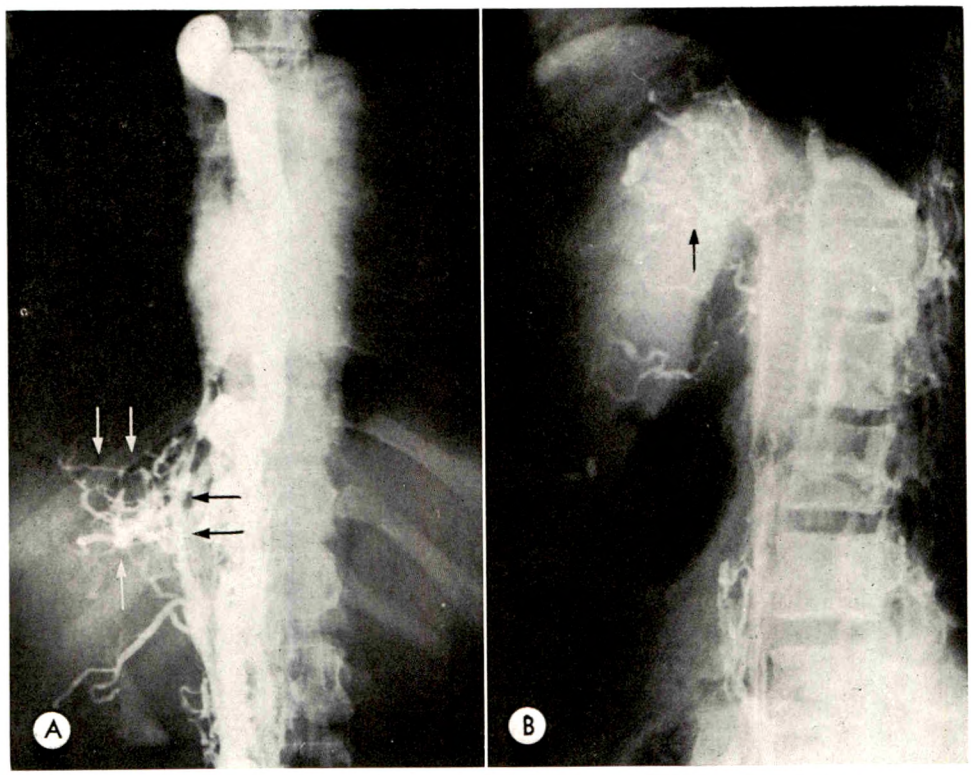


Fig. 2. Inferior venacavograms in the (*A*) frontal and (*B*) lateral projections. The inferior vena cava is completely occluded except the hepatic segment which is extremely narrow with irregular contours (horizontal arrows). Most of the contrast material drains through the azygos system. From the partially patent hepatic segment of the vena cava a coarse network of collaterals fills retrograde into the liver substance (vertical arrows).

ment, which showed an irregular long narrowed channel. This is consistent with either recanalization or with partial thrombosis, the first possibility being more likely. In this case a coarse collateral network was noted filling retrograde from the vena cava into the liver substance (Fig. 2, *A* and *B*).

The fourth pattern, seen in 4 cases, consisted of complete occlusion of the vena cava. In 1 case the contrast material drained through the azygos system into the superior vena cava and through it into the right atrium. A filling defect was suspected in the lower part of the right atrium (Fig. 3*A*). For a more conclusive demonstration, intravenous angiocardiography was performed through the left cubital vein. This investigation clearly demonstrated a large polypoid mass at the distal part of the right atrium above the site of entrance of the inferior vena cava (Fig. 3*B*). The diagnosis of leiomyosarcoma of the inferior vena cava occluding the hepatic veins and extending into the atrium was made and proven at autopsy. A detailed description of this case was published elsewhere.[5]

SELECTIVE HEPATIC VENOGRAPHY

In 2 cases the hepatic veins were successfully catheterized, once via the inferior and once through the superior vena cava. In the first case a spider web network of collaterals was demonstrated around the catheter in the liver substance (Fig. 4*A*). On the serial roentgenograms the contrast material disappeared slowly without filling of the hepatic veins or portal vein. In the second case the hepatic segment of the inferior vena cava was occluded, but the catheter was forced into the right hepatic vein. The

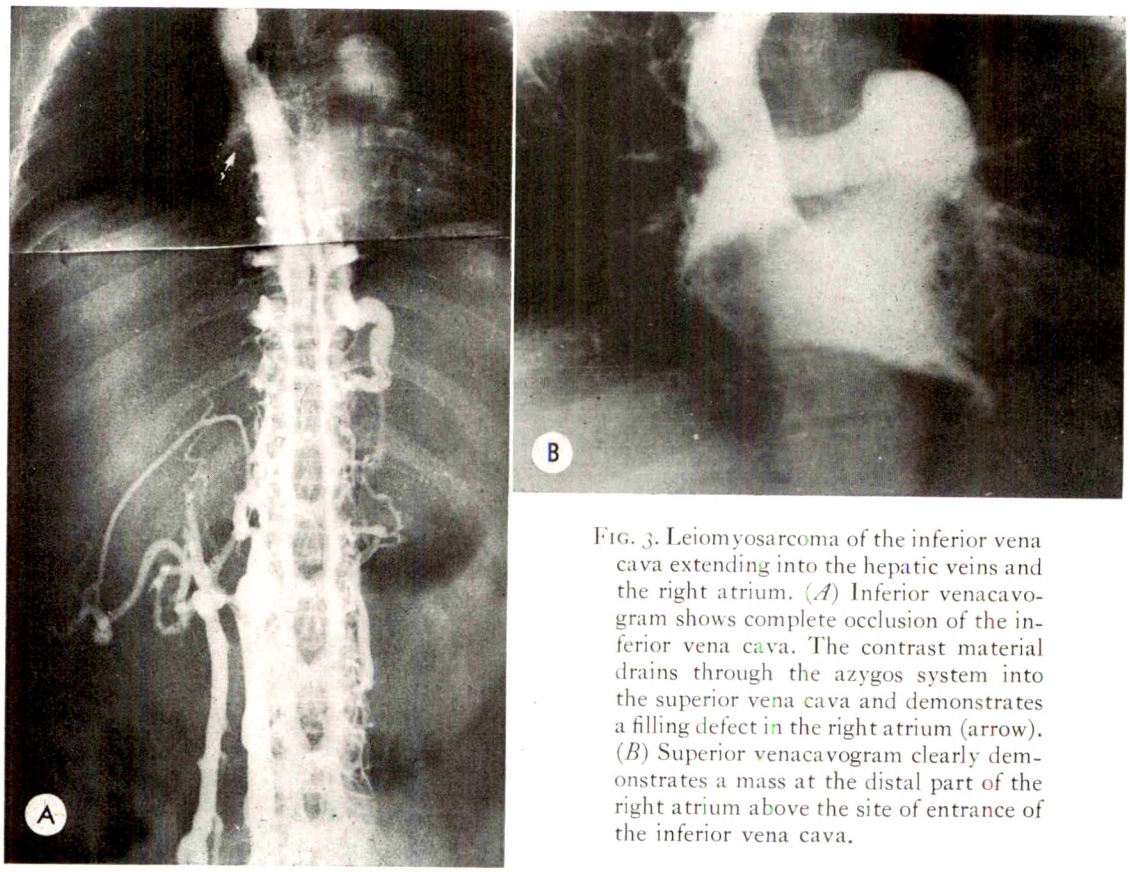


FIG. 3. Leiomyosarcoma of the inferior vena cava extending into the hepatic veins and the right atrium. (*A*) Inferior venacavogram shows complete occlusion of the inferior vena cava. The contrast material drains through the azygos system into the superior vena cava and demonstrates a filling defect in the right atrium (arrow). (*B*) Superior venacavogram clearly demonstrates a mass at the distal part of the right atrium above the site of entrance of the inferior vena cava.

injected contrast material demonstrated a coarser network of collaterals draining into larger branches which subsequently drained through perivertebral venous branches into the azygos system (Fig. 4*B*). In the latter case the pressure measured in the hepatic vein was 18 mm. Hg, while in the right atrium it was 4 mm. Hg.

SELECTIVE CELIAC ARTERIOGRAPHY

Selective celiac arteriography was performed in 3 cases. In 1, selective mesenteric arteriography was also necessary because of the varied origin of the hepatic artery from the superior mesenteric artery. The hepatic arteriogram demonstrated hepatomegaly in all cases. The hepatic arteries (Fig. 5*A*; and 6*A*) were markedly narrowed, stretched and bowed due to swollen liver substance. The splenogram (Fig. 5*B*; and 6*B*) showed a densely opacified enlarged spleen. The splenic and portal veins were patent; however, the hepatopetal flow was decreased and markedly delayed. The main portal vein branches in the liver were attenuated and the secondary branches were not visualized (Fig. 5*B*). In 2 cases, hepatofugal flow into mesenteric veins and retroperitoneal collateral veins was also demonstrated (Fig. 6*B*).

DISCUSSION

The angiographic investigation of a patient with suspected Budd-Chiari syndrome should preceed in stages. The simplest procedure—inferior venacavography—should be performed first. In most cases it is informative and may be diagnostic.

In order to obtain the greatest possible amount of information from this procedure, a large volume of contrast material should be injected at a high pressure, and the superior vena cava with the right atrium should be included in the exposed field.

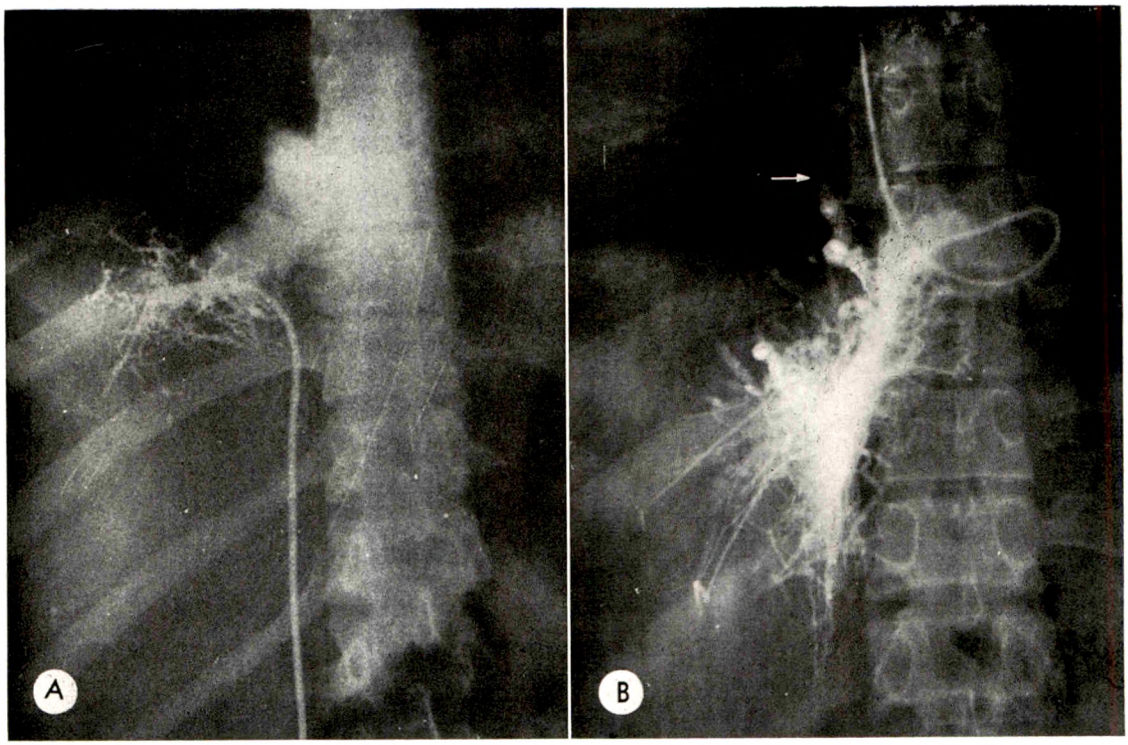


Fig. 4. Selective hepatic venograms in 2 different cases. (*A*) The catheter was introduced into the hepatic vein via the inferior vena cava. The contrast material entered into the intrahepatic venous collaterals, showing a spider web pattern. (*B*) The catheter was forced into a partially occluded hepatic vein. The injected contrast material entered into coarse intrahepatic venous collaterals, and from there into larger collaterals draining into the azygos system (arrow).

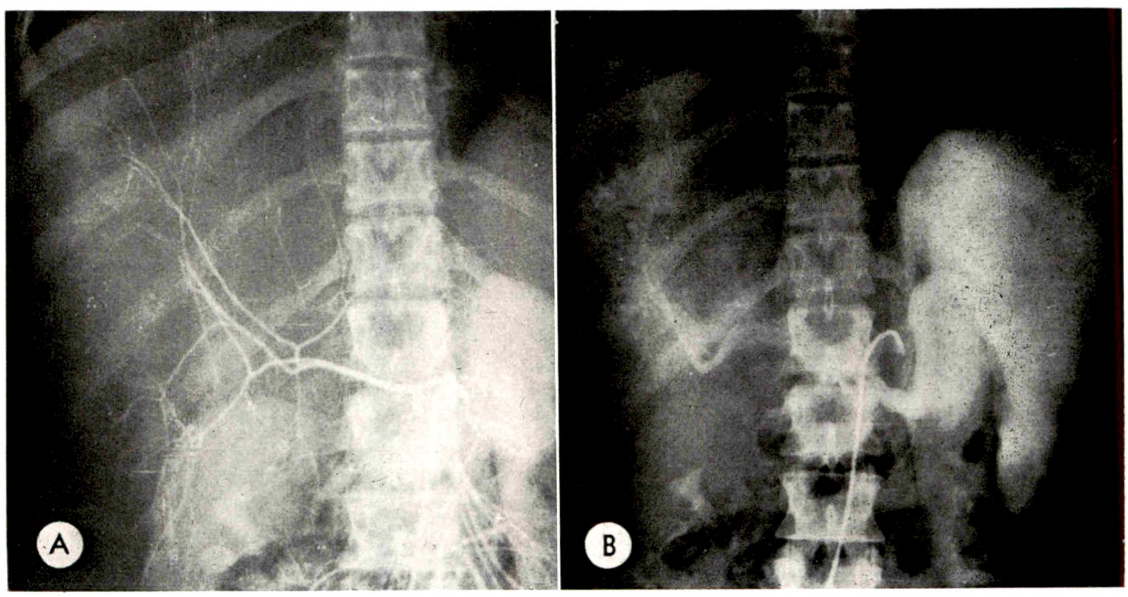


Fig. 5. (*A*) Selective mesenteric arteriogram demonstrating the hepatic artery. The hepatic artery branches are extremely thin and bowed. (*B*) Selective splenic arteriogram, portovenous phase. The portal vein is patent. The portal vein branches in the liver are attenuated due to increased resistance.

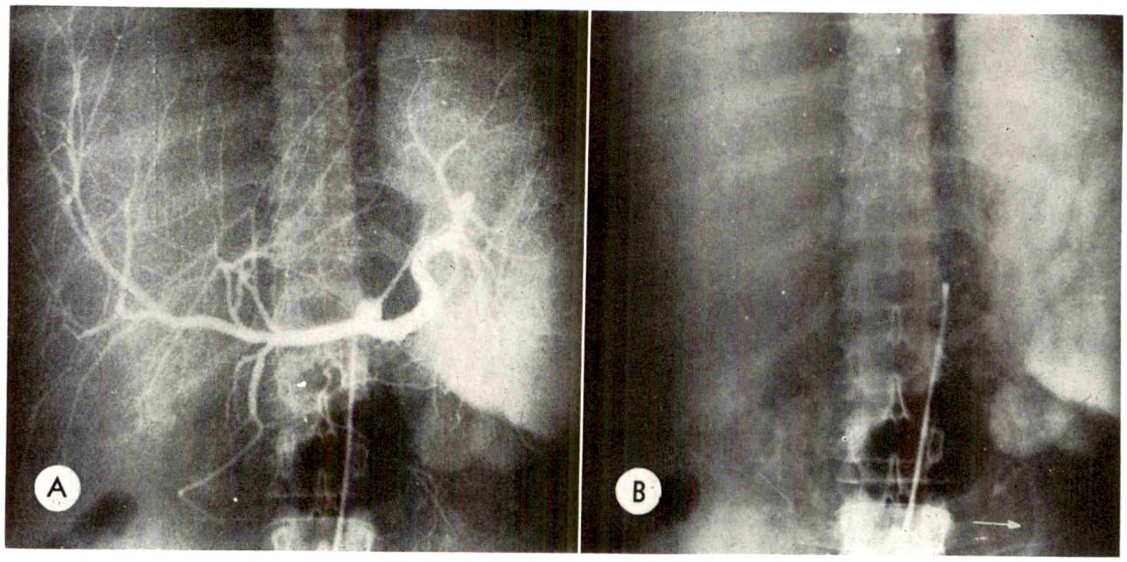


FIG. 6. Selective celiac arteriograms, in the arterial and portovenous phase, respectively. (*A*) The hepatic arteries are thin and stretched. (*B*) The splenic and portal veins are only faintly visualized. Hepatofugal flow into the inferior mesenteric and retroperitoneal collateral veins is seen (arrow).

This will ensure optimal visualization of the collaterals, and the eventual filling through collaterals of the superior vena cava and the right atrium. This in turn will ensure demonstration of any filling defect at the site of the entry of the inferior vena cava into the right atrium.

A further reason for injecting at a high pressure is to obtain absolute differentiation between external narrowing due to compression and actual thrombosis. External compression is seen as a long, smooth string-like narrowing of the lumen. Thrombosis may be complete, or the lumen may be seen narrowed with irregular borders. This situation may represent recanalization or incomplete thrombosis.

Complete occlusion of the hepatic segment of the inferior vena cava has been considered by Levander and Ponten[17] as evidence of Budd-Chiari syndrome. Although theoretically it is possible that the hepatic veins be spared in thrombosis of the hepatic segment of the inferior vena cava, we have not encountered any such case in our material or in the literature.

An irregular intrinsic filling defect of the inferior vena cava at the site of entrance of the hepatic veins can also be considered pathognomonic, as it represents extension of thrombus from the hepatic veins into the inferior vena cava.

Extrinsic compression of the hepatic vena cava, seen as a long narrowing, represents evidence of a swollen liver, but is not pathognomonic of the Budd-Chiari syndrome.

Hepatic venography had been thought previously to be inapplicable in cases of Budd-Chiari syndrome because the hepatic veins were occluded.[30] Recently, however, Kreel *et al.*[16] have used this method successfully in these cases. According to these authors, a typical network of abnormal venous collaterals may be demonstrated in the form of a "spider's web." Occasionally, slightly larger coarser collaterals may be seen.

In our cases both types of collaterals have been noted. The spider web pattern was seen in the case in which the hepatic vein was catheterized via the inferior vena cava (Fig. 4*A*). The coarser network was seen in an unusual case in which the hepatic vein was catheterized via the superior vena cava (Fig. 4*B*), in spite of the fact that the inferior vena cava was completely occluded. The catheter entered the right atrium and

then was apparently forced through a partially occluded or recanalized segment of the hepatic vein. An unusual feature in this case was the demonstration of filling of the azygos system from the hepatic vein through the coarse collateral network. Another interesting feature was the pressure measured in the hepatic vein; *i.e.*, 18 mm. Hg (compared with 4 mm. Hg in the right atrium). This increased pressure may be an expression of the portal hypertension due to a postsinusoidal block, similar to that seen in cirrhosis. An alternative explanation in this specific case may be that the high pressure in the hepatic vein was due to the occluding thrombus in the vena cava, central to the point of measurement.

The third example of hepatic vein collateral visualization, obtained inadvertently at the time of inferior venacavography (Fig. 2, *A* and *B*), also represents an example of the coarse collateral network. The visualization of the collateral pathway in this manner has not been previously described.

Selective hepatic venography is also used in cirrhosis.[36] In this disease the sinusoidal pattern is distorted and fine vascular channels may drain the contrast material beyond the point of wedge injection. In severe cirrhosis these channels drain the contrast material into the portal system. However, these abnormal venous channels are much finer than the spider web or the coarse collaterals seen in the Budd-Chiari syndrome.

The stretching and the bowing of the intrahepatic arteries seen on celiac arteriography cannot be considered absolutely diagnostic; however, these findings do aid in interpretation of the pathologic process occurring in this condition. We observed these changes in all 3 cases in which this procedure was done. We have also noted similar changes in other cases of passive congestion, fatty degeneration and even in some cases of cirrhosis.

Several other features were demonstrated by this procedure in our cases: the spleen was enlarged; the portal vein was patent, but delayed filling was noted in the portal vein. The primary branches of the portal vein were attenuated and the secondary branches were not visualized at all. These features are nonspecific effects of portal hypertension.

Selective hepatic arteriography was not performed in these cases; however, Pollard and Nebesar[24] have shown that with this procedure more specific information may be obtained. In 4 cases of Budd-Chiari syndrome they demonstrated retrograde filling of portal venous branches after selective hepatic artery injection. It appears that the possibility of obtaining this added information justifies the application of this method in cases suspected of having the Budd-Chiari syndrome.

The various possible etiologic factors in our cases also deserve some mention. Trauma has been described as an etiologic factor in the Budd-Chiari syndrome. There have been relatively few reports of such cases. The first report, in 1952, was that of Little and Montgomery[18] whose case presented a short time after receiving a heavy blow to the lower right chest with fracture of the 4th and 5th ribs. Three years later Brink and Botha[2] reported an 18 year old patient who presented with abdominal swelling a few months after receiving a crush injury to the upper right abdomen.

One of our cases was somewhat unusual in that the onset of symptoms was several hours following a friendly blow on the upper abdomen. The patient died 2 weeks following the onset of the disease with extension of the thrombus through the right atrium and ventricle into the pulmonary arteries. The second case had more severe trauma, but the course was prolonged.

The appearance of the Budd-Chiari syndrome following delivery and during puerperium has been described in a number of cases.[3,11,14,15,33] The short time interval between delivery and the onset of the Budd-Chiari syndrome in our 2 cases and in those in the literature suggests that the association is not incidental.

The Budd-Chiari syndrome has also been described in female patients on oral contra-

ceptives. Oral contraceptives are known to cause a hormonal state closely mimicking pregnancy.[4,6,10,13,25,27,31] The common denominator in these 2 conditions appears to be an alteration in the blood clotting factors.[23] In pregnancy there is a rise in factors VII, VIII, X and in fibrinogen, and in puerperium there is a rise in the platelet count with high levels of fibrinogen and factor VIII.[20,32] In females on oral contraceptives, there is a similar rise in coagulation factors, and numerous thrombotic episodes have been described in those who take the "pills,"[7,19 34,35] indicating that hypercoagulability of blood plays an important role in the pathogenesis of the syndrome, as is also suspected in one of our cases.

Of 3 cases with primary Budd-Chiari syndrome and no apparent etiologic factor, 2 were Arab patients, 1 a child of 3 years of age. We have become aware that the Budd-Chiari syndrome is relatively frequent in the Arab people. There is no clear cause for this. Safouh and Shehata[26] reported 59 cases of the Budd-Chiari syndrome in Egyptian children. All were between 1–4 years old and most of them were from low socio-economic groups. It is possible that the leaves of the Senecio plant were responsible for this syndrome in these patients.

Selzer and Parker[29] have shown that diet poor in protein potentiates the effect of the Senecio plant. Gore *et al.*[9] described this syndrome of veno-occlusive disease in Jamaican children. They also connected the disease with bush tea (made from Senecio) and the Senecio plant itself. Hershko *et al.*[12] reported 2 cases of Budd-Chiari syndrome in Arab children in Israel. In these cases there were signs of defibrination. It appears that this type of thrombotic disease is relatively more common in the Arab population. The cause of this is not yet clear, but dietetic factors may be responsible.

SUMMARY

Nine cases of Budd-Chiari syndrome are reported. There were 8 cases of primary hepatic vein thrombosis: 2 following abdominal trauma, 2 following pregnancy, 1 in a patient taking contraceptive pills and 3 without definite etiology. The ninth case was a patient with hepatic vein occlusion, secondary to leiomyosarcoma of the inferior vena cava.

Angiographic patterns seen on venacavography, selective hepatic venography and selective celiac and mesenteric arteriography were helpful in confirming the diagnosis.

Venacavography showed marked compression or complete occlusion of the hepatic segment of the vena cava. In other cases, an irregular circular filling defect at the level of the hepatic veins, or partial occlusion of the hepatic caval lumen with irregular borders were seen.

Selective hepatic venography showed: (1) a "spider web" collateral network; or (2) a coarse collateral network.

Selective celiac and mesenteric arteriography showed stretched and bowed branches of the hepatic artery, and delayed portal venous filling, with collateral circulation.

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REFERENCES

1. BEAIRD, J. B., and SCOFIELD, G. F. Budd-Chiari syndrome: hepatic vein occlusion due to leiomyosarcoma primary in inferior vena cava. *A.M.A. Arch. Int. Med.*, 1962, *110*, 435–441.
2. BRINK, A. J., and BOTHA, D. Budd-Chiari syndrome: diagnosis by hepatic venography. *Brit. J. Radiol.*, 1955, *28*, 330–331.
3. CHIARI, HANS. Über die selbständige Endophlebitis obliterans der Hauptstamme der Venae Hepaticae als Todesursache. *Ziegler's Beitr.*, 1899, *26*, 1–18.
4. CLUBB, A. W., and GILES, C. Budd-Chiari syndrome after oral contraceptives. *Brit. M. J.*, 1968, *1*, 252.
5. DEUTSCH, V., FRANKEL, O., FRAND, U., and HULU, N. Leiomyosarcoma of inferior vena cava propagating into right atrium. *Brit. Heart J.*, 1968, *30*, 571–574.
6. ECKER, J. A., MCKITTRICK, J. E., and FAILING,

R. M. Thrombosis of hepatic veins—Budd-Chiari syndrome–possible link between oral contraceptives and thrombosis formation. *Am. J. Gastroenterol.*, 1966, *45*, 429–443.

7. Egelberg, O., and Owren, P. A. Oral contraceptives and blood coagulability. *Brit. M. J.*, 1963, *1*, 220–221.
8. Erlic, D., Schramek, A., Brandstaetter, S., and Basson, H. Surgical cure of primary hepatic vein occlusion by side to side portocaval shunt. *Surg., Gynec. & Obst.*, 1962, *114*, 368–374.
9. Gore, D. O., Stuart, K. L., and Bras, G. Veno-occlusive disease of liver: surgical aspects. *Surgery*, 1961, *49*, 334–346.
10. Grayson, M. J., and Reilly, M. C. Budd-Chiari syndrome after oral contraceptives. *Brit. M. J.*, 1968, *1*, 512–513.
11. Hanock, K. W. Budd-Chiari syndrome in pregnancy. *J. Obst. & Gynaec. Brit. Empire*, 1968, *75*, 746–748.
12. Hershko, C., Shapira, E., Shvil, Y., Polliack, A., and Russell, A. Budd-Chiari syndrome in childhood. *Israel J. M. Sc.*, 1971, *7*, 592–597.
13. Hoyumpa, A. M., Schiff, L. G., and Helfman, E. L. Budd-Chiari syndrome in women taking oral contraceptives. *Am. J. Med.*, 1971, *50*, 137–140.
14. Kahn, S., and Spring, M. Thrombosis of hepatic veins—Chiari's syndrome: report of case with biopsy and venous pressure determination. *Ann. Int. Med.*, 1940, *14*, 1075–1083.
15. Krass, I. M. Chiari's syndrome: report of case following pregnancy. *J. Obst. & Gynaec. Brit. Empire*, 1957, *64*, 715–719.
16. Kreel, L., Freston, J. W., and Clain, D. Vascular radiology in Budd-Chiari syndrome. *Brit. J. Radiol.*, 1967, *40*, 755–759.
17. Levander, M., and Ponten, J. Budd-Chiari's syndrome diagnosed by means of phlebography in case of retroperitoneal sarcoma. *Acta med. scandinav.*, 1959, *163*, 251–255.
18. Little, R. D., and Montgomery, P. O. Case report of stenosis of vena cava with vena caval and hepatic vein thrombosis related to trauma. *Ann. Int. Med.*, 1952, *37*, 197–203.
19. McIntyre, M., Phillips, M. J., and Voight, J. C. Two cases of thromboembolic disease associated with oral contraceptives. *Brit. M. J.*, 1962, *2*, 1029–1031.
20. Nilsson, I. M., and Kullanders, S. Coagulation and fibrinolytic studies during pregnancy. *Acta obst. & gynaec. scandinav.*, 1967, *46*, 273–285.
21. Palmer, E. D. Budd-Chiari syndrome—occlusion of hepatic veins: seven cases. *Ann. Int. Med.*, 1952, *41*, 261–271.
22. Parker, R. G. Occlusion of hepatic veins in man. *Medicine*, 1959, *38*, 369–397.
23. Pechet, L., and Alexander, B. Increased clotting factors in pregnancy. *New England J. Med.*, 1961, *265*, 1093–1097.
24. Pollard, J. J., and Nebesar, R. A. Altered hemodynamics in Budd-Chiari syndrome demonstrated by selective hepatic and selective splenic angiography. *Radiology*, 1967, *89*, 236–243.
25. Rothwell-Jackson, R. L. Budd-Chiari syndrome after oral contraceptives. *Brit. M. J.*, 1968, *1*, 252.
26. Safouh, M., and Shehata, A. H. Hepatic vein occlusion: disease of Egyptian children. *J. Pediat.*, 1965, *67*, 415–422.
27. Samayajy, B. N., Eeles, G. H., Paton, A., and Parker, R. G. Budd-Chiari syndrome after oral contraceptives. *Brit. M. J.*, 1968, *1*, 53–54.
28. Schaffner, F., Gadboys, M., Safran, A. P., Baron, M. G., and Aufses, A. H. Budd-Chiari syndrome caused by web in inferior vena cava. *Am. J. Med.*, 1967, *42*, 838–843.
29. Selzer, G., and Parker, R. G. Senecio poisoning exhibiting as Chiari's syndrome. *Am. J. Path.*, 1951, *27*, 885–908.
30. Sherlock, S., and Shaldon, S. Selective hepatic phlebography. In: Vascular Roentgenology. Edited by R. A. Schobinger and F. F. Ruzicka. Macmillan and Company, New York, 1964, pp. 567–571.
31. Sterup, K., and Mosbech, J. Budd-Chiari syndrome after taking oral contraceptives. *Brit. M. J.*, 1967, *4*, 660.
32. Talbert, L. M., and Langdell, R. D. Normal values of certain factors in blood clotting mechanism in pregnancy. *Am. J. Obst. & Gynec.*, 1964, *90*, 44–50.
33. Thran, J. H. Über einen Fall von Lebervenen Thrombosis. Inaug. Diss. Kiel, 1899. Cited by Hanock, K. W.[11]
34. Vessey, M. P., and Doll, R. Investigation of relation between use of oral contraceptives and thromboembolic disease. *Brit. M. J.*, 1968, *2*, 199–205.
35. Vessey, M. P., and Doll, R. Investigation of relation between use of oral contraceptives and thromboembolic disease: further report. *Brit. M. J.*, 1969, *2*, 651–657.
36. Viamonte, M., Jr., Warren, D. W., Fernon, J. J., and Martinez, L. O. Angiographic investigations in portal hypertension. *Surg., Gynec. & Obst.*, 1970, *130*, 37–53.

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EDITORIAL

THE ABDOMINAL AORTOGRAM: AN HISTORIC PERSPECTIVE

ARTERIOGRAPHY of the aorta and its branches is a common, frequently used diagnostic technique. Its historic evolution, like other major scientific advances, came about through directed, methodical research coupled at times with serendipity. Since the story of the abdominal aortogram is part and parcel of the lore of diagnostic radiology, it must begin when radiology itself begins: with the production of the first x ray. The story does not, of course, begin in 1895, nor does it initially involve Roentgen.

Professor Arthur Goodspeed of the University of Pennsylvania and a British coworker, using a Crookes' tube, produced an image of coins lying on a photographic plate. This was done in 1890. Unfortunately for Goodspeed's place in history, he made 2 fatal mistakes: he did not recognize the vast medical potential of his discovery, nor did he publish his results. These 2 mistakes were not repeated by Roentgen 5 years later.

It was apparent within days after the presentation of Roentgen's paper that even primitive radiographs were useful in evaluating stones and bones. This in itself was a great medical advance; and no one could reasonably fault those physicians who felt that radiography would be restricted to such investigations. That radiography was not so restricted, however, was due to the imaginative efforts of some early investigators who did not share the prevailing view. We are not certain whether this urge to improve upon a perfectly reasonable diagnostic method was due to man's innate dissatisfaction with imperfection or whether there was more than a little merit in this theory proffered by some nameless editor in the *American Journal of Surgery*, 1904:

> It is now claimed that men who work much with the x-ray become irascible and irritable, not from failure of apparatus to run properly but because of an irritating effect upon the central nervous system. Some very ugly reports are being made as to trophic disturbances also. Amateurs are therefore cautioned against too constant exposure to the fascinating light.[24]

Nevertheless, only a month after the presentation of Roentgen's paper, there were areas of the body not naturally radiopaque being studied; some of the earliest studies involved the vascular tree. In 1896, radiographs of the arteries of a hand were made.[16] Two years later, Codman opacified the aorta and its branches with mercury salts.[6] These early efforts, duplicated and modified by others of the same era, all involved the use of cadavers. The multiminute exposure times of the old gas tubes precluded the study of a dynamic system; the contrast material simply washed away too quickly.

But the sanctity of the living arterial tree was finally violated in 1912, when Bleichroder[4] inserted a catheter into the femoral artery with the idea of perfusing an antiseptic solution directly into the uterine arteries of patients with puerperal sepsis. He used the same technique to insert a balloon-tip catheter into a dog's aorta to make possible bloodless surgery of the leg. Interestingly, Bleichroder had the notion of moistening his catheters with leech extract (heparin), but found it unnecessary. It apparently did not occur to Bleichroder, however, or to his readers, that his technique was adaptable for the injection of contrast media.

Indeed, when living arteries were ini-

tially opacified and radiographed, more than 10 years later, only the segment distal to a needle puncture site was visualized.[2,5] The concept of the intra-arterial catheter had clearly been established, but not yet put to radiologic use.

By 1928, these injections of contrast agents into the living arteries of the extremities were at least provisionally accepted. It was at this time that Reynaldo Dos Santos of Portugal found that accidental puncture of the major vessels of the abdomen had a surprisingly low morbidity. Dos Santos then deliberately injected methylene blue as a vital stain into the aorta via a percutaneous translumbar approach in a patient suffering from tuberculous peritonitis. Fifteen days later, the patient died from respiratory complications of surgery, and Dos Santos was considerably impressed that the aortic puncture site was barely detectable. Consequently, he used the same technique to inject sodium iodide in order to radiologically visualize the aorta and its branches.[8]

Over the next several months, Dos Santos performed aortographies in a large number of patients, initially with a hand injection of 100 per cent sodium iodide, and later using a compressed-air pump injector. It was necessary to perform these studies under general or spinal anesthesia because of the severe pain induced by the contrast agent.

In his paper of 1929, Dos Santos[8] was enthusiastic about the diagnostic credibility of translumbar aortography and more than enthusiastic about its safety. Others, however, were less certain. Over the next several years, objections to the procedure developed on two levels. Subjectively, the very idea of blindly puncturing the aorta with a large-caliber needle and injecting a noxious agent was repugnant. Friedman's *Textbook of Diagnostic Radiology* (1937), found Dos Santos' technique "... an heroic measure and not generally practical."[15] In addition, by 1937, Dos Santos himself had done approximately 1,000 cases, and, despite his continuing vibrant enthusiasm for the procedure, admitted to having serious complications involving the toxicity of sodium iodide, including 4 deaths.[9] Even as late as 1946, Doss,[7] in his review of aortography, found Dos Santos' method to have "... advanced little beyond the field of medical curiosity."

More objective criticisms of Dos Santos' procedure were voiced most conspicuously in a 1936 article by Henline and Moore.[17] In that paper, the 2 authors inferred that Dos Santos actually had 7 deaths among his original 300 patients; they further cited the work of 3 French authors who had 4 life-imperiling complications among only 20 translumbar aortographies. Henline and Moore described their own experience with dogs, in which they used Dos Santos' method to perform translumbar aortography with a variety of contrast agents. In this series, 8 animals of 20 succumbed during or immediately after the procedure.

This paper caused widespread consternation and cast doubt over the validity of Dos Santos' results. Dos Santos himself furnished some vindication of his work by suggesting that dogs' aortas were more susceptible to injury from puncture, and that canine toxicity to sodium iodide exceeded that of man.[18] The influence of Henline and Moore's paper remained considerable for a number of years, however; Lindgren,[19] as late as 1953, felt it necessary to dispute their conclusions, and stated that the complications they described in dogs were unknown in clinical use of aortography. Lindgren was further prompted to comment that some radiologists in 1953 were still using sodium iodide, even though a family of the safer, water-soluble contrast agents had been developed in the decades 1930–1950.

Among those apprehensive of the wisdom of the Dos Santos method of translumbar aortography was Dr. Pedro Fariñas of Havana. In response to the manifold problems involved in the Dos Santos procedure, and armed with the safer water-soluble contrast agent Diodrast, Fariñas chose to discard the needle in favor of the catheter (he was not the first to do so). His 1941 paper[13]

described his new method: surgical exposure of the femoral artery, followed by insertion of a rubber catheter into the aorta, followed by injection of the contrast agent. Using this basic method, Fariñas achieved selective organography, including renal arteriography, by positioning the tip of the catheter at the orifice of the branch artery of the desired organ, placing the patient in the appropriate dependent position, and injecting the heavy contrast agent so that it flowed selectively into the chosen artery.

Thus, Fariñas is widely considered, not without justification, to be the father of catheter arteriography. What is generally not appreciated, however, is that in 2 subsequent papers Fariñas, by inference, abandoned his own catheter technique. In a 1946 paper,[11] Fariñas stated that he was doing retrograde aortographies by puncturing the exposed femoral artery with a needle. He made no mention of a catheter. In that same year, Fariñas in another paper,[12] offered at least a partial explanation of why he chose the needle technique: "In view of the present difficulty, due to war conditions, in obtaining rubber catheters . . . " Ironically, and for whatever reason, Fariñas' abandonment of his catheter technique fulfilled the prophesy voiced at the end of his original 1941 paper by Dr. C. Sidney Burwell, who could not persuade himself of the efficacy of Fariñas' new procedure.

The story of arteriography at this point bears out the old axiom that good things come in threes. Three papers worthy of note appeared in 1949, 3 more in 1951. In that former year, Radner[22] studied the aorta through catheters introduced via left radial artery cutdowns. What was unique in his study was the use of a wire guide to stiffen the catheter, and to make possible more accurate placement of the catheter tip. At roughly the same time, Freeman and his associates[14] assisted those radiologists who preferred aortography by simple percutaneous puncture of the femoral artery. They suggested the use of the Valsalva maneuver to decrease cardiac output in order to make counter-current injection more feasible. Perhaps the most cavalier approach to aortography appeared in the German literature in 1949;[10] in that interesting paper, the author performed esophagoscopy in order to locate the area where the esophagus and aortic arch come into juxtaposition. Dr. Euler then pierced the aorta through the esophageal wall to perform arteriographies.

Of the 3 noteworthy papers of 1951, Peirce's[21] description of a method of percutaneous introduction of a catheter was the most important. He punctured the femoral artery with a large bore (12–15 ga.) needle and through it threaded a polyethylene catheter. This eliminated the need to expose an artery, or repair it after arteriography. Dr. Peirce stated that he used only straight catheters and could not do selective injections. Curved catheters for selective renal arteriography were used in 1951, however. Appropriately-shaped intracardiac catheters were placed in the renal artery through carotid or right brachial artery cut-downs.[3] This method was not widely accepted because of its not inconsiderable morbidity. As was the case in 1949, the third notable paper of 1951 diverged most imaginatively from contemporary radiologic ideology. Dr. Hans Tillander[25,26] developed a catheter with multiple metal segments at the end. The segments were joined with ball-and-socket connections. The last segment was soft iron (gold plated to prevent rusting), and could be guided into place by a large, steerable electromagnet. After using this technique for 5 years, Dr. Tillander remained enthusiastic, despite recognizing that the bulky catheter and unwieldy magnet posed considerable technical difficulties.

By 1953, all the prerequisites for modern arteriography had been met: safe, water-soluble contrast agents were widely available, as were wire guides and suitable catheters. Arteriography was clearly acceptable as worthwhile, even mandatory in some instances. The desirability of the percutaneous approach was generally recognized. What remained was for one man to unite these concepts comprehensively; S. I.

Seldinger was that man.[25] His technique is now so thoroughly familiar that further elaboration here is pointless.

There was at first no great rush to employ Seldinger's method. This was particularly true in the United States, where arteriography was performed much less frequently than in Europe. The concept of selective preformed catheters devised by Ödman in 1956,[20] and Abrams and his coworkers' studies[1] of the effect of epinephrine on renal circulation provided points of impetus towards a more widespread usage.

It was 10–15 years after Seldinger's publication that the initial resistance to routine abdominal arteriography was eroded. These studies are now performed routinely in medical facilities of every dimension.

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REFERENCES

1. Abrams, H. L., Boijsen, E., and Borgström, K. E. Effect of epinephrine on renal circulation: angiographic observations. *Radiology*, 1962, *79*, 911–922.
2. Berberich, J., and Hirsch, S. Die röntgenographische Darstellung der Arterien und Venen am lebenden Menschen. *Klin. Wchnschr.*, 1923, *2*, 2226–2228.
3. Bierman, H. R., Miller E. R., and Byron, R. L., Jr. Intra-arterial catheterization of viscera in man. Am. J. Roentgenol. & Rad. Therapy, 1951, *66*, 555–568.
4. Bleichroder, F. Intraarterielle Therapie. *Berlin. Klin. Wchnschr.*, 1912, *32*, 1503–1505.
5. Brooks, B. Intra-arterial injection of sodium iodide. *J.A.M.A.*, 1924, *82*, 1016–1019.
6. Codman, E. A. Experiments on application of roentgen rays to study of anatomy. *J. Exper. Med.*, 1898, *3*, 383–392.
7. Doss, A. K. Translumbar aortography; its diagnostic value in urology. *J. Urol.*, 1946, *55*, 594–606.
8. Dos Santos, R., Lamas, A., and Saldas, J. Arteriografia da aorta e dos vasos abdominais. *A. Med. Contemp.*, 1929, *47*, 93–97.
9. Dos Santos, R. Technique de l'aortographie. *J. internat. Chir.*, 1937, *2*, 609–634.
10. Euler, H. E. Die peroesophagale Aortenpunktion; ihre diagnostische und terapeutische Möglichkeiten. *Arch. f. Ohren Nasen u. Kehlkopfk.*, 1945, *155*, 536–567.
11. Fariñas, P. L. Retrograde abdominal aortography. *Radiology*, 1946, *47*, 344–348.
12. Fariñas, P. L. Retrograde abdominal aortography. Am. J. Roentgenol. & Rad. Therapy, 1946, *54*, 488–451.
13. Fariñas, P. L. New technique for arteriographic examination of abdominal aorta and its branches. Am. J. Roentgenol. & Rad. Therapy, 1941, *46*, 641–645.
14. Freeman, N. E. Fullenlove, T. M., Wylie, E. J., and Gilfillan, R. S. Valsalva maneuver: aid for contrast visualization of aorta and great vessels. *Ann. Surg.*, 1949, *130*, 398–416.
15. Friedman, L. J. Textbook of Diagnostic Roentgenology. Appleton-Century-Crofts, Inc., New York, 1937, p. 417.
16. Haschek, E., and Lindenthal, O. Ein Beitrag zur praktischen Verwerthung der Photographie nach Röntgen. *Wien. Klin Wchnschr.*, 1896, *9*, 63–64.
17. Henline, R. B., and Moore, S. W. Renal arteriography: preliminary report of experimental study. *Am. J. Surg.*, 1936, *32*, 222–229.
18. Larsson, H., and Palmlöv, A. Abdominal aortography with special reference to its complications. *Acta radiol.*, 1952, *38*, 111–123.
19. Lindgren, E. Technique of abdominal aortography. *Acta radiol.*, 1953, *39*, 205–218.
20. Ödman, P. Percutaneous selective angiography of main branches of aorta. *Acta radiol.*, 1956, *45*, 1–14.
21. Peirce, E. C., II. Percutaneous femoral artery catheterization in man with special reference to aortography. *Surg., Gynec. & Obst.*, 1951, *93*, 56–74.
22. Radner, S. Technical equipment for vasal catheterization. *Acta radiol.*, 1949, *31*, 152–154.
23. Seldinger, S. I. Catheter replacement of needle in percutaneous arteriography: new technique. *Acta radiol.*, 1953, *39*, 368–376.
24. Surgical notes. *Am. J. Surg. & Gynec.*, 1904, *17*, 36.
25. Tillander, H. Magnetic guidance of catheter with articulated steel tip. *Acta radiol.*, 1951, *35*, 62–64.
26. Tillander, H. Selective angiography of abdominal aorta with guided catheter. *Acta radiol.*, 1956, *45*, 21–26.

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INTERNATIONAL COMMISSION ON RADIATION UNITS AND MEASUREMENTS (ICRU)

THE publication of 2 new reports was announced by the International Commission on Radiation Units and Measurements (ICRU). The 2 new reports are: (1) ICRU Report 21, *Radiation Dosimetry: Electrons with Initial Energies Between 1 and 50 MeV;* and (2) ICRU Report 22, *Measurement of Low-Level Radioactivity.*

1. *ICRU Report 21* (available since May 15, 1972) sets out the Commission's recommendations on how the quantities previously defined by the Commission, particularly absorbed dose, can be determined for electrons with initial energies between 1 and 50 MeV. The energy range was chosen to include those radiations produced by linear and circular accelerators which are used for medical, radiochemical and radiobiologic purposes.

The report is primarily concerned with methods which are sufficiently precise and well established to be useful in standardization. However, solid-state dosimetry with thermoluminescent materials is also included, since solid radiation detectors with very small volumes are especially useful as field instruments for practical electron dosimetry.

The report summarizes methods which can be utilized for the measurement and control of the physical properties of the electron beam. Since reliable monitoring is essential in the medical application of electron beams, the report gives recommendations for the construction and use of monitor systems. Although the report does not give extensive information on absorbed dose distributions for clinical application, the general aspects of depth absorbed dose curves, absorbed dose distributions and the influence of inhomogeneities in the irradiated media are discussed in Report 21.

2. *ICRU Report 22* (available since June 1, 1972) describes and discusses many methods of measurement of low-level radioactivity and presents an assessment of the capabilities of various methods. The report includes guidelines developed to assist the reader in choosing those procedures best suited to particular problems.

While a numerical definition of "low-level radioactivity" cannot usefully be given for an unspecified context, it may be said in general that the new report is concerned with procedures that are particularly relevant when achievement of the desired precision of counting is difficult in the counting time available. Report 22 focuses attention on problems of special importance in the assay of low activity samples, especially those encountered in the field of environmental monitoring and in some aspects of medical and biological work. Consideration of the measurement of radioactivity in the living subject, however, is not included in the new report.

The introductory chapter of the report treats extraneous counts, counting statistics, analytical procedures and standardized reference sources. This is followed by five major chapters—sample treatment, assay of gamma- and x-ray emitters, assay of beta-particle emitters, assay of alpha-particle emitters and standardized sources and reference sources.

Individuals and organizations already on the ICRU Publications' Standing Order List will receive copies of the new reports automatically and will be invoiced for the order. Others may purchase copies of the new reports or place their names in the Standing Order List by directing their order or requests to: ICRU Publications, P.O. Box 30165, Washington, D.C. 20014, U.S.A.

For additional information please write to: W. R. Ney, Technical Secretary, International Commission on Radiation Units and Measurements, 7910 Woodmont Avenue, Suite 1016, Washington, D.C. 20014.

NEWS ITEMS

AMERICAN SOCIETY OF NEURORADIOLOGY

The American Society of Neuroradiology announces its 1973 meeting, which will be held at the Statler-Hilton Hotel in Boston, Massachusetts, April 26–28, 1973.

The deadline for abstracts is March 1, 1973. Abstracts of papers to be considered for presentation and requests for information may be sent to the Secretary of the Society, David O. Davis, M.D., Radiology Department, 901 Twenty-third Street, N.W., Washington, D.C. 20037.

In addition, The American Society of Neuroradiology would like to announce that competition is open for its annual prize, the Cornelius G. Dyke Award, to be given annually for the best original paper by trainees in neuroradiology, residents in radiology, or staff radiologists or neuroradiologists through the rank of assistant professor. The Prize is $250.00 and the winner's expenses to the meeting will be paid. The Dyke Award contribution should be well-written, interesting and show some evidence of original research and may be concerned with any aspect of neuroradiology. For further information regarding the Award contact Norman E. Chase, M.D., Professor and Chairman, Department of Radiology, New York University Medical Center, 550 First Avenue, New York, New York 10016.

NEW OFFICERS OF THE ASSOCIATION OF UNIVERSITY RADIOLOGISTS

The newly elected officers of the Association of University Radiologists are: *President*, Elliott Lasser; *President-Elect*, Byron G. Brogdon; and *Secretary-Treasurer*, Melvyn Schreiber, University of Texas Medical Branch, Galveston, Texas 77550.

The officers were selected at the Annual Meeting in Albuquerque, New Mexico.

The 1973 Annual Meeting will be held from May 9 to May 12, 1973 at the University of British Columbia in Vancouver. Program Chairman is J. Scott Dunbar, Department of Diagnostic Radiology, The University of British Columbia, 10th Avenue and Heather Street, Vancouver 9, Canada.

AMERICAN OSTEOPATHIC COLLEGE OF RADIOLOGY

The Annual Meeting and Scientific Sessions of the American Osteopathic College of Radiology will be held at the Regency Hyatt House, Atlanta, Georgia, October 29–November 2, 1972.

For further information please contact Charles Ballinger, D.O., Suite 216, 1550 South Dixie Highway, Coral Gables, Florida 33146.

SIXTH ANNUAL CONFERENCE ON RADIOLOGY IN OTOLARYNGOLOGY AND OPHTHALMOLOGY

University of Illinois at the Medical Center

The Department of Radiology, University of Illinois Hospital, is presenting the Sixth Annual Conference on Radiology in Otolaryngology and Ophthalmology at the Illinois Eye and Ear Infirmary, University of Illinois at the Medical Center, Chicago, Friday and Saturday, November 24 and 25, 1972.

The course, under the guidance of Galdino E. Valvassori, M.D., will include lectures and visual representations by a distinguished faculty. Enrollment is limited.

For further information please write to: Division of University Extension, University of Illinois at the Medical Center, 715 S. Wood Street, Room 302-A, Chicago, Illinois 60612.

THIRD ANNUAL POSTGRADUATE SEMINAR, "RADIOLOGICAL SPECIAL PROCEDURES—BASIC AND NEW INFORMATION"

This Seminar is sponsored by the Department of Radiology at Mount Sinai

Medical Center, in collaboration with the Department of Radiology at Jackson Memorial Hospital, University of Miami School of Medicine, Greater Miami, Florida.

It will be held January 9–13, 1973, at the Playboy Plaza Hotel in Miami Beach, Florida.

For further information, please contact Manuel Viamonte, Jr., M.D., Department of Radiology, Mount Sinai Medical Center, Miami Beach, Florida 33140.

RADIOLOGY SEMINAR IN "SELECTED TOPICS OF CANCER MANAGEMENT"

This Seminar is sponsored by the Division of Radiation Therapy of the Department of Radiology, University of Miami School of Medicine, Jackson Memorial Hospital, in collaboration with the Departments of Radiotherapy at Cedars of Lebanon Hospital and Mount Sinai Hospital, and the Divisions of Radiotherapy at the Veterans Administration Hospital and Mercy Hospital, Greater Miami, Florida.

It will be held January 28–31, 1973, at the Americana Hotel in Miami Beach, Florida.

For further information, please contact Komanduri K. N. Charyulu, M.D., Division of Radiation Therapy, University of Miami School of Medicine, Post Office Box 875, Biscayne Annex, Miami, Florida 33152.

MIDWINTER RADIOLOGICAL CONFERENCE

Los Angeles Radiological Society

The Los Angeles Radiological Society is pleased to announce its Twenty-fifth Annual Midwinter Radiological Conference for 1973. In the past 24 years, this Conference has achieved great popularity among radiologists and other physicians from throughout the United States and many parts of the world because of its varied and pertinent program which includes Lecture Series featuring outstanding speakers, Luncheon Roundtable discussions with the faculty, and a Saturday evening banquet. One thousand registrants attended the 1972 Conference.

For its Silver Anniversary, the Conference will again present a panel of distinguished speakers. Concomitant sessions in diagnostic and therapeutic radiology will be offered for the first time. The 2 day lecture sessions will be preceded by an elective day of small size workshops in nuclear medicine, ultrasonics, and thermography. Technical exhibits will be available on all 3 days.

The Conference will be held February 2–4, 1973, at the Century Plaza Hotel, Los Angeles, California.

Speakers and Workshop Faculty members are: *Diagnosis*, Benjamin Felson, M.D., Cincinnati, Ohio; John F. Holt, M.D., Ann Arbor, Michigan; David Sutton, M.D., London, England; and Manuel Viamonte, Jr., M.D., Miami, Florida. *Therapy*, Henry Kaplan, M.D., Stanford, California; and Philip Rubin, M.D., Rochester, New York. *Nuclear Medicine*, Henry N. Wagner, Jr., M.D., Baltimore, Maryland. *Ultrasonics*, Atis K. Freimanis, M.D., Columbus, Ohio; and George R. Leopold, M.D., San Diego, California.

For advance registration please apply to William Kimball, M.D., 540 North Central Avenue, Glendale, California 91203.

Hotel accommodations should be made with the Convention Manager, Century Plaza Hotel, Los Angeles, California 90067.

EIGHTH ANNUAL SAN FRANCISCO CANCER SYMPOSIUM

Postgraduate Medical Education Course

The Eighth Annual San Francisco Cancer Symposium presented by the Claire Zellerbach Saroni Tumor Institute of Mount Zion Hospital and Medical Center, will be held February 2nd and 3rd, 1973, at the Sir Francis Drake Hotel, San Francisco, California.

The program will be devoted to current concepts and controversies in Oncology, with emphasis on the current application of new diagnostic and therapeutic procedures. The multimodality approach to

cancer diagnosis and therapy will be stressed.

For further information please contact Harry Weinstein, M.D., Director of Medical Education, Department of Continuing Education, Mount Zion Hospital and Medical Center, P.O. Box 7921, San Francisco, California 94120.

ELEVENTH ANNUAL SEMINAR, "SELECTED TOPICS IN GENITO-URINARY ROENTGENOLOGY"

This Seminar will be held March 26–31, 1973 at the Playboy Plaza Hotel in Miami Beach, Florida; it is sponsored by the Department of Radiology, University of Miami School of Medicine at Jackson Memorial Hospital.

For further information, please contact Manuel Viamonte, Jr., M.D., Department of Radiology, University of Miami School of Medicine, Post Office Box 875, Biscayne Annex, Miami, Florida 33152.

NEW DEVELOPMENTS IN RADIOPHARMACEUTICALS AND LABELLED COMPOUNDS

The International Atomic Energy Agency (I.A.E.A.) in collaboration with the World Health Organization (W.H.O.) will hold a Symposium which is primarily concerned with the chemical and pharmaceutical aspects of radiopharmaceuticals, in Copenhagen, Denmark, March 26–30, 1973.

A major objective will be to summarize by means of invited papers some of the recent developments in the areas listed below, to be followed by contributions on new work. Topics to be covered include nuclide generators, kits for preparing radiopharmaceuticals from short-lived nuclides, accelerator production of medically-useful nuclides, synthesis of radiopharmaceutical and labelled compounds from short-lived nuclides, labelled compounds for *in vitro* clinical tests, and stable isotopes in medicine.

A panel will discuss various aspects of regulatory and pharmaceutical responsibilities for radiopharmaceuticals, after which discussion will be invited from the floor. Another panel will attempt to project the directions which nuclear medicine is likely to take in the future, and the demands that this would place on the development of new radiopharmaceuticals.

Participation in the Symposium, whether or not a paper is presented, must be through nomination by the Government of a Member State of the I.A.E.A. or W.H.O. Each participant should complete a Participation Form, and a Form for Submission of a Paper if a paper is to be submitted, and send it (them) to the competent official authority for transmission to the Agency. The Form for submission of a paper should be received by the Agency before October 16, 1972. Further information and forms may be obtained from national authorities for atomic energy matters.

The address of I.A.E.A. is Kärntnerring 11, P.O. Box 590, A-1011 Vienna, Austria; and of the W.H.O., Ch-1211, Geneva 27, Switzerland.

HEALTH PHYSICS SOCIETY
Eighteenth Annual Meeting

The Eighteenth Annual Meeting of the Health Physics Society will be held at the Deauville Hotel, Miami Beach, Florida, June 17–21, 1973.

Information regarding the program may be obtained from the Program Chairman, Stewart C. Bushing, Department of Radiology, Baylor College of Medicine, Houston, Texas 77025.

Information regarding local arrangements may be obtained from the Local Arrangements Chairmen, Donald F. Menker, Department of Radiology, Basic Sciences Building, 1600 N.W. 10th Avenue, Miami, Florida 33136, and Billy G. Dunavant, Nuclear Sciences Building, University of Florida, Gainesville, Florida 32601.

Exhibitor information such as floor plan, size of booth, fee and contract conditions should be obtained from Professional Associates, 2012 Big Bend Boulevard, St. Louis, Missouri 63117.

Forms and instructions for abstracts will be sent to all members in the Society Newsletter or may be obtained from the Program Chairman. Abstracts are requested as early as possible. The deadline is January 15, 1973.

For further information please write to Health Physics Society Executive Secretary: Russell F. Cowing, P.O. Box 156, East Weymouth, Massachusetts 02189.

FOURTH INTERNATIONAL CONGRESS OF LYMPHOLOGY

Under the auspices of the University of Arizona College of Medicine, this Congress will be held March 6–10, 1973, at the Tucson Community Convention Center, Tucson, Arizona.

The format of this meeting will consist of symposia, workshops, free discussion as well as formal presentations and lectures in basic and clinical lymphology—the study of lymph nodes, lymphatics, lymphocytes and lymph in health and disease.

Topics include lymphography and lymphadenography, neoplastic diseases (diagnosis and treatment), basic lymphology (refresher course), lymphedema (pathogenesis, diagnosis and treatment), immunology (the lymphocyte and transplantation), regional lymphatics (liver and gastrointestinal tract, head and neck, peripheral, heart and lung, kidney and endocrine), and techniques in lymphatic surgery.

The meeting will be conducted entirely in English.

Post-meeting tours to Mexico, California and Grand Canyon are being arranged.

A 3 day informal Seminar Course on Advances in Lymphography will be offered by a distinguished guest faculty just prior to the Congress on March 2–4, 1973.

Abstract deadline is November 1, 1972. The final program and list of participants will be mailed shortly thereafter.

For further information please write to Marlys Hearst Witte, M.D., or Charles L. Witte, M.D., Local Organizers for the Fourth International Congress of Lymphology, Arizona Medical Center, Tucson, Arizona 85724.

BOOK REVIEW

The Acute Abdomen in Infancy and Childhood. Edited by John G. Raffensperger, M.D., Associate Professor of Surgery, University of Illinois College of Medicine; Division Head, General Surgery, The Children's Memorial Hospital, Chicago, Ill.; Ruth Andrea Seeler, M.D., Assistant Professor of Pediatrics, University of Illinois College of Medicine; Pediatric Hematologist, Cook County Children's Hospital, Chicago, Ill.; and Rogelio Moncada, M.D., Professor of Radiology, Director of Education, Department of Radiology, Loyola University of Chicago. Cloth. Pp. 130, with many illustrations. Price, $12.00. J. B. Lippincott Company, East Washington Square, Philadelphia, Pa. 19105, 1970.

This book is the result of collaboration of 3 clinical disciplines: surgery, pediatrics and radiology. The result is a very useful survey of the major acute abdominal surgical programs of the pediatric patient. Steps in the diagnosis of causes of the acute abdomen are the pattern of the book; in sequence, for intestinal obstruction, abdominal masses, pneumoperitoneum, neonatal ascites, abdominal trauma, pyloric stenosis, intussusception, foreign substances, trauma, appendicitis, and gastrointestinal bleeding. The final chapters of the book consider some of the medical causes of the acute abdomen under the titles of medical bellyaches, scrotal pain, and miscellaneous abdominal problems.

Although there are only 124 pages of text, the book is generously illustrated by reproductions of roentgenograms, surgical specimens and line drawings. The chapters are referenced with some of the latest articles on each of the subjects mentioned. The importance of having the pediatric patient well attended while in the radiology department and the need for gonadal shielding are emphasized to minimize radiation hazards.

In the chapter on intestinal obstruction in the newborn, much emphasis is placed upon Hirschsprung's disease and the contribution of roentgen-ray in the diagnosis. Pyloric stenosis is given a good roentgen discussion including the string sign, the pyloric beak sign, the shoulder sign and the pyloric tit sign. The discussion of intussusception is complete and the value of barium enema reduction with lesser morbidity is given equal billing with surgical reduction which predisposes to postoperative adhesions. The technique of barium enema reduction is described. The value of abdominal arteriography in abdominal trauma is emphasized.

The authors list 9 findings to support the use of roentgenograms in the confirmation of the diagnosis of appendicitis.

This book, for its size, carries a wealth of information for the general radiologist, the pediatrician and the general surgeon. Trainees in these specialties will find it quick and easy, yet interesting to read and digest.

Lionel W. Young, M.D.

BOOKS RECEIVED

Increased Intracranial Pressure in Children. By William E. Bell, M.D., Professor, Departments of Pediatrics and Neurology; Director, Section of Pediatric Neurology, University of Iowa College of Medicine, Iowa City, Iowa; and William F. McCormick, M.D., Professor, Departments of Neurology and Pathology; Chief, Division of Neuropathology, University of Iowa College of Medicine, Iowa City, Iowa. Volume VIII in the Series: Major Problems in Clinical Pediatrics. Consulting Editor, Alexander J. Schaffer. Cloth. Pp. 260, with 122 illustrations. Price, $16.00. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1972.

Radiation Effect and Tolerance, Normal Tissue: Basic Concepts in Radiation Pathology. Proceedings of the Sixth Annual San Francisco Cancer Symposium. Volume 6 of Frontiers of Radiation Therapy and Oncology. Editor, Jerome M. Vaeth. Cloth. Pp. 529, with 166 figures. Price, $29.50. University Park Press, Chamber of Commerce Building, Baltimore, Md. 21202, 1972.

The Radiologic Clinics of North America. Symposium on Pediatric Radiology. Guest Editor, John L. Gwinn, M.D. August, 1972, Volume X, No. 2. Cloth. Pp. 234, with many illustrations. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1972.

Modern Trends in Radiotherapy—2. Edited by Thomas J. Deeley, M.B., Ch.B., F.F.R., D.M.R.T., Director, South Wales and Monmouthshire Radiotherapy Service, Velindre Hospital and University Hospital of Wales, Cardiff; Lecturer, Welsh Na-

tional School of Medicine, Cardiff, Wales. Cloth. Pp. 315, with some figures. Appleton-Century-Crofts, New York, N. Y., 1972.

The Fundamentals of X-Ray and Radium Physics. By Joseph Selman, M.D., Clinical Assistant Professor of Radiology, University of Texas, Southwestern Medical School; Director, School of Radiologic Technology, Tyler Junior College, Mother Frances Hospital, Medical Center Hospital; Attending Radiologist, Mother Frances Hospital, Medical Center Hospital; Consultant in Radiology, East Texas Chest Hospital, Tyler, Texas. Cloth. Pp. 524, with some figures. Price, $12.95. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62717, 1972.

Pediatric Cardiology. Third edition. By Alexander S. Nadas, M.D., F.A.A.P., Professor of Pediatrics, Children's Hospital, Harvard Medical School; Chief, Cardiology Department, Children's Hospital Medical Center, Boston; and Donald C. Fyler, M.D., F.A.A.P., Associate Professor of Pediatrics, Children's Hospital, Harvard Medical School; Associate Chief, Cardiology Department, Children's Hospital Medical Center, Boston. Cloth. Pp. 749, with 579 illustrations. Price, $25.00. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1972.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga. 30322. Annual Meeting: Washington-Hilton Hotel, Washington, D. C., October 3–6, 1972.

American Radium Society
Secretary, Dr. Felix N. Rutledge, M. D. Anderson Hospital and Tumor Institute, Houston, Texas 77025. Annual Meeting: Broadmoor Hotel, Colorado Springs, Colorado, April 22–26, 1973.

Radiological Society of North America
Secretary, Dr. Hillier L. Baker, Jr., Mayo Clinic, Rochester, Minn. 55901. Annual Meeting: Palmer House, Chicago, Ill., November 26–December 1, 1972.

American College of Radiology
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago, Ill. 60606. Annual meeting: San Francisco, Calif., April 2–7, 1973.

Section on Radiology, American Medical Association
Secretary, Dr. Antolin Raventos, Davis, Calif. Annual Meeting: New York, N. Y., June 24–28, 1973.

American Board of Radiology
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.

Oral examinations will be held in the following cities during the next 2 years: Atlanta, Ga., Dec. 4–8, 1972, Sheraton-Biltmore Hotel; Chicago, Ill., June 4–8, 1973, Marriott Motor Hotel; and Dallas, Tex., Dec. 3–7, 1973, Statler Hilton Hotel.

Written examinations are scheduled in 13 large centers.

Applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.

Deadline for filing applications for any examination in 1973 was September 30, 1972.

American Board of Nuclear Medicine, Inc. A Conjoint Board of the American Boards of Internal Medicine, Pathology and Radiology and sponsored by the Society of Nuclear Medicine.
Chairman, Dr. Merrill A. Bender, Suite 2402, 211 E. 43rd St., New York, N. Y. 10017.

Examination for certification in the Specialty of Nuclear Medicine will be given April 7, 1973. The deadline for filing application forms is January 1, 1973.

American Association of Physicists in Medicine
Secretary, D. John Wright, Ph.D., Department of Radiology, Temple Univ. Health Sciences Center, Philadelphia, Pa. 19140. Annual Meeting in Chicago, Ill., Nov. 30 and Dec. 1, 1972.

American Society of Therapeutic Radiologists
Secretary, Dr. Herman D. Suit, Radiation Medicine, Massachusetts General Hospital, Boston, Mass. 02114. Annual Meeting: Biltmore Hotel, Phoenix, Ariz., November 1–4, 1972.

American Institute of Ultrasound in Medicine
Secretary, John M. Reid, Ph.D., 556 18th Ave., Seattle, Wash. 98122. Annual Meeting, Philadelphia, Pa., Oct. 29–Nov. 1, 1972.

American Society of Neuroradiology
Secretary, Dr. David O. Davis, Department of Radiology, 901 Twenty-third St., N. W., Washington, D. C. 20037. Annual Meeting, Boston, Mass., April 26–28, 1973.

Thirteenth International Congress of Radiology
President-Elect, Dr. Juan Gómez López. *Secretary General*, Dr. José Bonmatí, Lagasca, 27 Madrid 1, España. Meeting: Madrid, Spain, Oct. 15–20, 1973.

International Society of Radiology
Hon. Secretary-Treasurer, Prof. Eric Samuel, Radiologist-in-Charge, The Royal Infirmary, Edinburgh EH3 9Y-W, Scotland.

Eleventh Inter-American Congress of Radiology
Counselor for the United States, Dr. Juan A. del Regato, Penrose Cancer Hospital, 2215 North Cascade Ave., Colorado Springs, Colorado 80907. Meeting: Bogotá, Colombia in 1975.

Inter-American College of Radiology
President, Dr. Miguel Dao y Dao, Apartado Postal 14213, Candelaria, Caracas, Venezuela, S.A.
Secretary General, Dr. Rubén Merenfeld, Instituto Diagnóstico, Apartado Postal 14213, Candelaria, Caracas, Venezuela, S.A. Meeting: Bogotá, Colombia in 1975.

Third Congress of the European Association of Radiology
Meeting in June, 1975 in Edinburgh, Scotland.

Second Asian and Oceanian Congress of Radiology
Honorary Secretary, Dr. J. J. Martin, Box 805 F., G.P.O., Melbourne, 3001, Australia.
Meeting in 1975.

Alabama Chapter of ACR
Secretary-Treasurer, Dr. Lowry R. Young, Jr., 905 Madison St., Huntsville, Ala. 35801. Meets time and place of Alabama State Medical Association.

Alaska Radiological Society
Secretary, Dr. Bruce C. Wright, Providence Hospital, Anchorage, Alaska. Meets third Wednesday each month.

American Thermographic Society
Secretary-Treasurer, Dr. Marc. S. Lapayowker, Department of Radiology, Temple Univ. Hosp., 3401 N. St., Philadelphia, Pa. 19140.

Arizona Radiological Society, Chapter of ACR
Secretary, Dr. Wesley S. Fee, 2421 E. 6th St., Tucson, Ariz. 85719. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

Ark-La-Tex Radiological Society
Secretary, Dr. Erich K. Lang, Confederate Memorial Medical Center, LSU School of Medicine, Shreveport, La. 71101.

Arkansas Chapter of ACR
Secretary-Treasurer, Dr. David H. Newbern, 4301 W. Markham, Little Rock, Ark. 72205. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

Association of University Radiologists
Secretary-Treasurer, Dr. Melvyn H. Schreiber, 2909 Dominique Dr., Galveston, Tex. Annual Meeting, Vancouver, B. C., Canada, May 9–12, 1973.

Atlanta Radiological Society
Secretary-Treasurer, Dr. Ernst Ruder, Dept., of Radiol., Crawford Long Memorial Hosp., Atlanta, Ga. 30308. Meets on four Thursday evenings during the academic year at a time announced in early September of each year, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

Bavarian-American Radiologic Society
Secretary, Lucien F. Burke, Maj. MC, Radiol. Service, US Army Gen. Hosp., Frankfurt, APO, New York 09757.

Blockley Radiological Society
Secretary-Treasurer, Dr. R. John Gould, 41 Lombardy Rd., Drexel Hill, Pa. 19026.

Bluegrass Radiological Society
Secretary-Treasurer, Dr. Eugene Slusher, Lexington

* Secretaries of societies are requested to send timely information promptly to the Editor.

Clinic, 1221 S. Broadway, Lexington, Ky. 40504. The Society meets once each month during the school year.

Bronx Radiological Society, New York State, Chapter ACR
Secretary-Treasurer, Dr. Leon J. Corbin, 1369 Rosedale Ave., Bronx, N. Y. 10472. Meets 4 times a year.

Brooklyn Radiological Society
Secretary-Treasurer, Dr. Sarah Lieberson, 1298 St. Marks Ave., Brooklyn, N. Y. 11213. Meets first Thursday of each month, October through June.

Buffalo Radiological Society
Secretary, Dr. Charles J. Riggio, 5301 Willow Lake Ct., Clarence, N. Y. Meets second Monday evening each month, October to May inclusive, at University Club.

California Radiation Therapy Association
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets semiannually in San Francisco and Los Angeles.

California Radiological Society, California Chapter of ACR
Secretary-Treasurer, Dr. John L. Gwinn, 4650 Sunset Blvd., Los Angeles, Calif. 90027.

Catawba Valley Radiological Society
Secretary, Dr. Emmett R. White, P.O. Box 10, Rutherford College, N. C. 28671. Meets every Thursday, Dept. of Radiology, Valdese General Hosp., Valdese, N. C., at 12:30 P.M.

Central New York Radiological Society
Secretary-Treasurer, Dr. John Sanborn, Radiology Department, Crouse-Irving Memorial Hosp., Irving Ave., Syracuse, N. Y. 13210. Meets first Monday each month October through May.

Central Ohio Radiological Society
Secretary-Treasurer, Dr. James V. Blazek, 2586 Lane Rd., Columbus, Ohio 43220. Meets second Thursday in October, November, January, and March 15, and May 19 at Fort Hayes Hotel, Columbus, Ohio.

Chicago Radiological Society, Division of the Illinois Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Raymond L. Del Fava, 355 Ridge Ave., Evanston, Ill. 60201. Meets third Thursday of each month, October to April, except December, at the Bismarck Hotel, Chicago, Ill.

Cleveland Radiological Society
Secretary-Treasurer, Dr. Daniel E. Wertman, 11311 Shaker Blvd., Cleveland, Ohio 44104. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and April.

Colorado Radiological Society, Chapter of ACR
Secretary, Dr. Jerome D. Sutherland, 3705 E. Colfax Ave., Denver, Colo. 80206. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society
Secretary, Dr. Carl W. Scheer, 335 Cook Ave., Meriden, Conn. 06450. Meets in April and October.

Dallas-Fort Worth Radiological Society
Secretary-Treasurer, Dr. Norman L. Presley, 222 Medical Tower, 1550 W. Rosedale, Fort Worth, Tex. 76104. Meets the 3rd Monday of every month at 6:30 P.M., at the Cibola Inn, Arlington, Tex.

Delaware Chapter of ACR
Secretary, Dr. Seymour R. Kaplan, Kent Gen. Hosp., Dover, Del. 19901.

East Bay Radiological Society
Secretary-Treasurer, Dr. Peter E. Kane, Children's Hosp. Med. Ctr., Oakland, Calif. 94609. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

East Tennessee Radiological Society
Secretary-Treasurer, Dr. George H. Wood, 205 Medical Arts Bldg., Knoxville, Tenn. 37902. Meets in January and September.

Florida Radiological Society, Chapter of ACR
Secretary, Dr. Ralph C. Aye, Tampa General Hospital, Tampa, Fla. 33606. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. J. C. Hewitt, 1 Davis Blvd., Tampa, Fla. 33606. Meets in January, March, May, July, September and November.

Georgia Radiological Society, Chapter of ACR
Secretary, Dr. Richard S. Colvin, Emory University Clinic, Atlanta, Ga. 30322. Meets in spring and fall at Annual State Society Meeting.

Greater Cincinnati Radiological Society
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

Greater Louisville Radiological Society
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

Greater Miami Radiological Society
Secretary-Treasurer, Dr. Stanley I. Worton, 1400 N.W. 10th Avenue, Miami, Florida 33136. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

Greater St. Louis Society of Radiologists
Secretary-Treasurer, John J. Lang, 507 Fairways Circle, St. Louis, Mo. 63141.

Hawaii Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ghim L. Yeoh, 1481 King St., Ste. 202, Honolulu, Hawaii 96814. Meets third Monday of each month at 7:30 P.M.

Health Physics Society
Secretary, John H. Pingel, Argonne National Laboratory, 9700 S. Cass Ave., Argonne, Ill. 60439. Annual Meeting: Deauville Hotel, Miami Beach, Fla., June 17–21, 1973.

Houston Radiological Society
Secretary, Dr. Neill B. Longley, 6436 Fannin, Suite 601, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025.

Idaho State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Hugh P. Smith, Jr., 130 E. Bannock, Boise, Id. 83702. Meets in the spring and fall.

Illinois Radiological Society, Inc., Chapter of ACR
Secretary, Dr. B. Jay Hill, Mercy Hospital, Stevenson at King Drive, Chicago, Ill. 60616. Meets in the spring and fall.

Indiana Roentgen Society, Inc., Chapter of ACR
Secretary, Dr. L. Ray Stewart, 852 S. Alvord Blvd., Evansville, Ind. 47714.

Iowa Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John Huston Jr., 1948 First Ave. N.E., Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ralph H. Baehr, 310 Medical Arts Bldg., Topeka, Kan. 66604. Meets in spring with State Medical Society and in winter on call.

Kentucky Chapter of ACR
Secretary-Treasurer, Dr. Joseph G. Whelan, Jr., 1313 St. Antony Place, Louisville, Ky. 40204. Meets in April and September.

Kings County Radiological Society
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N.Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Knoxville Radiological Society
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Long Island Radiological Society
Secretary, Dr. Jerome Zwanger, 1200 Stewart Ave., Garden City, N. Y. 11533. Meets monthly.

Los Angeles Radiological Society
Secretary, Dr. Mathew E. O'Keefe, 13303 E. Hadley St. Whittier, Calif. 90601. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif. Midwinter Radiological Conference, Century Plaza Hotel, Los Angeles, Calif. 90067, Feb. 2–4, 1973.

Louisiana Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Robert T. Cook, Southern Baptist Hosp., 2700 Napoleon Ave., New Orleans, La. 70115.

Louisiana-Texas Gulf Coast Radiological Society
Secretary-Treasurer, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Tex. 77701.

Maine Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Richard W. Taylor, Radiology Department, St. Mary's General Hospital, Lewiston, Maine 04240. Meets in June, September, December and April.

Maryland Radiological Society, Chapter of ACR
Secretary, Dr. George Burke, 4000 North Charles St., Baltimore, Md. 21218.

Massachusetts Radiological Society, Chapter of ACR
Secretary, Dr. Norman L. Sadowsky, The Faulkner Hosp., 1153 Centre St., Jamaica Plain, Mass. 02130.

Memphis Roentgen Society
Secretary-Treasurer, Dr. Jerry C. Phillips, The University of Tennessee College of Medicine, Department of Radiology, Walter C. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38103. Meets first Monday Oct. through May at the University Club.

Miami Valley Radiological Society
Secretary, Dr. Darwood B. Hance, Reid Memorial Hospital, Richmond, Ind. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

Michigan Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. George A. Kling, Harper Hospital, 3825 Brush St., Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

Mid-Hudson Radiological Society
Secretary-Treasurer, Dr. Herbert S. Berlin, Hopewell Junction, N. Y. Meets 7:00 P.M., first Wednesday of each month, September to May.

Milwaukee Roentgen Ray Society
Secretary-Treasurer, Dr. Thomas C. Lipscomb, 1004 North Tenth St., Milwaukee, Wis. 53233. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Harlan W. Hawkinson, 6 Buffalo Rd., St. Paul, Minn. 55110. Meets twice annually, fall and winter.

Mississippi State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Nancy W. Burrow, 1327 Peachtree St., Jackson, Miss. 39202. Meets third Thursday of each month at the Heidelberg Hotel, Jackson, at 6:00 P.M.

Missouri Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ronald G. Evens, Mallinckrodt Institute of Radiology, St. Louis, Mo. 63110.

Montana Radiological Society, Chapter of ACR
Secretary, Dr. Dexter W. Johnson, Columbus Hosp., Great Falls, Mont. 59401.

Nebraska Chapter of ACR
Secretary-Treasurer, Dr. John T. McGreer, III, 924 Sharp Bldg., Lincoln, Nebr. 68508. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

Nevada Radiological Society, Chapter of ACR
Secretary, Dr. William G. Arbonies, 887 Marsh, Reno, Nev. 89520.

New England Roentgen Ray Society
Secretary, Dr. Stefan C. Schatzki, 1180 Beacon St. Brookline Mass. 02146. Meets the third Friday of each month, October through April, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 4:30 P.M.

New Hampshire Roentgen Ray Society, Chapter of ACR
Secretary-Treasurer, Dr. Edward P. Kane, Claremont Gen. Hosp., Claremont, N. H. 03743. Meets four to six times yearly.

New Mexico Society of Radiologists, Chapter of ACR
Secretary, Dr. Carole R. Simmons, Dept. of Radiol., Presbyterian Hospital Center, Albuquerque, N. M. 87106. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

New Orleans Radiological Society, Inc.
Secretary-Treasurer, Dr. Jimmy L. Mains, P.O. Box 446, Gretna, La. 70053. Meets bimonthly at local restaurants selected by the President.

New York Roentgen Society
Secretary-Treasurer, Dr. Howard R. Gould, 153 West 11th Street, New York, N. Y. 10011. Meets monthly on the 3rd Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Conference, Waldorf-Astoria Hotel, New York, N. Y., Apr. 26–28, 1973. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

New York State Chapter of ACR
Secretary-Treasurer, Dr. John J. Magovern, 74 N. Village Ave., Rockville Centre, N. Y. 11570.

North Carolina Chapter of ACR
Secretary-Treasurer, Dr. James F. Martin, 300 S. Hawthorne Road, Winston-Salem, N. C. 27103.

North Dakota Radiological Society, Chapter of ACR
Secretary, Dr. Marshall Landa, 1702 13th St., So., Fargo, N. D. 58102. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. David F. Bew, University Hospital of Jacksonville, 655 W. Eighth Street, P.O. Box 2751, Jacksonville, Florida 32203. Meets quarterly in March, June, September and December.

Northeastern Oklahoma Radiological Society
Secretary-Treasurer, Dr. Theodore J. Brickner, Jr., 5919 S. Gary Place, Tulsa, Okla. 74110.

Northeastern New York Radiological Society
Secretary, Dr. Robert C. Friedman, 325 Presidential Way, Guilderland, N. Y. 12085. Meets in Albany area on third Wednesday of October, November, March, April, and May.

Northern California Radiation Therapy Association
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets quarterly.

Northern California Radiological Society
Secretary, Dr. Julian B. Holt, 3704 Mission Ave., Suite 26, Carmichael, Calif. 95608. Meets fourth Monday of Sept., Nov., Jan., March and May at Aldo's Restaurant in Sacramento.

Northwestern New York Radiological Society
Secretary, Dr. Barbara E. Chick, Glens Falls Hospital, Glens Falls, N. Y. 12801.

Northwestern Ohio Radiological Society
Secretary, Dr. Vito J. Zupa, Mercy Hospital, Department of Radiology, Toledo, Ohio.

Ohio State Radiological Society, Chapter of ACR
Secretary, Dr. Thomas F. Ulrich, Barberton Citizens Hospital, Barberton, Ohio 44203.

Oklahoma State Radiological Society, Chapter of ACR
Secretary, Dr. George H. Ladd, 4616 Denison, Muskogee, Okla. 74401. Meets in January, May and October.

Orange County Radiological Society
Secretary, Dr. Norman Arnett, 400 North Pamona, Fullerton, Calif. 92632. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at the Orange County Medical Association Bldg., Orange, Calif.

Oregon State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ronald W. Philips, 2305 Washington, Milwaukie, Ore. 97222. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary-Treasurer, Dr. Robert S. Miller, 13753 S.W. Farmington Rd., Beaverton, Oregon 97005. Annual meeting: Benson Hotel, Portland, Ore., May 11–13, 1973.

Pennsylvania Radiological Society, Chapter of ACR
Secretary, Dr. Theodore A. Tristan, Harrisburg Polyclinic Hosp., Harrisburg, Pa. 17105.

Philadelphia Roentgen Ray Society
Secretary, Dr. Howard M. Pollack, Department of Radiology, Episcopal Hospital, Philadelphia, Pa. 19125. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Stephen C. Bruno, Shadyside Hospital, 5230 Centre Ave., Pittsburgh, Pa. 15232. Meets second Wednesday of month, October through June, at Park Schenley Restaurant.

Radiation Research Society
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016. Annual Meeting: St. Louis, Mo., April 29–May 2, 1973.

Radiological Society of Connecticut, Inc., Chapter of ACR
Secretary-Treasurer, Dr. Carl W. Scheer, 335 Cook Ave., Meriden, Conn. 06450. Meetings are held quarterly.

Radiological Society of Greater Cincinnati
Secretary-Treasurer, Dr. Alvin Nathan, c/o Academy of Medicine, 320 Broadway, Cincinnati, Ohio 45202. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of Greater Kansas City
President-Secretary, Dr. Arch W. Templeton, Univ. of Kansas Med. Ctr., Kansas City, Kansas 66103. Meets 5 times a year on given dates.

Radiological Society of Louisiana, Chapter of ACR
Secretary-Treasurer, Dr. Robert T. Cook, 1820 Octavia St., New Orleans, La. 70115. Meets semiannually during Louisiana State Medical Society meeting and 6 months later.

Radiological Society of New Jersey, Chapter of ACR
Secretary, Dr. Sidney Ketyer, St. Elizabeth Hosp., 225 Williamson St., Elizabeth, N. J. 07207. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

Radiological Society of Rhode Island, Chapter of ACR
Secretary-Treasurer, Dr. James B. Leach, Jr., 115 Cass Ave., Woonsocket, R. I. 02895.

Radiological Society of Southern California
Secretary-Treasurer, Dr. Marshall Rowen, Dept. Radiol., St. Joseph Hosp., 1100 Stewart Dr., Orange, Calif. 92668. Meets three times a year, usually October, February and May.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

Redwood Empire Radiological Society
Secretary, Dr. Lee F. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. Donald P. King, 218 Gun Club Rd., Richmond, Va. 23221. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary-Treasurer, Dr. Kenneth E. Robinson, 1441 East Avenue, Rochester, New York 14610. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

Rocky Mountain Radiological Society
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual meeting: Brown Palace Hotel, Denver, Colo., Aug. 1973.

San Antonio-Civilian Military Radiological Society
Secretary, Dr. Robert M. Maurer, Santa Rosa Medical Center, San Antonio, Tex. 78207. Meets third Wednesday of each month at Fort Sam Houston Officers' Club at 6:30 P.M.

San Diego Radiological Society
President-Secretary, Dr. James M. Lee, Scripps Clinic and Research Found., 476 Prospect St., La Jolla, Calif. 92037. Meets first Wednesday of each month at the Town & Country Hotel.

San Francisco Radiological Society
Secretary-Treasurer, Dr. James G. Moore, Mary's Help Hospital, 1900 Sullivan Ave., Daly City, Calif. 94015.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
Secretary-Treasurer, Dr. Louis Wener, Cafritz Memorial Hosp., 1310 Southern Ave., S.E., Washington, D. C. 20032. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section on Radiology, Southern Medical Association
Secretary, Dr. Mario A. Calonje, 1514 Jefferson Highway, New Orleans, La. 70121. Annual Meeting, New Orleans, La., Nov. 13–16, 1972.

Section on Radiology, Texas Medical Association
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

Shreveport Radiological Club
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

Society for Pediatric Radiology
Secretary, Dr. John L. Gwinn, Children's Hospital, 4614 Sunset Blvd., Los Angeles, Calif. 90027. Annual meeting: Washington-Hilton Hotel, Washington, D. C., October 1–2, 1972.

Society of Gastrointestinal Radiologists
Secretary-Treasurer, Dr. Wm. B. Seaman, The Presbyterian Hospital, Columbia-Presbyterian Medical Center, 622 W. 168th St., New York, N. Y. 10032.

Society of Nuclear Medicine
Secretary, Dr. E. James Potchen, Edward Mallinckrodt Institute of Radiology, 660 S. Euclid Ave., St. Louis, Mo. 63110. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. Annual meeting.

South Bay Radiological Society
Secretary, Dr. A. D. Benjamin, 596 Carroll St., Sunnyvale, Calif. 94086. Meets the 2nd Monday of each month at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

South Carolina Radiological Society, Chapter of ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Dakota Radiological Society, Chapter of ACR
Secretary, Dr. M. F. Petereit, 2416 S. Phillips, Sioux Falls, S. D. 57105. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Society
Secretary-Treasurer, Dr. John Byfield, 1000 West Carson St., Torrance, Calif. 90502. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 7544, Mobile, Ala. 36607. Seventeenth Annual Meeting: Grand Hotel, Point Clear, Ala., Jan. 26–28, 1973.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Carl T. Stubblefield, Bedford County Hospital, Columbia, Tenn. 38401. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas State Radiological Society, Chapter of ACR
Secretary, Dr. Herman C. Sehested, 815 Medical Tower, Room 100, 1550 W. Rosedale St., Fort Worth, Tex. 76104. Annual meeting.

The Fleischner Society
President, Dr. Robert G. Fraser, 1555 Summerhill Ave., Montreal 109, P.Q., Canada. Annual meeting.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John F. Stucki, Department of Radiology, McKay-Dee Hosp., 3939 Harrison Blvd., Ogden, Utah 84403. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society, Chapter of ACR
Secretary, Dr. J. Lorimer Holm, R.R. #3, Barre, Vt. 05641.

Virginia Chapter of ACR
Secretary-Treasurer, Dr. James S. Redmond, Suite 7, Medical Center, Lynchburg, Va. 24501. Next Meeting: Nov. 6, 1972 at the Conference Ctr., Williamsburg, Va.

Washington, D. C., Chapter of ACR
Secretary-Treasurer, Dr. Albert M. Zelna, 9110 Seven Locks Rd., Bethesda, Md. 20014.

Washington State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John N. Roehr, 1011 Medical Dental Bldg., Seattle, Wash. 98101.

West Virginia Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Andrew W. Goodwin, II, 200 Med. Arts Bldg., Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society; other meetings arranged by program committee.

Westchester County Radiological Society
Secretary, Dr. Max C. King, 218 Forest Ave., New Rochelle, N. Y. 10804. Meets on second Tuesday of October, December, February and May.

Wisconsin Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Marvin L. Hinke, 630 S. Central Ave., Marshfield, Wisc. 54449. Meets twice a year, May and September.

Wisconsin Society of Therapeutic Radiologists
Secretary, Dr. Robert W. Edland, Gundersen Clinic, LaCrosse, Wisc. 54601. Meets quarterly.

Wyoming Radiological Society, Chapter of ACR
Secretary, Dr. Ross J. Collie, Box 96, Lander, Wyo. 82520. Meets in fall with State Medical Society and in spring on call of President.

Mexico, Puerto Rico and Central America

Asociación Costarricense de Radiología
Secretary, Dr. Jorge Vargas Segura, Apartado 5367, San José, Costa Rica.

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá
Secretary-General, Dr. Francisco Mirambell, Apartado 3352, San José, Costa Rica, Central America.
Meets annually in a rotating manner in the six countries.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad de Radiología de El Salvador
Secretary, Dr. Carlos Mejia, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

Sociedad Mexicana de Radiología, A.C.
Coahuila No. 35, México 7, D.F., México.
Secretary-General, Dr. Jorge Cano Coquí. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. Heriberto Pagán Sáez, Box 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
Moyne, 121 Boul. Taschereau, Greenfield Park, P.Q., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretary, Dr. G. H. du Boulay, 32 Welbeck St., London, W. 1, England. Meets monthly from October until May.

Canadian Association of Physicists, Division of Medical and Biological Physics
Honorary Secretary-Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada.

Canadian Association of Radiologists
Honorary Secretary, Dr. Champlain Charest, 1555 Summerhill Ave., Montreal 109, Que., Canada. Annual meeting: Montreal, Que., Canada, March 11–17, 1973.

Edmonton and District Radiological Society
Secretary, Dr. L. A. Koller, Suite 360, Professional Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada. Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

Faculty of Radiologists
Honorary Secretary, Robert Morrison, 47 Lincoln's Inn Fields, London, W.C.2, England. The scientific meetings held in January, February and March 1973 will be combined sessions with the British Institute of Radiology and the Section of Radiology of The Royal Society of Medicine.

Faculty of Radiologists, Royal College of Surgeons in Ireland
Hon. Secretary, Dr. D. F. Canwell, 123 St. Stephens Green, Dublin 2, Ireland.

Montreal Radiological Study Club
Secretary, Dr. W. Paul Butt, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1, England.

Société Canadienne-Française de Radiologie
Secretary General, Dr. Hubert Sasseville, Verdun Hospital, Montréal, P.Q., Canada. Meets every third Tuesday from October to April.

The Hospital Physicists' Association
Honorary Secretary, J. B. Massey, B.Sc., Christie Hosp. and Holt Radium Institute, Physics Department, Withington, Manchester M20 9BX, England.

Toronto Radiological Society
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September through May.

Royal College of Radiologists of Australasia
Honorary Secretary, Dr. T. P. Loneragan, 147 Macquarie St., Sydney, N.S.W. 2000, Australia.

South America

Colégio Brasileiro de Radiologia
Secretary-General, Dr. Miguel Mario Céntola, Caixa Postal 5984, São Paulo, Brazil.

Federación Argentina de Sociedades de Radiología
Branches of the Federation are: Sociedad de Radiólogos de la Provincia de Córdoba; Sociedad Argentina de Radiología; Asociación Argentina de Radiología; Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario; Sociedad de Radiología y Medicina Nuclear del Centro y Sudeste de la Provincia de Buenos Aires (Mar del Plata); Sociedad Salteña de Radiología y Medicina Nuclear; Sociedad de Radiología de Tucumán; Sociedad de Radiologia del Nordeste Argentino; Sociedad de Radiología de La Plata; and Sociedad de Radiología de San Juan.
Secretary-General-Treasurer, Dr. Roberto Habichayn, Av. Colón 637, Córdoba, Argentina.

Sociedad Argentina de Radiología
Secretary-General, Dr. Armando B. de Onaindia, Santa Fé 1171, Buenos Aires, Argentina. Meets first Wednesday evening, April through December.

Sociedad Boliviana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Armando Rocha Amoédo, Caixa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 p.m. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. Tomás Strauszer, Casilla, 13426 Santiago, Chile. Meets fourth Friday of each month at Av. Santa María at 7:00 p.m.

Sociedad Colombiana de Radiologia
Secretary-General, Dr. Rodrigo Caro Molina, Cali, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología
Secretary, Dr. Carlos Palau Jimenez, Casilla 5469, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Hugo Volpe Rios, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary-General, Dr. Miguel Chiappori Cambana, Colegio Médico del Perú, Casilla Correo, 10307, Lima, Perú. Meets monthly except during January, February, and March.

Sociedad de Radiología del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

Sociedad de Radiología del Nordeste Argentino
Secretary-General, Dr. Francisco J. Velar, Catamarca 561, Corrientes, Argentina.

Sociedad de Radiología de La Plata
Secretary, Dr. Hiram D. Haurigot, Calle 50 No. 374, La Plata, Argentina.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Manuel Gonzáles Maseda, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiología de Pernambuco
Secretary, Dr. Manoel Medeiros Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenología y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Córdoba, Argentina.

Sociedad de Radiología, Radioterapéutica y Medicina Nuclear, de Rosario
Secretary-General, Dr. Federico Frágola, Santa Fe 1798, Rosario, Argentina.

Sociedad Salteña de Radiología y Medicina Nuclear
Secretary, Dr. Julio Luis Baldi, Av. Sarmiento 536, Salta, Argentina.

Sociedad Venezolana de Radiología y Medicina Nuclear
Secretary-General, Dr. Salvador Itriago Borgas, Apartado Postal 9213, Candelaria, Caracas, Venezuela. Meets monthly, third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Röntgen-Gesellschaft
Secretary, Dr. Ernst Kotcher, Wiener Allgemeine Poliklinik Mariannengasse 10, A-1090 Wien, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik. The 1973 Annual Meeting is a joint meeting with the Deutsche Röntgengesellschaft and will be held in Vienna, April 12–14, 1973.

Société Royale Belge de Radiologie
General Secretary, Dr. Joseph Baeyens, Alost, Belgium. Meets in February, March, May, June, September, October, November and December.

Société Européenne de Radiologie Pédiatrique
Secretary, Dr. P. Hicken, The Children's Hosp., Ladywood Middleway, Birmingham B16 8ET, Great Britain. Tenth Annual Meeting, Birmingham, April 11–13, 1973.
Permanent Secretary, Clément Fauré, Hôpital des Enfants Malades, 149, rue de Sèvres, 75 Paris 15e, France.

Société Française de Radiologie Médicale, Médecine Nucléaire et Electrologie, and its branches: Société du Sud-Ouest, du Littoral méditerranéen, du Centre et Lyonnais, du Nord, de l'Ouest, de l'Est, et d'Alger et du Massif Central. Parent Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru Paris 8e, France.

Société Française de Neuroradiologie
Secretary-General, Professeur agr. R. Djindjian 16, rue de l'Université 75, Paris 7e, France. Annual meeting.

Československá Radiologická Společnost
Secretary, Associate Professor Jaromír Kolář, M.D., Sc.D., Radiological Clinic, Praha 2, U nemocnice 2, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

Deutsche Röntgengesellschaft
President, Professor Dr. med. Walter Frommhold, Director of Medizinisches Strahleninstitut der Universität Tübingen, Röntgenweg 11, 7400 Tübingen, Germany. The 1973 Annual Meeting is a joint meeting with the Österreichische Röntgengesellschaft and will be held in Vienna, April 12–14, 1973.

Società Italiana di Radiologia Medica e Medicina Nucleare
Secretary, Prof. Giorgio Cittadini, Ospedale S. Martino, 16132 Genova, Italy. Meets annually.

Societas Radiologica Danica
Secretary, Dr. Bent Langefeldt, Tretommervej 20a, DK-8240 Risskov, Denmark.

Nederlandse Vereniging voor Radiologie
Secretary, Dr. J. D. Mulder, Dept. of Rad., Akademisch Ziekenhuis, Leiden, Netherlands.

Scandinavian Radiological Society
Secretary-General, Professor C. E. Unnérus, M.D., Hagalung-Tapiola, Havsvindsvägen 5 C., Finland.

Sociedad Española de Radiología y Electrología Médicas y de Medicina Nuclear
Secretary, Dr. José Bonmatí, Villanueva, 11, Madrid 1, España. Meets every second Friday of each month, Oct. to June, inclusive, in Madrid.

Sociedade Portuguesa de Radiologia e Medicina Nuclear
Secretary-General, Dr. Luís Aires de Sousa, Av. Elias Garcia, 123- 1, Esg.-Lisboa i-Portugal.

Schweizerische Vereiningung für Radiologie, Nuklearmedizin und Strahlenbiologie (Association Suisse de Radiologie, Médecine Nucléaire et de Radiobiologie)
Secretary, Dr. med. Gustav Schoch, Bethesdaspital, Gellerstrasse 144, 4000 Basel, Switzerland.

Asia

Indian Radiological Association
Secretary, Dr. L. R. Parthasarathy, 44 Constable Road, Madras—23, India.

Indonesian Radiological Society
Secretary, Dr. Gani Iljas Sasmitaatmadja, Radiology Dep., Faculty of Medicine, University of Indonesia, Salemba 6, Djakarta, Indonesia.

Iranian Radiological Society
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ABSTRACTS OF RADIOLOGIC LITERATURE

E. FREDERICK LANG, M.D., Editor

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ROENTGEN DIAGNOSIS

Head

Riggs, B. Lawrence, Randall, Raymond V., Wahner, Heinz W., Jowsey, Jenifer, Kelly, Patrick J., and Singh, Manmohan. The nature of the metabolic bone disorder in acromegaly. *J. Clin. Endocrinol. & Metabol.*, June, 1972, *34*, 911–918. (From: Mayo Clinic and Mayo Foundation, Rochester, Minn. and Michael Reese Hospital and Medical Center, Chicago, Ill.)

Acromegalics commonly show a negative calcium balance, hypercalciuria and occasionally radiolucent vertebrae. Growth hormone results in increased urinary calcium excretion and stimulates skeletal growth.

The authors studied 26 acromegalics, although not all tests were done on all patients. The results of their study showed:

(1) Microradiographic analysis: in cortical rib, bone formation and resorption were both increased and formation was higher than resorption; in bone from the iliac crest the reverse was found. There was a positive correlation between serum growth hormone and bone formation and resorption; trabecular width was increased in acromegalic bone.

(2) Densitometry: measurements of the mid-radius, which is almost all cortical bone, showed a significant increase in bone mass.

(3) Trabecular index: as determined in the upper femora, the acromegalics showed a lower trabecular index than normal.

Features of normal skeletal growth are also characteristic of adults with chronic growth hormone excess, with reorganization of the trabecular bone and radial growth but no linear growth. In the acromegalic spine the vertebrae show increased anteroposterior diameters, presumably normal cortical bone (as in the radius) and trabeculae normal or increased in width; these findings are not found in osteoporosis.

Thus, it is shown that acromegalics do not have osteoporosis. They have high bone formation activity; the cortical bone is normal or increased; the trabecular bone is decreased; and the remodeling characteristics, as noted in (1) above, differ from those in osteoporosis.—*Martha Mottram, M.D.*

Leeds, Norman E., and Rosenblatt, Ruth. Arterial wall irregularities in intracranial neoplasms: the shaggy vessel brought into focus. *Radiology*, Apr., 1972, *103*, 121–124. (From: Departments of Radiology, Montefiore Hospital and Medical Center, and the Albert Einstein College of Medicine, Bronx, N. Y.)

Localized arterial wall irregularity as an indication of tumor was first noted by abdominal angiographers in the examination of patients with pancreatic neoplasms. However, arterial wall abnormalities are seen with greater frequency in intracranial lesions since the advent of direct roentgenographic enlargement angiography. The enhanced resolution, from this technique, permits careful examination of all vascular structures.

The authors report 14 patients with arterial wall irregularities secondary to neoplasms seen from 1968 to 1971. All were examined by biplane serial cerebral angiography and in 6 cases direct serial enlargement angiography was also used.

The various arterial wall abnormalities include: narrowing; irregularity of margins (shaggy vessels); focal areas of dilatation; and a combination of these changes. These abnormalities were noted in astrocytoma, 1 case; glioblastoma multiforme, 3 cases; medulloblastoma, 1 case; metastases, 6 cases; and lymphoma, 3 cases. The vascular irregularities were limited to the area of deformity produced by the neoplasm. Noncontiguous areas of involvement were identified only in patients with lymphomas of the brain, apparently secondary to the diffuseness of the lesion.

An important aspect of the authors' report is that, when arterial wall irregularity occurs, the following lesions must be considered: subarachnoid hemorrhage; inflammatory disease of the brain (meningitis or abscess); arteritis; trauma; intracerebral hematoma; hemorrhagic leukoencephalopathy; cerebral vascular disease; and drug abuse.

Subarachnoid bleeding will result in smooth vasoconstriction with involvement of long or short segments which are occasionally diffuse and often noncontiguous. In purulent meningitis, shaggy vessels may develop as a result of involvement of the blood vessel wall by inflammatory exudate, and in brain abscess there is usually an area of vasoconstriction at the margin of the mass as a result of wall edema.

Intracerebral arteritis may be observed in the following conditions: rheumatoid disease; giant cell arteritis; lupus erythematosus; periarteritis nodosa; and a "nonspecific" arteritis.

The changes in the arterial wall are varied and occur without the presence of a mass. With the other lesions of trauma, intracerebral hemorrhage, and hemorrhagic leukoencephalopathy, changes are similar to those observed with subarachnoid hemorrhage. Cerebrovascular occlusive disease customarily produces diffuse changes, but focal disease may occur rarely. The wall irregularity is discrete, not shaggy and frequently diffuse.

The authors conclude that shaggy vessels are not unique to brain tumors, but, in fact, occur uncommonly, and are more characteristic of arteritis or meningitis. The distinguishing feature of the wall irregularity with tumors is focal involvement of the single vessel or a group of vessels within a mass. Occasionally, noncontiguous vascular involvement occurs when the tumor is diffuse, but the arterial

changes will be seen only within the mass.—*E. B. Best, M.D.*

McCullough, David C., Harbert, John C., Miale, August, Jr., Landis, Glen A., and DeBlanc, Harold J., Jr. Radioisotope cisternography in the evaluation of hydrocephalus in infancy and childhood. *Radiology*, March, 1972, *102*, 645–652. (From: Divisions of Neurosurgery and Nuclear Medicine, Georgetown University Medical Center, Washington, D. C.)

The value of ventriculography and pneumoencephalography for anatomic investigation is well established. Radioisotope cisternography is now the acceptable means for studying the physiology of the cerebrospinal fluid pathway. The latter has been applied mostly to adults, but its use in the study of childhood hydrocephalus is now propounded.

Eighty individual radiocisternographic studies are included: 66 on hydrocephalic patients; and 14 with miscellaneous neurologic problems exclusive of neoplasms and active meningitis. Each patient had at least 1 air study, and several had pre- and postoperative radiocisternographic studies. The patients' ages ranged from 1 week to 12 years.

The technical performance of this study is stressed. The lumbar puncture must be done carefully to avoid extradural extravasation. The radiopharmaceutical is important. High specific activity I^{131} human serum albumin is the current tracer of choice; 2 to 3 μc/kg. is the recommended dose for children, but no less than 25 μc is feasible. No more than 1 mg. of albumin should be injected, and appropriate blockage of the thyroid should be carried out with oral iodides. Tc^{99m} human serum albumin and Yb^{169} DTPA have been used for radiocisternography, but at this time each has certain limitations to routine use. The Anger camera is proposed for imaging children due to the rapidity with which each image can be obtained. The spine should be imaged in the first hour to exclude extradural extravasation, and matched anterior and lateral images of the head should be obtained at 30 minutes, 1 hour, 2 hours and as often thereafter as necessary to record changing cerebrospinal fluid.

The cerebrospinal fluid dynamics in children are accelerated in reference to adults, and the activity normally should approach the area of the sagittal sinus by 12 hours. Even in older children nearly all radioactivity should be concentrated in the sagittal sinus by 24 hours. Delayed tracer clearance is usually indicated, if much cisternal and cervical radioactivity persists at 24 hours, or if the radioactivity has not covered half the convexities by 12 hours. On this basis 5 characteristic patterns are described: (a) no ventricular filling with early clearance (normal); (b) ventricular filling with delayed clearance; (c) ventricular filling with early clearance; (d) no ventricular filling with delayed clearance; and (e) a foramen magnum block. These diverse patterns are discussed in relation to types of hydrocephalus noted on air studies, clinical severity, and presence or absence of surgical cerebrospinal fluid shunts.

Categories of communicating hydrocephalus included patients with progressive hydrocephalus and those with mild, or compensated hydrocephalus. Patients with progressive disease exhibited ventricular filling with delayed clearance prior to surgical shunting. Postshunt patients showed no ventricular filling and either early, or delayed cisternal clearance, unless the shunt was performed late in childhood, or was not functioning properly. In most mild, or compensated cases a normal pattern was found, and in the 1 case of ventricular filling there was rapid clearance.

All patients studied with noncommunicating hydrocephalus had cerebrospinal fluid diversions because of progressive hydrocephalus. Nineteen patients were studied. Sixteen were proven aqueductal atresia on air studies. Preoperative cisternograms were done on 7 of these, and 6 exhibited no ventricular filling, but delayed clearance from the cisterns. Postoperative studies were mostly normal. Two of the 3 remaining patients had blocks at the foramen magnum. The third had a proven Dandy-Walker cyst and, although the system was impermeable to air, the radiopharmaceutical readily penetrated the ventricular system.

Fourteen additional studies on a group of miscellaneous conditions presented either normal studies, transient ventricular filling, or slight delays in clearance.

The apparent aqueductal atresia that results from surgical shunting in cases of communicating hydrocephalus effectively converts the disease to a noncommunicating status, rendering the patient shunt dependent. Apparently, many situations spontaneously compensate, or never become clinically significant. Therefore, the need for radiocisternographic classification of childhood hydrocephalus to differentiate progressive from compensated states, and to determine the appropriate therapeutic approach, seems to be a valid application of the procedure.—*F. C. Petty, M.D.*

Cha, Eung Man, Khoury, George H., and Waly, Farid A. K. Collateral circulation in superior vena caval obstruction following ventriculoatrial shunt catheterization in hydrocephalus. *Radiology*, March, 1972, *102*, 605–611. (From: Department of Radiology and the Division of Pediatric Cardiology, West Virginia University School of Medicine, Morgantown, W. Va.)

This article is a review of 7 cases of ventriculoatrial shunt in which follow-up superior vena cavograms were obtained for the purpose of studying the roent-

genographic anatomy of the collateral circulation of the superior vena cava.

The development of the main collateral circulation in obstruction depends upon the site of the thrombosis in the superior vena cava. The thrombus site is classified as: (a) proximal to the azygos orifice; (b) above the azygos orifice; (c) at the junction of the azygos vein and the superior vena cava; and (d) filling the entire vena cava and the subclavian vein beyond the internal mammary vein.

The thromboses were due to the catheter and produced either a partial or complete obstruction.

Four main collateral channels in superior vena caval obstruction which were previously described by McIntire and Okay are: (1) the azygos route; (2) the vertebral route; (3) the internal mammary route; and (4) the lateral thoracic route.

The authors use excellent reproductions of venograms to demonstrate the detailed roentgenographic anatomy of these main collaterals.—*John G. Hutka, M.D.*

Neck and Chest

Powell, David, Shimkin, Peter M., Doppman, John L., Wells, Sam, Aurbach, Gerald D., Marx, Stephen J., Ketcham, Alfred S., and Potts, John T., Jr. Primary hyperparathyroidism: preoperative tumor localization and differentiation between adenoma and hyperplasia. *New England J. Med.*, June, 1972, *286*, 1169–1175. (Address: David Powell, M.R.C.P., Endocrine Unit, Massachusetts General Hospital, Boston, Mass. 02114.)

Selective blood sampling of small thyroidal veins and radioimmunoassay for parathyroid hormone were utilized for preoperative localization of parathyroid adenomas.

Thirty-three patients were studied. Twenty-one patients with primary hyperparathyroidism diagnosed by this method were confirmed at surgery. Five cases await surgery and 7 cases were considered not primary hyperparathyroidism. Differentiation between single adenomas and their accurate localization (13 cases) and multiple sources (hyperplasia *vs.* multiple adenomas 8 cases) was correct in all 21 cases.

Catheterization and radioimmunoassay techniques are presented.

Further uses of this method to confirm atypical cases of primary hyperthyroidism and atypical locations of adenomas in the mediastinum are suggested. —*Donald P. Harrington, M.D.*

Shimkin, Peter M., Doppman, John L., Powell, David, Marx, Stephen J., and Ketcham, Alfred S. Demonstration of parathyroid adenomas by retrograde thyroid venography. *Radiology*, Apr., 1972, *103*, 63–67. (Address: P. M. Shimkin, M.D., Department of Diagnostic Radiology, Clinical Center, National Institutes of Health, Bethesda, Md.)

Venography was performed in 22 cases of suspected hyperparathyroidism during selective thyroid vein catheterization to obtain blood for determination of parathyroid hormone levels.

Satisfactory venograms were obtained in 16 patients and 4 revealed a discrete contrast blush which correlated well with elevated concentration of parathyroid hormone found in the venous effluent from the blush area and with the size and location of a parathyroid adenoma removed at surgery. Three of the 12 patients with negative venograms had adenomas, and 3 of the 6 patients with inadequate venograms had adenomas.

The best filling of the thyroid venous bed was generally obtained by injecting through the inferior thyroid veins.

Insignificant contrast material extravasation occurred in only 2 instances, otherwise there were no complications.

The causes of inadequate venography are: (1) previous surgical ligation of thyroid veins; and (2) valves in the inferior thyroid veins. High quality venograms are necessary for the demonstration of the tumor blush.

Combined with venous sampling for parathyroid hormone, retrograde venography is a valuable adjunct in localizing parathyroid adenomas.—*William H. Roush, M.D.*

Poe, Robert H., Vassallo, Charles L., Plessinger, Virgil A., and Witt, Richard L. Pulmonary blastomycosis versus carcinoma—a challenging differential. *Am. J. Med. Sc.*, March, 1972, *263*, 145–155. (Address: Robert H. Poe, M.D., Pulmonary Section, Cincinnati Veterans Administration Hospital, 3200 Vine Street, Cincinnati, Ohio 45220.)

North American blastomycosis of the lung is a chronic fungus disease which presents difficult diagnostic problems. The clinical manifestations are often nonspecific, as are the roentgenographic signs. The pulmonary lesions may be mass-like or infiltrative. They may cavitate, and to a variable extent are associated with lymphadenopathy and pleural involvement. They may be associated with osseous or skin involvement, and are often erroneously suspected of being neoplastic.

The clinical and laboratory features are equally misleading. The intradermal skin tests are frequently negative, and the complement fixation tests are also negative quite often. A high index of suspicion and a meticulous sputum collection technique are necessary

for definitive diagnosis. The specimens must be freshly collected, cultured for several weeks, and controlled against bacterial contamination.

A review of the clinical records at several hospitals in Cincinnati over 10 years yielded 25 patients with proven pulmonary blastomycosis. Because of the clinical suspicion of neoplasm, several of these patients underwent major surgical procedures and were only later found to have positive sputum cultures for *Blastomyces dermatitidis*.

Percutaneous needle biopsy of lung lesions was presumably not employed in this patient group.—*Rosalind H. Troupin, M.D.*

Abdomen

Mitchinson, M. J. Some clinical aspects of idiopathic retroperitoneal fibrosis. *Brit. J. Surg.*, Jan., 1972, *59*, 58–60. (From: Department of Pathology, University of Cambridge, Cambridge, England.)

The clinical data on 40 patients with idiopathic retroperitoneal fibrosis are reviewed. Males outnumbered females by 3 to 1.

The presenting symptoms were mainly due to blockage of hollow structures in the retroperitoneum. There was ureteric blockage in all patients, except one who presented with claudication due to narrowing of the aorta, and another who had abdominal distention secondary to narrowing of the colon. Only 4 patients, however, presented with oliguria. The majority had complaints such as: loin pain (16); often radiating to the groin (10); vague abdominal or low back pain (19); vomiting (16); anorexia (7); and considerable weight loss (11). The physical signs were minimal except for hypertension which was present in 17 patients.

Urinary tract infection was present in 5 patients upon presentation; however, another 10 patients became infected after instrumentation. Pyelography showed the typical ureteric obstruction and medial deviation at the level of L-3 or L-4. The erythrocyte sedimentation rate was elevated above 45 mm./hr. in all but 3 of the 31 patients tested. This examination is of value in judging the progression of the disease, especially after treatment.

This disease may extend out of the retroperitoneal area. If this occurs, it has been called "systemic idiopathic fibrosis." Of the 40 patients reported, 4 had involvement of the mediastinum, and 1 of the thyroid gland. This widespread nature, plus occasional associated arthritis, vasculitis, positive L.E. cell prep. and rheumatoid factors, has increased speculation that idiopathic retroperitoneal fibrosis may be a collagen vascular disease. This concept, however, has not been completely supported.

The conventional treatment of ureteric obstruction has been ureterolysis. In his patients, 38 ureterolyses were performed. A success was defined as a functioning kidney on intravenous pyelography with no more than very slight hydronephrosis at least 6 months postoperatively. Surgery was successful in all 18 patients who had hydronephrosis on the initial intravenous pyelogram. Of the 9 with hydronephrosis demonstrated by retrograde pyelography, 5 were designated as a success. There were only 3 successes in the group of 11 patients who presented with a nonfunctioning kidney on intravenous pyelography. Although the short term results are promising, the disease may recur or progress to involve the initially uninvolved side. When ureterolysis is unsuccessful in restoring function, a nephrectomy is required to relieve the hypertension.

Corticosteroids alone or in combination with surgery may have a role in the therapy of idiopathic retroperitoneal fibrosis.—*Frederick Stitik, M.D.*

Eaton, S. Boyd, Jr., Ferrucci, Joseph T., Jr., Benedict, Karl T., Jr., and Margulis, Alexander F. Diagnosis of choledocholithiasis by barium duodenal examination. *Radiology*, Feb., 1972, *102*, 267–273. (Address: S. Boyd Eaton, Jr., M.D., Department of Radiology, Massachusetts General Hospital, Boston, Mass. 02114.)

The most frequent cause of biliary obstruction during adult life sufficient to cause an obstructive jaundice is a calculus of the common bile duct which has become impacted in its distal end. This impacted calculus produces mechanical irritation of the mucous lining of the duct resulting in edema of the duodenal papilla which becomes congested and swollen to a diameter several times that of the impacted stone. While obstruction secondary to neoplastic disease occurs less often, the symptom complexes produced by these 2 conditions can be almost identical and it is felt that all jaundiced patients should undergo roentgen examination of the gastrointestinal tract to try and differentiate between these 2 causes.

The authors have studied 17 patients in whom the roentgen signs of choledocholithiasis were demonstrated preoperatively by conventional upper gastrointestinal barium examination and/or hypotonic duodenography. The size of the normal papilla varies somewhat, but it is felt that any papilla larger than 1.5×1.0 cm. should be viewed with suspicion. The edematous duodenal papilla resulting from an impacted duct stone generally produces a smoothly rounded indentation on the inner duodenal border and this was found in 14 of these 17 patients, while 3 showed irregular margins. In this series the average papillary size was 2.4×2.0 cm., but the dimensions ranged from 4.0×4.0 cm. to 1.3×1.0 cm.

The differential diagnosis of a prominent duodenal papilla includes periampullary carcinoma, papillary edema secondary to peptic ulceration and pancreatitis, leiomyomas and other spindle cell tumors, ectopic pancreas, and unusually large but otherwise normal papilla.

Careful correlation of the clinical features and roentgen findings frequently will permit a definite clinical diagnosis, and because of this, the authors feel that radiologists should participate significantly in the preoperative work-up and diagnosis of choledocholithiasis.—*Donald N. Dysart, M.D.*

Legge, David A., and Carlson, Harley C. Cholangiographic appearance of primary carcinoma of the bile ducts. *Radiology*, Feb., 1972, *102*, 259–266. (From: Mayo Graduate School of Medicine, University of Minnesota, and the Department of Diagnostic Roentgenology, Mayo Clinic and Mayo Foundation, Rochester, Minn.)

All cases of primary carcinoma of the bile ducts seen at the Mayo Clinic in the past 10 years were reviewed and, of these, 32 cases had satisfactory cholangiograms made either *via* percutaneous transhepatic injection or *via* T-tube injection. All 32 cases were either biopsied at surgery or evaluated at autopsy. All of them presented with obstructive jaundice; only 10 complained of abdominal pain.

Of those 11 patients having lesions in the common bile duct, cholangiography in 7 cases showed abrupt occlusion of the common duct with dilatation of both intrahepatic and extrahepatic ducts proximal to the occlusion; 4 of these had abrupt but smooth occlusion and 3 had abrupt and irregular (polypoid) occlusion. Three of the 11 cases had an irregular partial occlusion of a short segment of duct, again with proximal dilatation. One case had irregular narrowing and deformity with poor definition between normal and abnormal segments and with only slight proximal dilatation.

Of those 20 patients having lesions in the porta hepatis, cholangiography showed appearances varying from narrowing to total occlusion of the common hepatic duct and proximal branches. If total occlusion was present, it often involved one of the hepatic ducts so that no contrast material (injected above the occlusion) would enter the other hepatic duct or the common hepatic duct. Contrast material instilled below the lesion would show a normal common bile duct but would only poorly, if at all, fill the biliary tree above the lesion. One case showed smooth, tapered, benign-appearing narrowing of the involved hepatic ducts, but both T-tube and percutaneous transhepatic instillations of contrast material were necessary to visualize both hepatic ducts due to the obstruction.

The one remaining case had a lesion involving both the common bile duct and the porta hepatis. Cholangiography showed diffuse narrowing of hepatic ducts and segmental narrowing of the common bile duct which produced a beaded appearance.

The authors state that a cholangiographic study, showing a short, irregular stenosing lesion with sharply defined proximal and distal edges, is diagnostic of ductal carcinoma as is an occlusive lesion of the duct associated with an irregular polypoid mass within the lumen. With an abrupt but smooth occlusion of the common duct, carcinoma should at least be strongly considered. To determine the full extent of the lesion, all of the biliary tree should be opacified by whatever means necessary.

The differential diagnosis should also include carcinoma of the head of the pancreas or of the ampulla of Vater if the lesion is in the distal portion of the common bile duct. Postoperative stricture should only be considered after reviewing the patient's surgical history to determine that its site correlates with the site of the lesion. Sclerosing cholangitis may produce strictures of varying lengths and be associated with chronic inflammatory disease of the bowel, but 3 cases in this series of carcinomas had chronic inflammatory disease of the bowel.

The authors stress that carcinoma must be considered in all patients in whom cholangiography reveals regions of ductal stenosis or occlusion. Patients with obstructive jaundice should not be considered normal, unless the entire biliary system is opacified. —*L. M. Nikolai, M.D.*

Warshaw, Andrew L., Chesney, Thomas McC., Evans, George W., and McCarthy, Hugh F. Intrasplenic dissection by pancreatic pseudocysts. *New England J. Med.*, July, 1972, *287*, 72–75. (Address: Andrew L. Warshaw, M.D., Massachusetts General Hospital, Boston, Mass. 02114.)

Pancreatic pseudocysts are formed by secretions escaping from the pancreas when it has been disrupted by pancreatitis. Aided by the protolytic enzymes in these secretions, pseudocysts tend to expand and extend along tissue planes, occasionally entering areas as remote from the pancreas as the neck and the scrotum. Since the splenic capsule is continuous with the peritoneum that encloses the splenic vessels and covers the anterior surface of the pancreas, the splenic hilum is also a natural route of spread from pseudocysts in the tail of the pancreas.

The authors report 4 patients, seen within a 6 month period, in whom pancreatic pseudocysts penetrated into the spleen. The expected complications of splenic rupture, splenic infarction, splenic artery hemorrhage, and splenic vein thrombosis were seen in varying combinations.

Radioactive spleen scanning and selective celiac angiography clearly delineated the intrasplenic component of the pseudocysts.

In each of these cases, surgery was successful and included a distal pancreatectomy as well as splenectomy.—*Rosalind H. Troupin, M.D.*

Genitourinary System

Metyš, R., Kňavová, D., and Čekanová, J. Beitrag zur Verbesserung der Infusionsuro-

graphie bei Patienten mit herabgesetzter Nierenfunktion mittels Nephrotomographie. (Contribution by means of nephrotomography to the improvement of infusion pyelography in patients with impaired renal function.) *Röntgenblätter*, Feb., 1972, *25*, 61–66. (Address: Dr. med. René Metyš, Institut für Kreislaufforschung, Prag 4, Budějovická 809, Č.S.S.R.)

In patients with impaired renal function, drip infusion pyelography yields more information than conventional intravenous pyelography.

Further improvement can be obtained by means of 1 to 2 linear tomographic cuts from 7 to 9 cm. from the table top at the end of the infusion.

The advantage of nephrotomography, particularly in patients with impaired renal function, was apparent in a comparison made between a group of 40 patients with impaired renal function (the daily average glomerular filtration was between 6.83 and 41.60 ml./min.) and a group of 20 patients with good or relatively good renal function (glomerular filtration above 50.0 ml./min.) who were subjected to drip infusion pyelography followed by nephrotomography.

In 7 of the 40 patients (17.5 per cent) of the first group, tomography yielded no additional information, in 28 patients (70 per cent) the renal contours were better seen on the tomogram, and in 17 (42.5 per cent) the calyceal system could be better evaluated.

In 9 of the 20 patients (45 per cent) with good or relatively good renal function (second group), nephrotomography had no advantage over the routine infusion pyelography; in addition, the improvement of the visibility of the renal contour and calyceal system was less than in the group with impaired renal function. The value of tomography becomes even more obvious, when the glomerular filtration is compared with the nephrotomograms: the importance of nephrotomography increases with the decrease of the glomerular filtration.—*Peter F. Winter, M.D.*

Secker-Walker, R. H., Shepherd, E. P., and Cassell, K. J. Clinical applications of computer-assisted renography. *J. Nuclear Med.*, April, 1972, *13*, 235–248. (Address: R. H. Secker-Walker, Department of Nuclear Medicine, The Mallinckrodt Institute of Radiology, 510 South Kingshighway, St. Louis, Mo. 63110.)

The renogram is a recording of the changing quantities of tracer hippuran in the nephron and renal pelvis, as well as blood within the kidney, and tissues around the kidney seen by the detector. The activity in the blood and the tissues around the kidney makes up a considerable part of the renogram curve in normal patients. As renal function decreases, this blood and tissue background accounts for even larger portions of the tracing, so that interpretation becomes difficult.

If blood clearance is monitored by a detector placed over the precordium, an indication of over-all renal function is obtained. If I^{131} RISA is administered intravenously prior to the renogram and the precordial and renal activity determined, then an estimate of renal and extrarenal vascular background activity can be made. Using a computer to calculate and subtract the background from the renogram curve, the method of computer assisted blood background subtraction (CABBS) renography (first described by Britton and Brown) was applied to 109 patients.

To perform the study, the kidneys were localized with Hg^{197} chlormerodrin, the blood background measured after 5 microcuries I^{131} RISA, and the renogram recorded after administration of a weight standardized dose of I^{131} hippuran. Summed data from the renogram were recorded every 10 seconds and punched on tape to be later processed by computer.

In addition to subtracting "background" the "efficiency" of hippuran transport was determined by measuring the quantity of hippuran entering the kidney during the first 10 minutes, and comparing it with that leaving the kidney during the 10 minutes after flow starts. Individual renal function, as estimated from areas under the renogram curve, was compared to the number of dots in the renal area on a chlormerodrin scan. The correlation coefficient was 0.96.

CABBS was evaluated in normal patients and in those with hypertension, chronic pyelonephritis, ureteral calculi, chronic obstruction, polycystic kidney disease, renal failure, and a miscellaneous group. There was good correlation between blood clearance of hippuran and creatinine clearance. Also, decreased transport efficiency correlated with increasing blood urea nitrogen. In poorly functioning kidneys, where the conventional renogram shows a flat graph difficult to distinguish from absent function, CABBS will indicate whether function is present, and whether poor function is due to parenchymal disease or obstruction.

CABBS renography, by providing a more physiologically realistic tracing, allows a more accurate determination of relative function than can be obtained from the conventional renogram.—*H. William Strauss, Major USAF, MC*

Shih, Samuel C., Tauxe, W. Newlon, Fairbanks, Virgil F., and Taswell, Howard F. Urinary excretion of ^{51}Cr from labeled erythrocytes: an index of erythrocyte survival. *J.A.M.A.*, May, 1972, *220*, 814–817. (From: Mayo Clinic and Mayo Foundation, Rochester, Minn.)

After intravenous administration of chromium 51 labeled red blood cells, the rate of urinary excretion of the chromium 51 is the function of the rate of destruction of the red blood cells and release of the chromium from the RES. Blood cells lost by hemorrhage do not contribute chromium to the urine. Eighteen patients, who had the following diagnoses were studied: normal; hemolytic anemia; severe blood loss (greater than 50 ml./day); and iron deficiency and chronic blood loss (less than 50 ml./day). Blood was labeled with chromium 51 by standard methods and the cells were washed and resuspended in saline. Separate 24 hour urine collections were obtained over 5 days for urine chromium 51 content; 10 ml. blood samples were drawn each day for 10 days to determine the conventional red blood cell half life; and stool collections were obtained each day for fecal blood loss.

The 3 normal patients had urinary chromium 51 excretion half times of 48, 56, and 48 days (normals from prior studies had a mean half time of 52 days). The red blood cell half life measured 36, 25, and 29 days (normal 28 days). Eight patients with hemolytic anemia had urinary chromium half times of 23 and 42 days. The conventional red blood cell survival gave values of 2 to 20 days. One patient with severe blood loss had a prolonged urinary chromium 51 half time excretion of 65 days, but a blood half life of only 16 days. In 6 patients with chronic iron deficiency, urinary chromium 51 excretion half time was slightly shortened, while the blood survival was also decreased.

The urinary chromium 51 excretion half time represents the biologic half life of the chromium. It has the advantage of requiring only a small dose of radiochromium for labeling, and a very small quantity of blood (1 ml.) for labeling. However, the study requires complete urinary collections over a 5 day period of time.

The urinary excretion of chromium can be used as an index of red blood cell survival, which can separate *in vivo* destruction of blood red cells from hemorrhage.—*H. William Strauss, Major, USAF, MC*

Flower, C. D. R. The role of radiology in the assessment of patients after renal transplantation. *J. Canad. A. Radiologists*, March, 1972, *23*, 10–15. (From: Toronto Western Hospital, Toronto, Ontario, Canada.)

A study of 26 recipients of renal transplants with reference to findings in chest, urinary system and musculoskeletal system revealed that relevant roentgenograms may be of considerable benefit in patient management.

The chest abnormalities may be those associated with rejection, infection, or nonspecific findings due to coexistent pulmonary disease or resulting from complications of a major operation. Rejection manifests itself as pulmonary edema or as the questionable entity of transplant lung. The latter may be a distinct pneumonitis associated with decreasing steroid therapy. Both are associated with typical clinical findings, in the absence of which any pulmonary infiltrate should be considered infectious in origin.

The organisms recovered from lung biopsies or autopsies in 20 patients were *Staphylococcus aureus*, *Diplococcus pneumoniae*, *Hemophilus influenzae*, *Pseudomonas aeruginosa*, *Candida albicans*, *Pneumocystis carinii*, *Aspergillus fumigatus*, and *Cytomegalovirus*.

Chest roentgenograms were suggestive of a specific disease in only 30 per cent of the cases.

Plain film roentgenograms of the kidneys are of limited value. The kidney transplanted into the iliac fossa has little perinephric fat, and size can rarely be assessed unless the poles have been marked with clips, and even these may become displaced.

Renal angiography, performed by catheterization of the iliac artery or, occasionally, the iliac vein, is useful in demonstrating abnormality (stenosis) at the site of anastomosis to renal artery or vein. Selective catheterization is considered inadvisable. Edema, an accompaniment of rejection, causes delayed filling and emptying of the arterial tree. Venous thrombosis usually requires venography for demonstration.

Evidence of renal osteodystrophy, especially in patients receiving hemodialysis, is often present preoperatively but may heal dramatically postoperatively. Avascular osteonecrosis, a complication of steroid therapy, and metastatic calcification and periostitis as a result of chronic renal failure are seen. In 1 patient, the calcifications cleared dramatically a few months after successful transplantation. In another patient, periostitis was seen arising after successful transplantation.—*Lois Cowan Collins, M.D.*

Bookstein, Joseph J., Abrams, Herbert L., Buenger, Richard E., Lecky, James, Franklin, Stanley S., Reiss, Mark D., Bleifer, Kenneth H., Klatte, Eugene C., Varady, Paul D., and Maxwell, Morton H. Radiologic aspects of renovascular hypertension. Part 2: The role of urography in unilateral renovascular disease. *J.A.M.A.*, May, 1972, *220*, 1225–1230. (Address: Joseph J. Bookstein, M.D., University of Michigan Medical Center, Ann Arbor, Mich. 48104.)

An attempt was made to determine the usefulness of urograms in predicting surgical results in unilateral renovascular hypertension. Satisfactory rapid sequence urograms were available in 398 patients with unilateral main renal artery disease, and 259 had reconstructive or ablative surgery. Follow-up in 179 found 138 favorable results and 41 failures.

Major urographic features were disparity in renal length, calyceal appearance time, and concentration on late roentgenograms. With 80 to 99 per cent reduc-

tion in luminal diameter of a renal artery as judged by arteriography, 83 per cent of patients had an abnormal urogram. Delayed appearance time was the most frequent abnormality. With stenosis of 50 per cent or more, this finding was present in 59 per cent of the patients.

Ancillary urographic features (ureteral notching, decreased volume of the collecting system, or spidery calyces) were present in 49 per cent of patients.

In patients with essential hypertension, the urogram was normal in approximately 90 per cent of patients.

Urography alone is a safe, simple and inexpensive screening procedure for unilateral renovascular disease, but there is an incidence of 10 per cent false negative results. The incidence of false positive results is not excessive.

Urography alone did not enable discrimination between favorable and unfavorable responses to surgery.—*Lois Cowan Collins, M.D.*

Nervous System

Houdart, R. Les angiomes de la moëlle et leur traitement chirurgical. (The angiomas of the spinal cord and their surgical treatment.) *Presse méd.*, July, 1971, *79*, 1533–1537. (From: Service de Neuro-Chirurgie, Hôpital Lariboisière, 2, rue Ambroise-Paré, Paris, France.)

Congenital vascular malformations within the spinal cord, or along it, are often little understood and wrongly diagnosed as multiple sclerosis, or myelitis.

Arteriography has greatly elucidated the study of such cases, and enabled the localizations of the lesion (consisting of abnormal arteriovenous shunts) and thereby pointing the way of the surgical approach (if feasible).

In the experience of the author, two-thirds of the 80 cases angiographed (method developed at their hospital by Djindjian) and subsequently treated surgically, showed improvement, and 1 case was cured.—*Jirair N. Sarian, M.D.*

Skeletal System

Schajowicz, F., Cabrini, R. L., and Simes, Y. R. J. Microscopía electrónica del "adamantinoma" de los huesos largos. (Electronic microscopy of adamantinoma of the long bones.) *Rev. Ortop. y Traumat.*, Nov., 1971, *16*, 185–194. (Address: Dr. F. Schajowicz, Centro de Patología Osea, Hospital Italiano, Buenos Aires, Argentina.)

This article describes in detail the histologic characteristics of these tumors in 2 cases studied by the authors. Both patients were young boys, 11 and 16 years old, and in both cases the tumor originated in the tibia.

The authors conclude that these tumors are definitely epithelial in origin. They found certain histologic features that would differentiate these long bone tumors from similar lesions found in the maxillary bones.—*Julian B. Alvarez, M.D.*

Wilppula, E., and Vahvanen, V. Chondromalacia of the patella: a follow-up study of 25 cases treated with chondrectomy. *Acta orthop. scandinav.*, 1971, *42*, 521–527. (From: Department of Orthopaedics and Traumatology, University Central Hospital, Helsinki, Finland.)

The authors discuss the clinical findings in 35 patients with chondromalacia of the patella and report a follow-up study of 25 of these cases, who were followed for a mean of 6.2 years after being treated with chrondrectomy.

Twenty-one of 35 patients give the history of some kind of injury to the knee joint. A previous tendency to luxation of the patella was observed in 2 patients. The average duration of clinical symptoms prior to operation was 3 years. Most patients experienced pain while walking. Compression of the patella against the femur was painful in 19 patients. Crepitation was noted on compression of the patella in 30 of the patients and not recorded in 5.

The preoperative roentgenographic findings were normal in 15 patients; unevenness of the under surface of the patella was observed in 3 cases. The remaining patients had other changes of osteoarthritis.

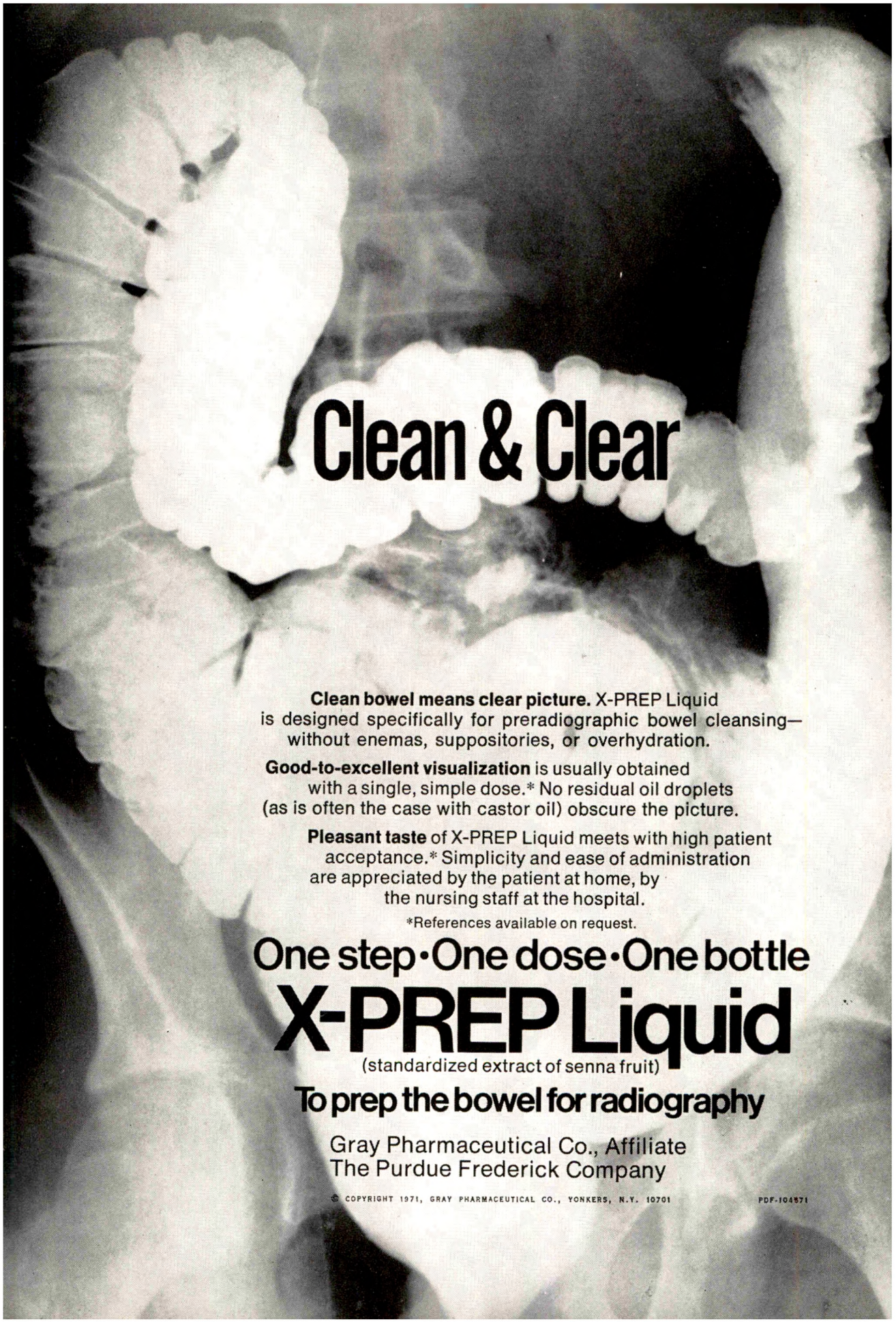
The patients were treated by shaving of the patellar cartilage. Patellectomy was not performed. At the time of operation meniscal rupture was noted to be present in 12 of 35 patients. The authors, therefore, feel that preoperative arthrography would be important in the work-up of these patients.

The clinical results of chondrectomy in patients with chondromalacia of the patella was considered good in 13 cases, fair in 8 and poor in 4.—*Eugene C. Klatte, M.D.*

van Andel, J. G. Synovial sarcoma: a review and analysis of treated cases. *Radiol. clin. et biol.*, 1972, *41*, 145–159. (Address: Consultant Radiotherapist, Rotterdamsch Radio-Therapeutisch Instituut, Groene Hilledijk 297, Rotterdam-Zuid, The Netherlands.)

The author reports on the results in 28 patients with synovial sarcoma treated at the Rotterdam Radiotherapy Institute in Rotterdam, Netherlands.

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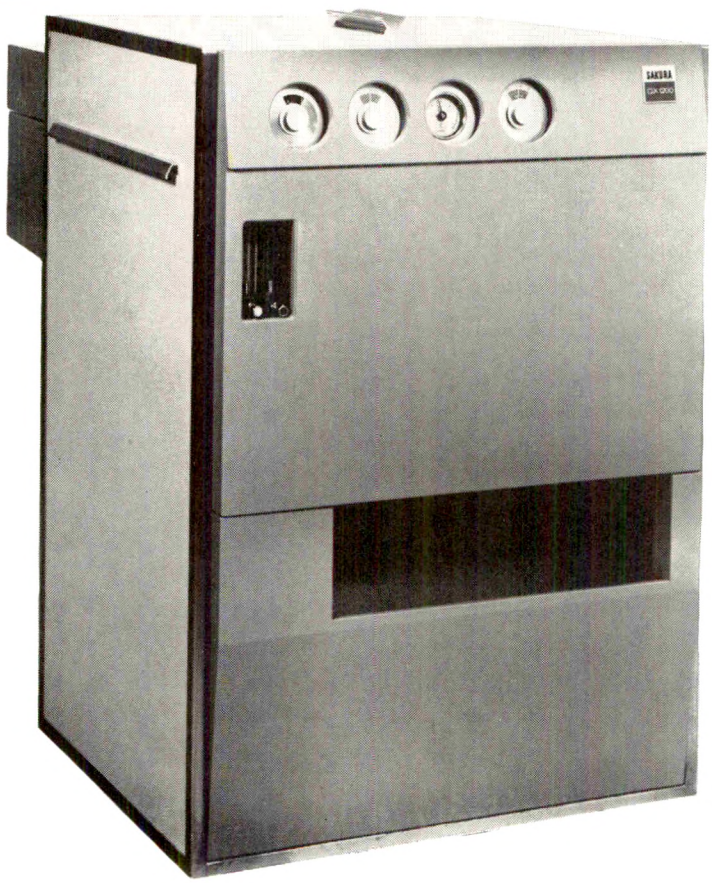
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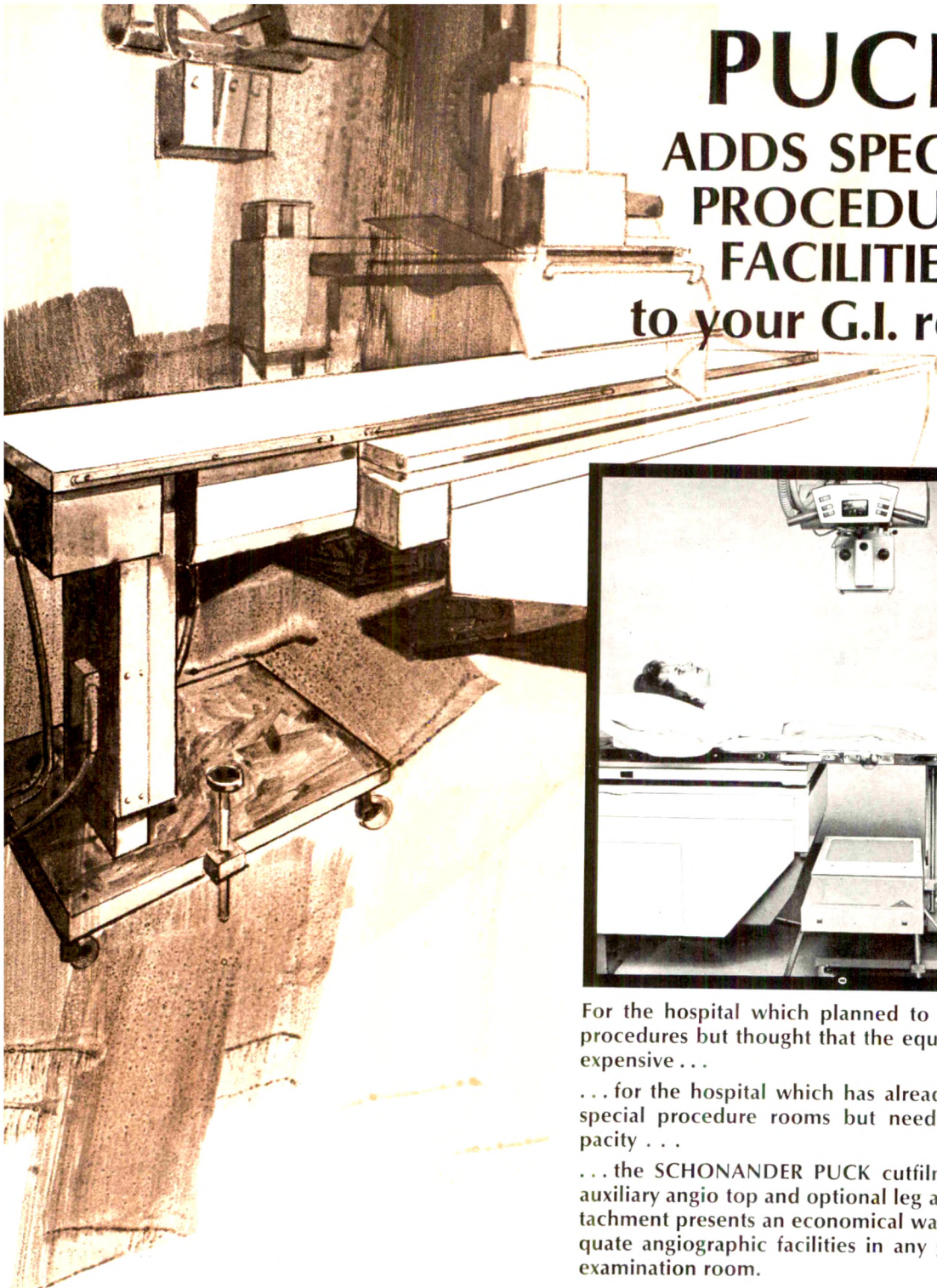
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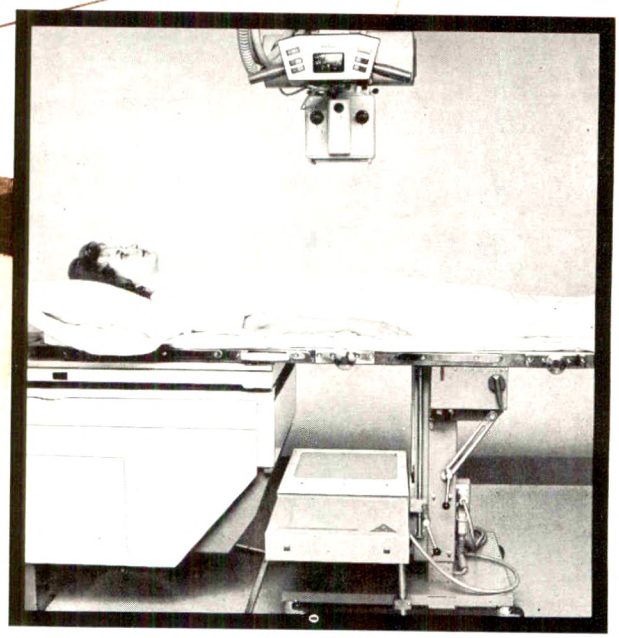
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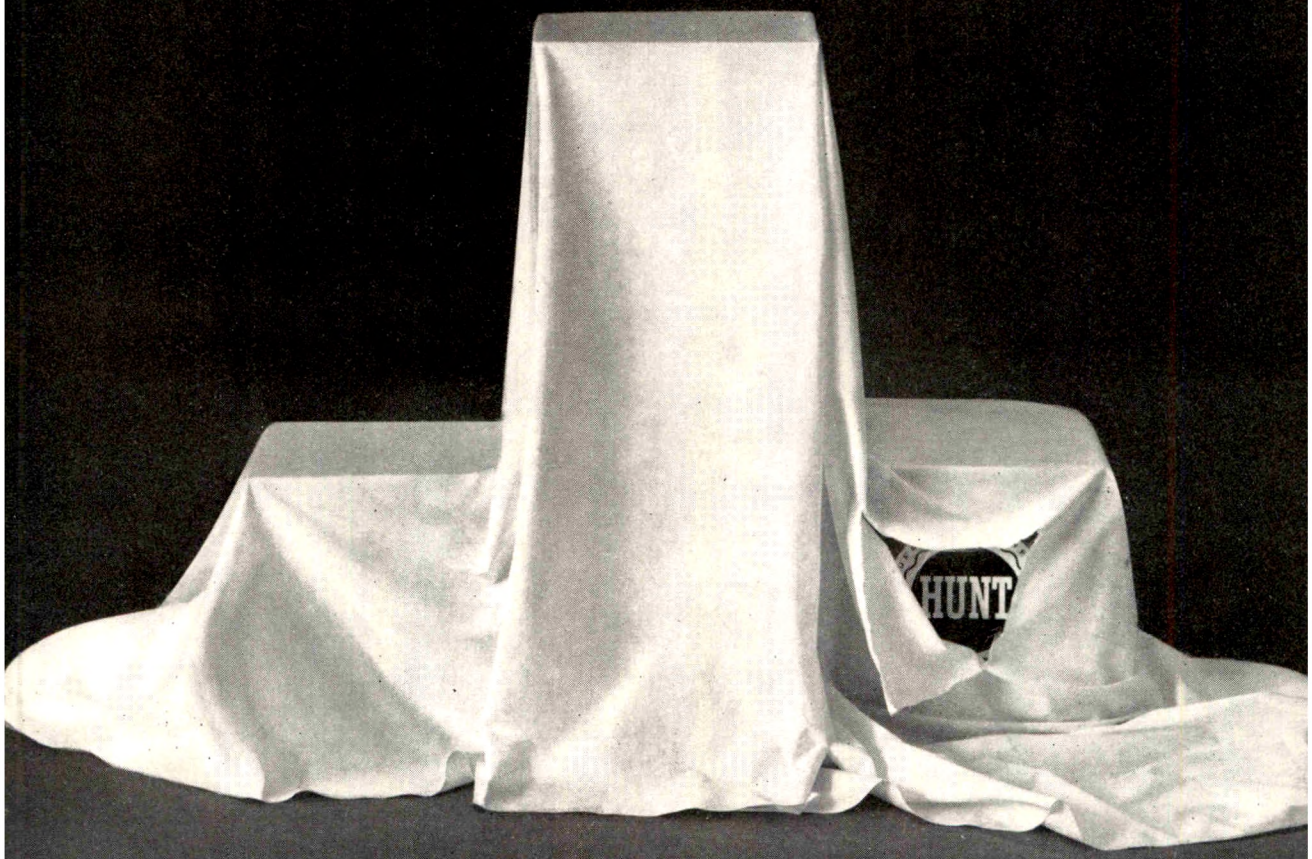
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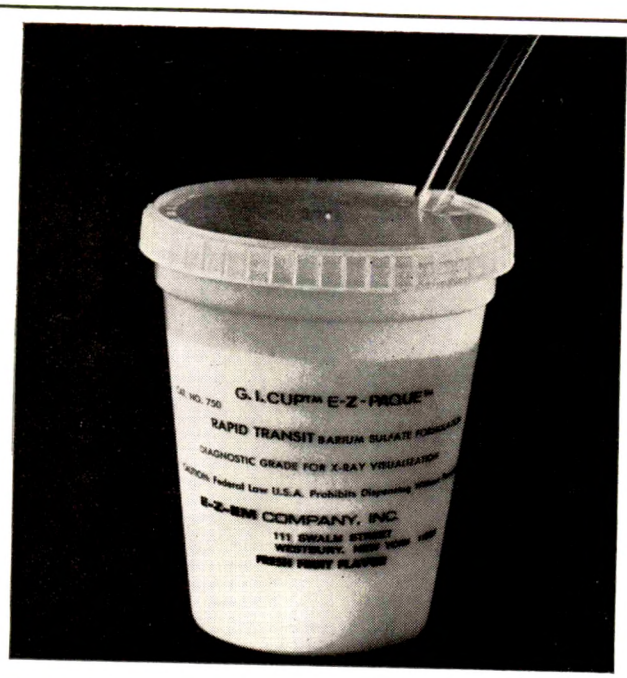
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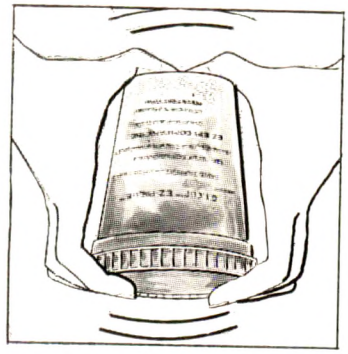
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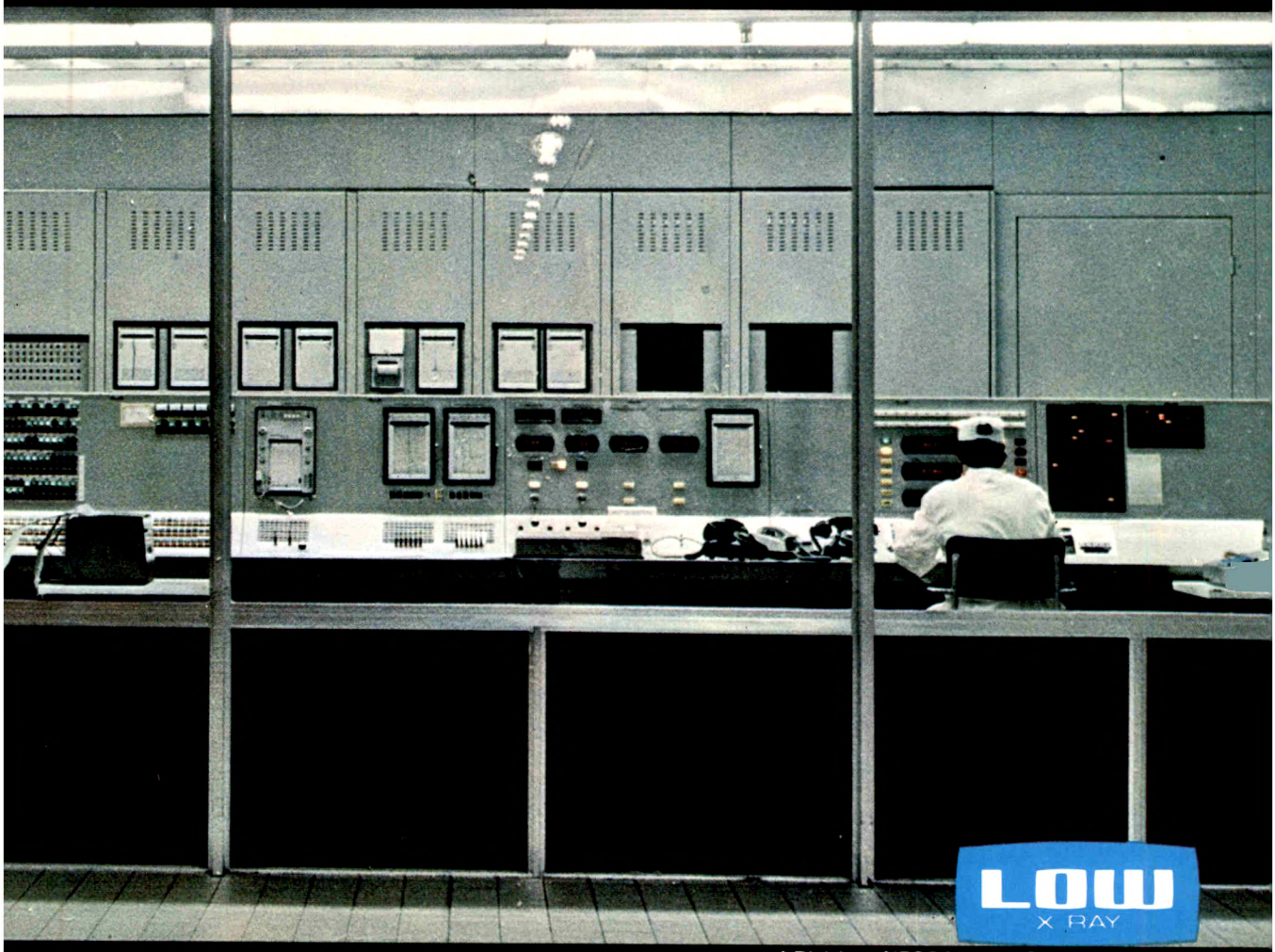
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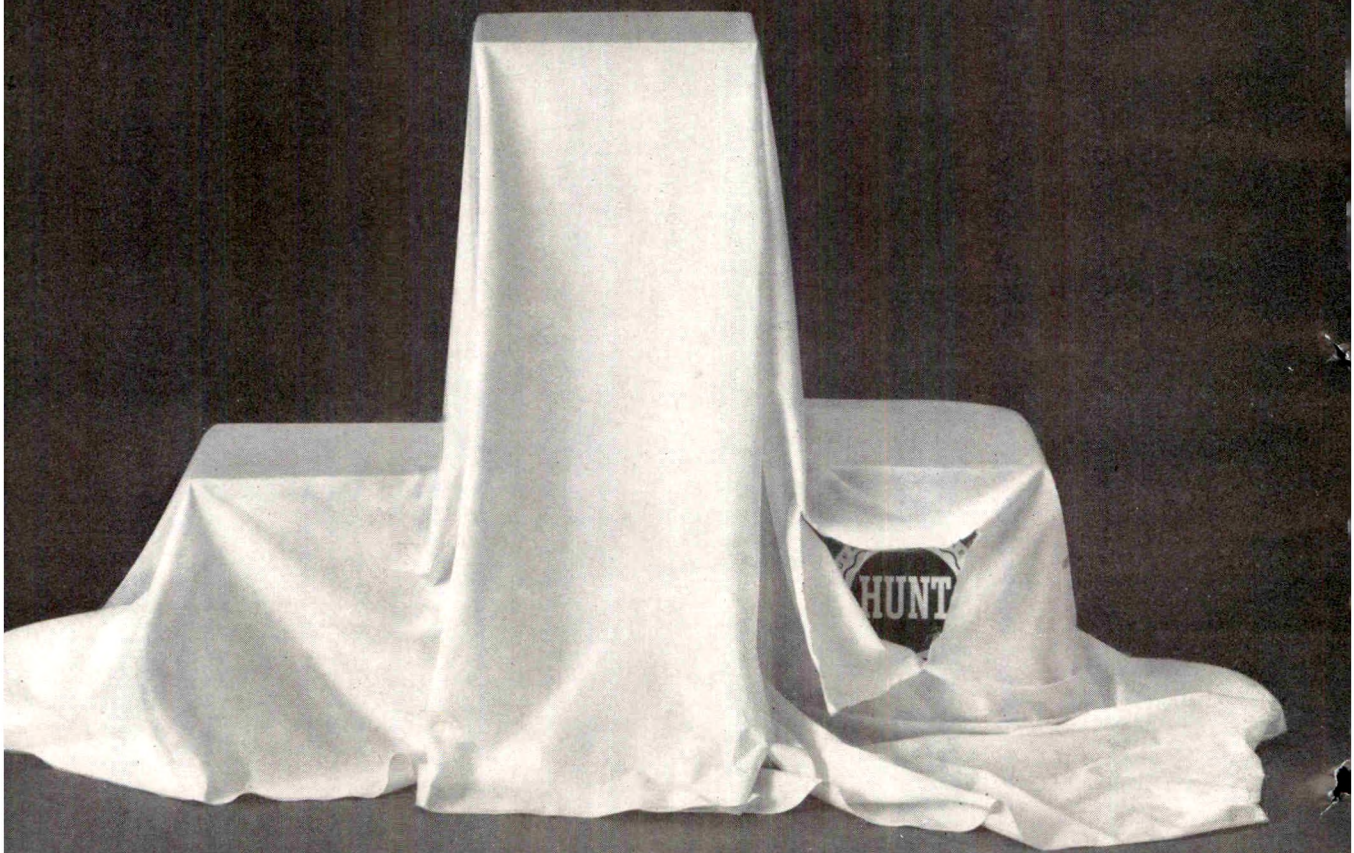
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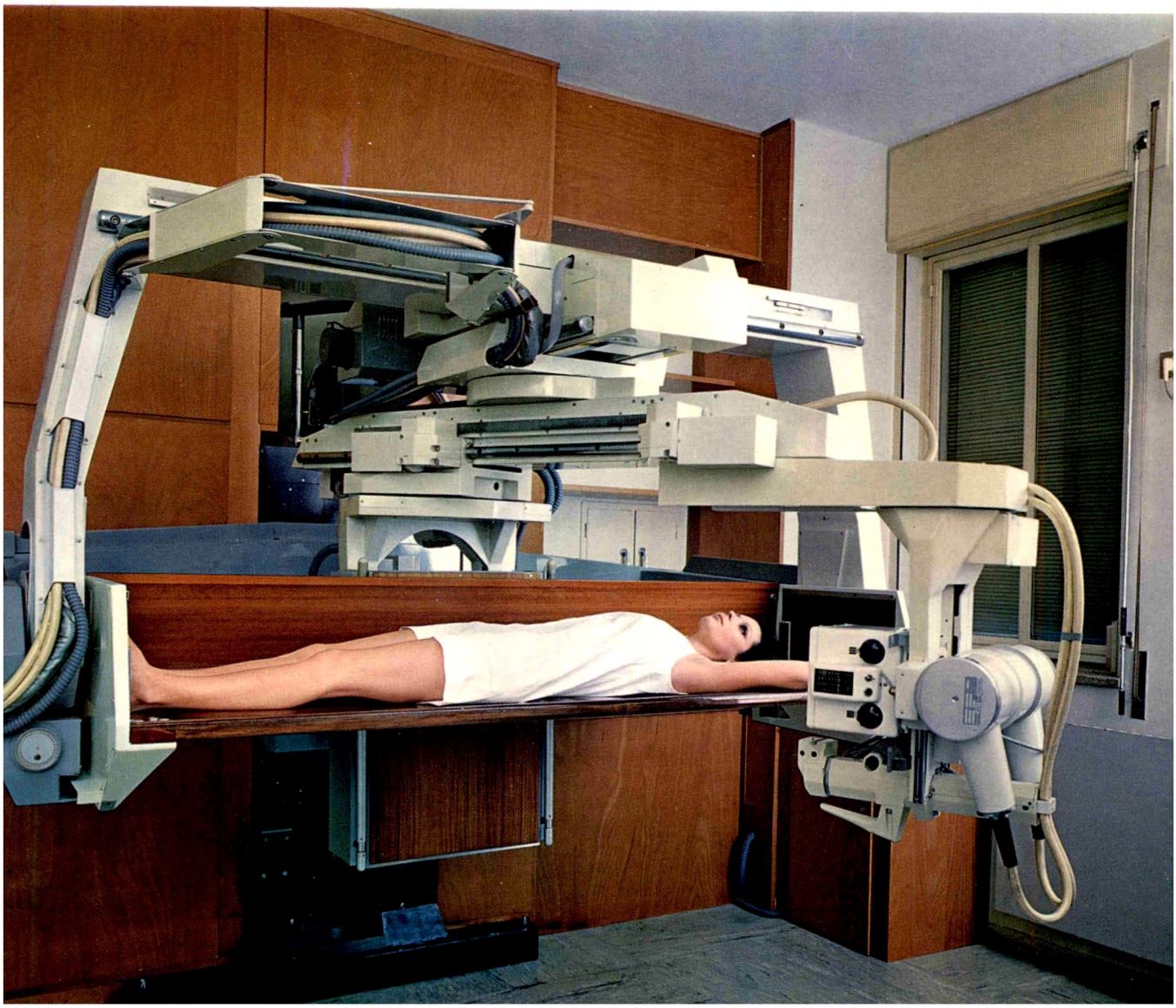
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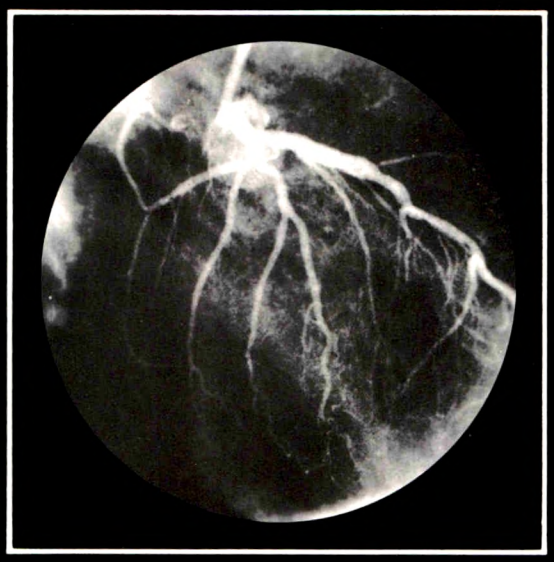
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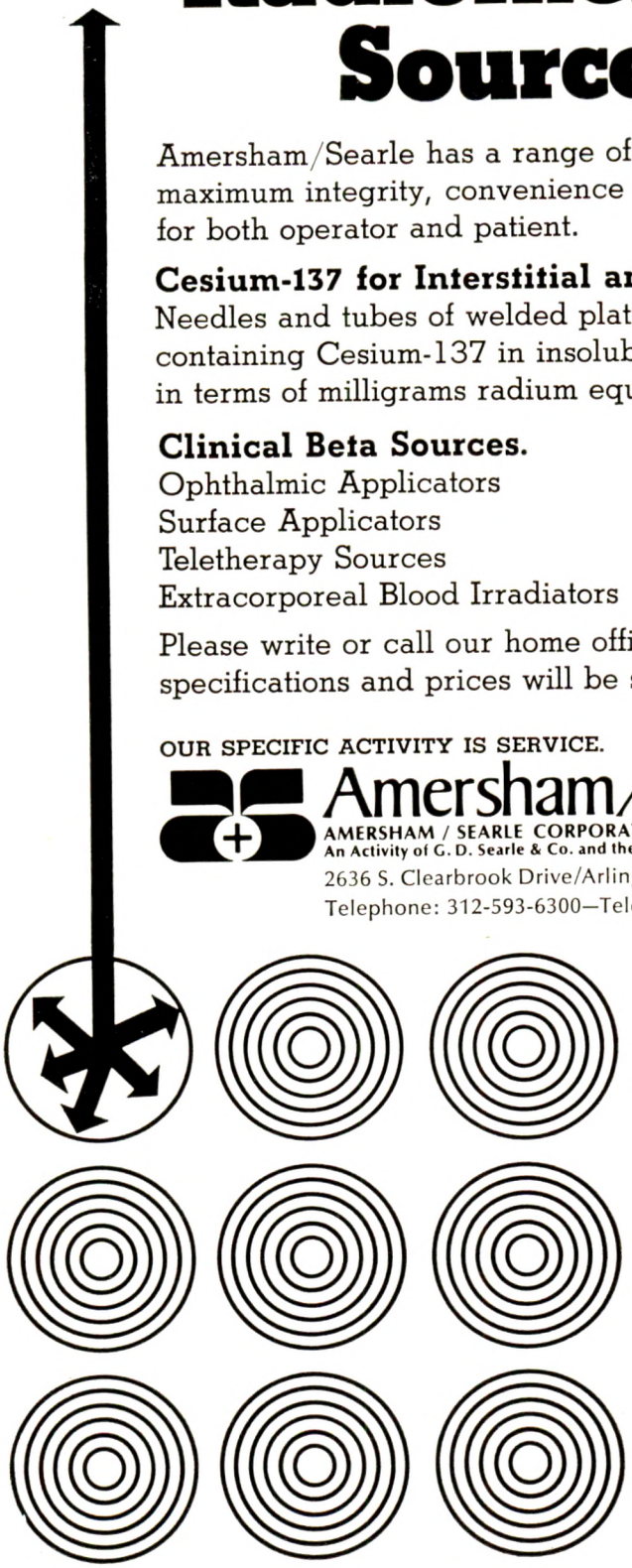
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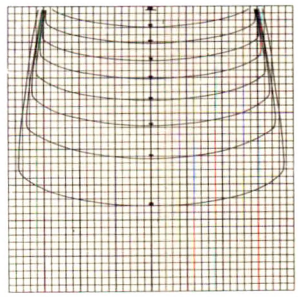
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also treated with irradiation, 3 of whom had metastatic disease and a fourth who had evidence of tumor remaining at the margins of enucleation.

The treatment modality was supervoltage therapy, with the exception of 1 case treated with an orthovoltage machine. Most patients were treated with lateral fields and the mid orbital dose ranged from 4,900 to 6,000 rads in 25 to 106 days.

The results revealed preservation of vision and survival with no evidence of disease in 5 of the 12 patients; 11 patients survived from 2 to 16 years without evidence of active disease. The 1 death occurred, when the parents refused enucleation after recurrent disease had become evident.

The authors conclude that there was no greater morbidity following radiotherapy as compared to primary enucleation and that there was effective control of recurrent tumor. On the basis of these results they believe that primary radiation therapy should be carried out in early unilateral or bilateral cases and that enucleation should be reserved for radiation failure.—*Robert P. Andrews, M.D.*

Simon, Norman, Lesnick, Gerson J., Lerer, William N., and Bachman, Arnold L. Roentgenographic localization of small lesions of the breast by the spot method. *Surg., Gynec. & Obst.*, April, 1972, *134*, 572–574. (From: Departments of Radiotherapy, Surgery, and Radiology, Mount Sinai Hospital, New York, N. Y.)

At times mammography can demonstrate small lesions in the breast that can not be felt. This has created a problem of precise localization of lesions, so that they can be removed for examination by a pathologist. The difficulties are augmented because the position of the breast during operation is different from its position during roentgenographic examination.

The authors have adopted the following technique for the precise localization of breast lesions: the cephalocaudad and lateral roentgenograms of the involved breast are studied to give an approximate localization of the nonpalpable but visualized lesions. A small syringe is then loaded with a mixture of less than 0.1 ml. ethiodol and 0.1 ml. Evans blue. The skin overlying the lesion is anesthetized and a 20 gauge needle is inserted into the breast tissue as close to the lesion as possible. An attempt is made to strike the lesion precisely and image intensified fluoroscopy is sometimes of technical assistance. The position of the needle is confirmed by repeat roentgenograms and the needle is repositioned if necessary. The mixture of radiopaque material and dye is then injected in a single spot at the presumed lesion. Cephalocaudad and lateral roentgenograms of the breast are repeated and a relationship is established between the suspect lesion and the radiopaque material and visible dye.

When the patient is operated, the surgeon finds the tissue colored by the dye. He is confident of removing the suspect lesion because of its relationship with the blue dye which is readily visible at operation.

The technique is illustrated with 6 roentgenographic reproductions.—*Eugene C. Klatte, M.D.*

Torné, E. Vendrell. (Barcelona, Spain.) Estudio del drenaje linfático fisiológico de la mama con isótopos radiactivos. (Radioisotopic study of physiologic lymphatic drainage of the normal breast.) *An. méd. Barcelona*, Aug., 1971, *57*, 344–357.

The lymphatic pathways of 250 normal breasts were studied.

The isotope used was colloidal Au^{198} with an average particle size of 33 Å. The injected volume was 0.3–0.5 cc., giving a dose of 200 microcuries.

Fifty breasts were injected in the superolateral quadrant, 50 in the inferolateral, 50 in the superomedial, 50 in the inferomedial, and 50 in the subareolar area. Scans were obtained 24 hours after the injection, depicting lymph node uptake.

The following table gives the percentage opacification of the different lymph node groups according to the site of injection:

	Axillary	Internal mammary	Supraclavicular
	%	%	%
Superomedial quadrant	94	62	6
Inferomedial quadrant	68	86	2
Superolateral quadrant	98	36	4
Inferolateral quadrant	90	64	4
Subareolar area	100	20	0

It is noted that the internal mammary lymph nodes are significantly involved in the drainage of all the areas of the breast; this should be considered in therapy planning.

The author favors surgical resection of the internal mammary lymph nodes in all cases of breast cancer. —*Julian B. Alvarez, M.D.*

Lubin, E., and Lewitus, Z. Blood pool scanning in investigating hepatic mass lesions. *Seminars in Nuclear Med.*, April, 1972, *2*, 128–132. (Address: E. Lubin, M.D., Department of Nuclear Medicine, Tel Aviv University Medical School, Petah-Tikva, Israel.)

Liver and spleen scans obtained with the administration of tracer labeled colloid disclose the presence, location, and size of space-occupying lesions in the liver, but do not distinguish their vascularity.

To differentiate the vascularity of space-occupying lesions (SOL) a blood pool scanning using in-

dium 113m was performed after the standard colloid scanning. A comparison of the relative vascularity in a lesion between the colloid scan and the blood pool scan was obtained in: cysts; abscesses; metastases; hepatoma; and hemangioma.

The only lesion with increased vascularity in a space-occupying lesion compared to the surrounding liver was hemangioma.

Cysts, abscesses, metastases and even hepatoma had vascularity usually less, or at most equal to that of the surrounding liver.

The technique of performing blood pool scanning on patients with space-occupying lesions in the liver scan is useful in differentiating the vascular from the nonvascular lesions.—*H. William Strauss, Major, USAF, MC*

Kagan, A. R., Hafermann, M., Hamilton, M., Pierce, R., Morton, D., and Johnson, R. Etiology, diagnosis and management of pericardial effusion after irradiation. *Radiol. clin. et biol.*, 1972, *41*, 171–182. (Address: A. R. Kagan, M.D., Cedars-Sinai Medical Center, Cedars of Lebanon Hospital Division, Division of Radiation Therapy and Nuclear Medicine, Los Angeles, Calif. 90029.)

The use of supervoltage radiation in the treatment of Hodgkin's disease has permitted higher subcutaneous tissue doses, which in case of radiotherapy over the mediastinum can lead to pericarditis with pericardial effusion. This can progress to constrictive pericarditis with complete obliteration and fibrosis of the pericardial space and may also lead to advanced myocardio-fibrosis and endocardiopathy.

In this study, supervised by the National Cancer Institute, in the 3½ years between January 1965 and June 1968, 109 patients were included. Twenty-two per cent of these patients developed pericardial effusion following irradiation; most of these patients were treated with anterior fields only.

Statistical analysis of the data with the aid of a computer reveals that the estimated risk of pericardial effusion at 90 per cent local control of Hodgkin's disease, is 8 per cent in those patients who have involvement of the mediastinum, 2 per cent in those who have a normal mediastinum and 4 per cent for all patients, when opposing portals are used. The calculated dose to the anterior heart is in the 5,000 to 7,000 rad range when equally weighted opposing fields are not used.

Preferred treatment of pericarditis with effusion secondary to irradiation for Hodgkin's disease is early pericardiectomy. This conclusion is drawn as most of the patients who were treated in this fashion, because of pericardial constriction or elevated venous pressure, did well. Why some patients with pericardial effusion secondary to irradiation will have an eventual benign course in the absence of surgery is not clear. The authors urge elective surgery in asymptomatic patients of this type with long-standing pericardial effusion.

Some interesting case reports are also presented in this study, emphasizing the need for early pericardiectomy and also proving that exploratory thoracotomy in these patients often reveals an unsuspected persistent Hodgkin's disease.

Among the conclusions drawn is a recommendation that opposing fields should be used to treat the mediastinum if a total dose of 4,000 rads to the mid chest is desired to produce a 90 per cent local control of Hodgkin's disease, without a major incidence of radiation-induced pericardial effusion.—*Robert P. Andrews, M.D.*

Cameron, S. J., Grant, I. W. B., Pearson, J. G., and Marques, C. Prednisolone and mustine in prevention of tumour swelling during pulmonary irradiation. *Brit. M. J.*, Feb., 1972, *1*, 535–537. (From: Respiratory Diseases Unit, Northern General Hospital, Edinburgh; Department of Respiratory Diseases, University of Edinburgh; and Radiotherapy Unit, Western General Hospital, Edinburgh, Scotland.)

Ventilatory function declines during the early stages of irradiation for bronchial carcinoma. This decline is potentially dangerous, if the tumor narrows the trachea or both main stem bronchi.

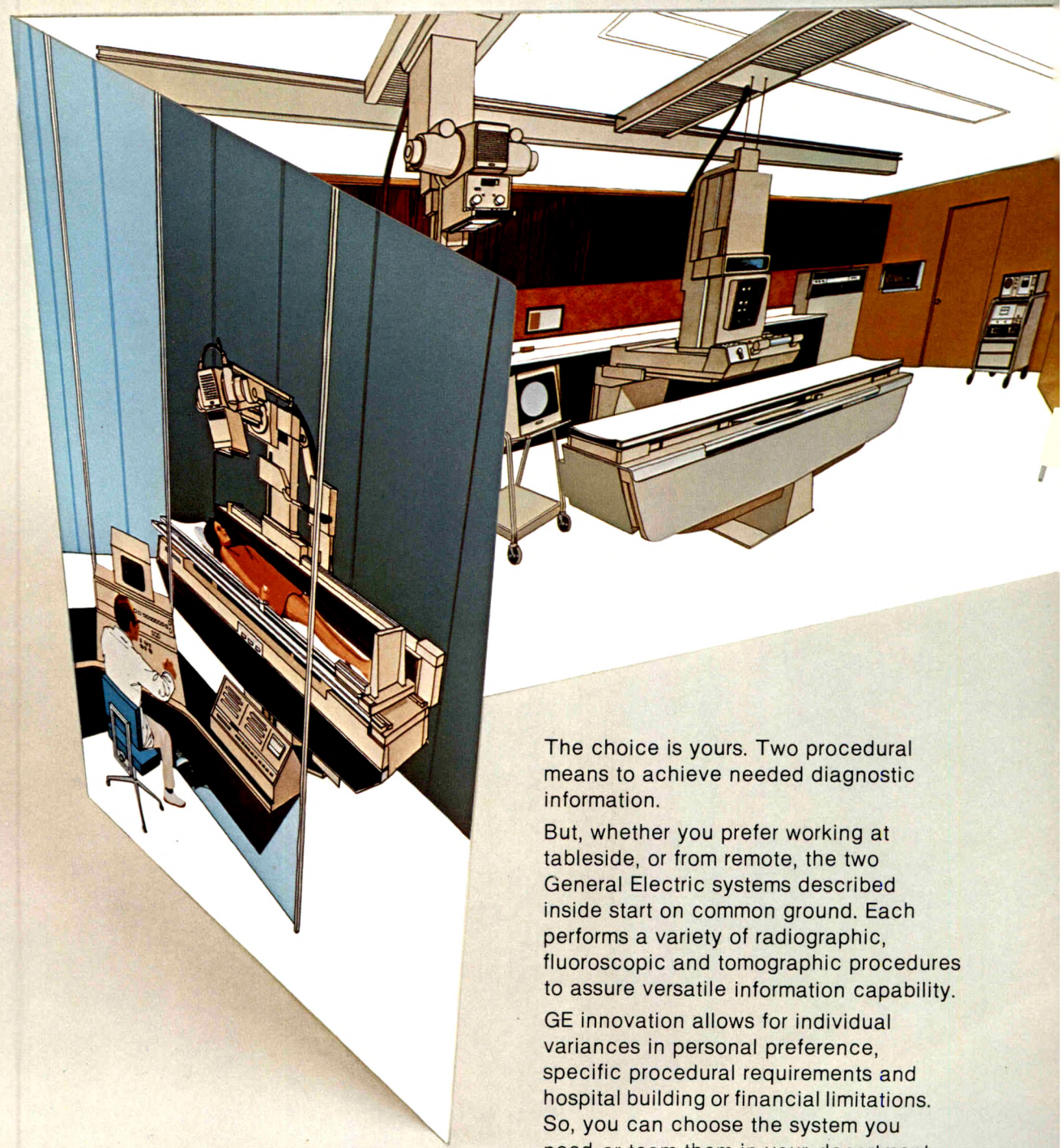
The protective effect of preliminary treatment with prednisolone or mustine before irradiation was studied in 88 patients by serial estimation of forced expiratory volume and forced vital capacity. Twenty-three patients received prednisolone by mouth, 24 patients had mustine intravenously and 41 had no preliminary treatment.

Both prednisolone and mustine prevented a significant decline in ventilatory function due to tumor swelling. Prednisolone was marginally superior.

It is concluded that all patients at risk should be treated with oral prednisolone, 20 mg. daily, for 1 day before and 2 days after the first fraction of irradiation.—*Eugene C. Klatte, M.D.*

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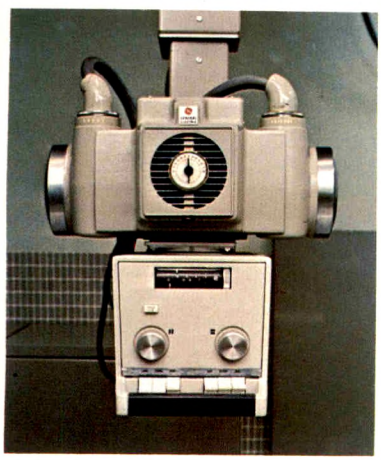
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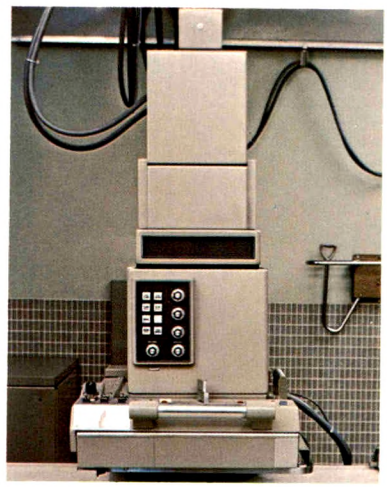
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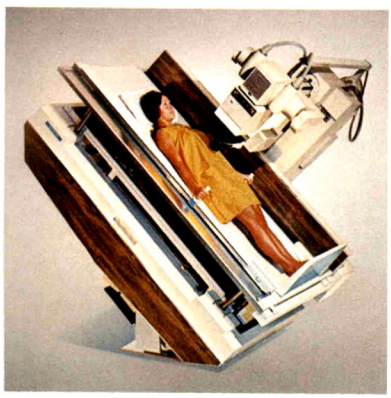
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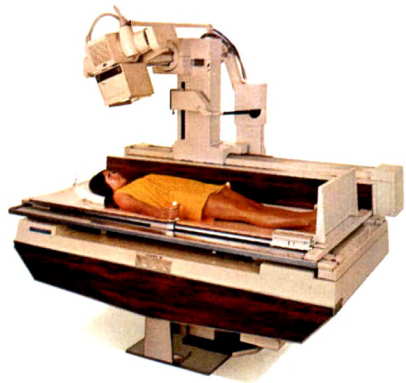
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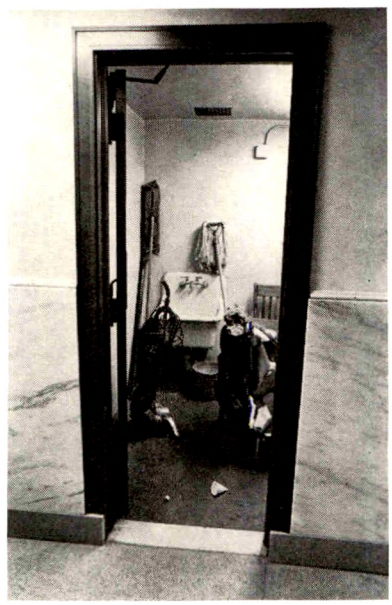
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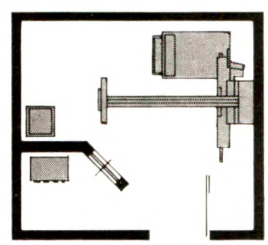


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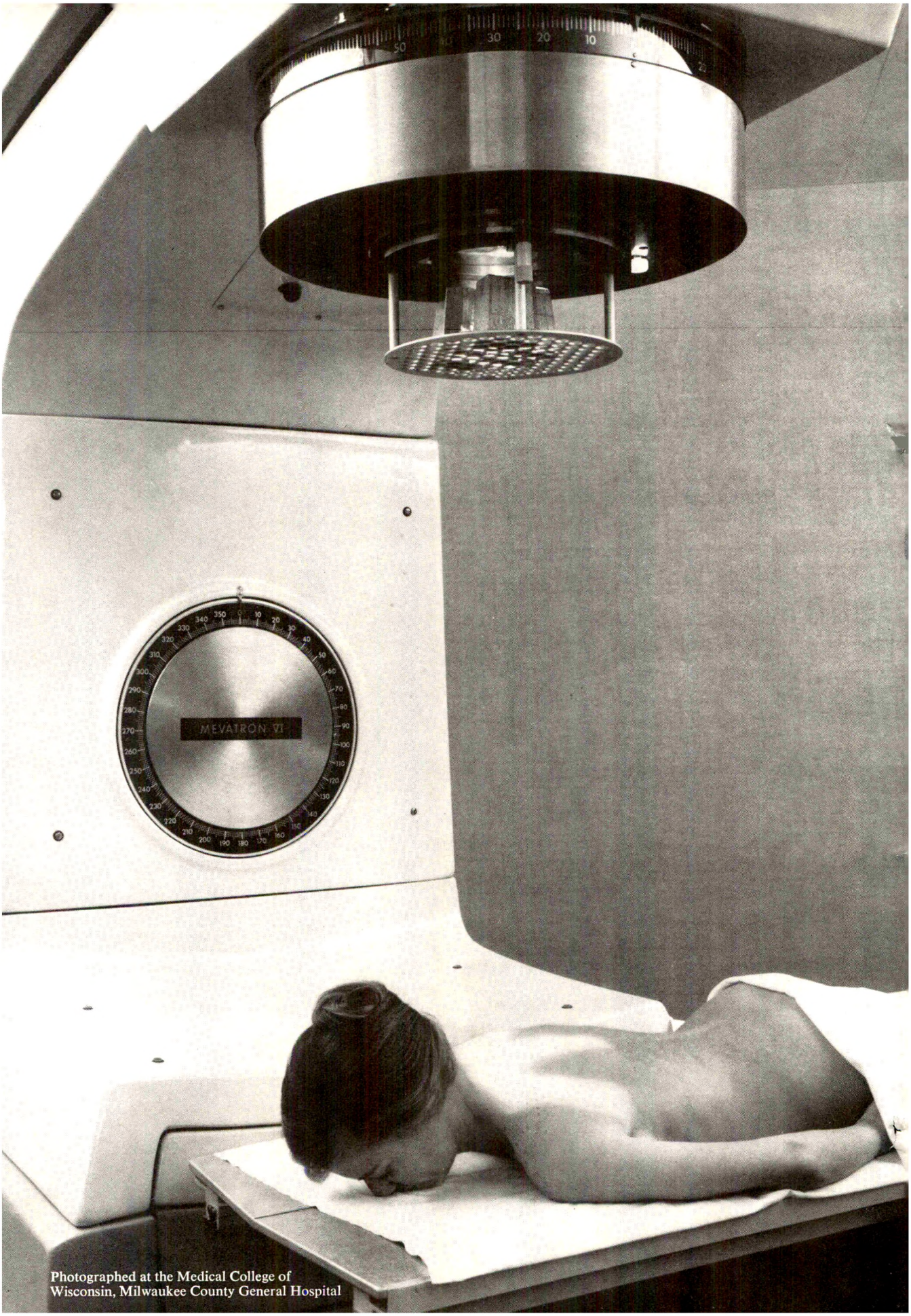
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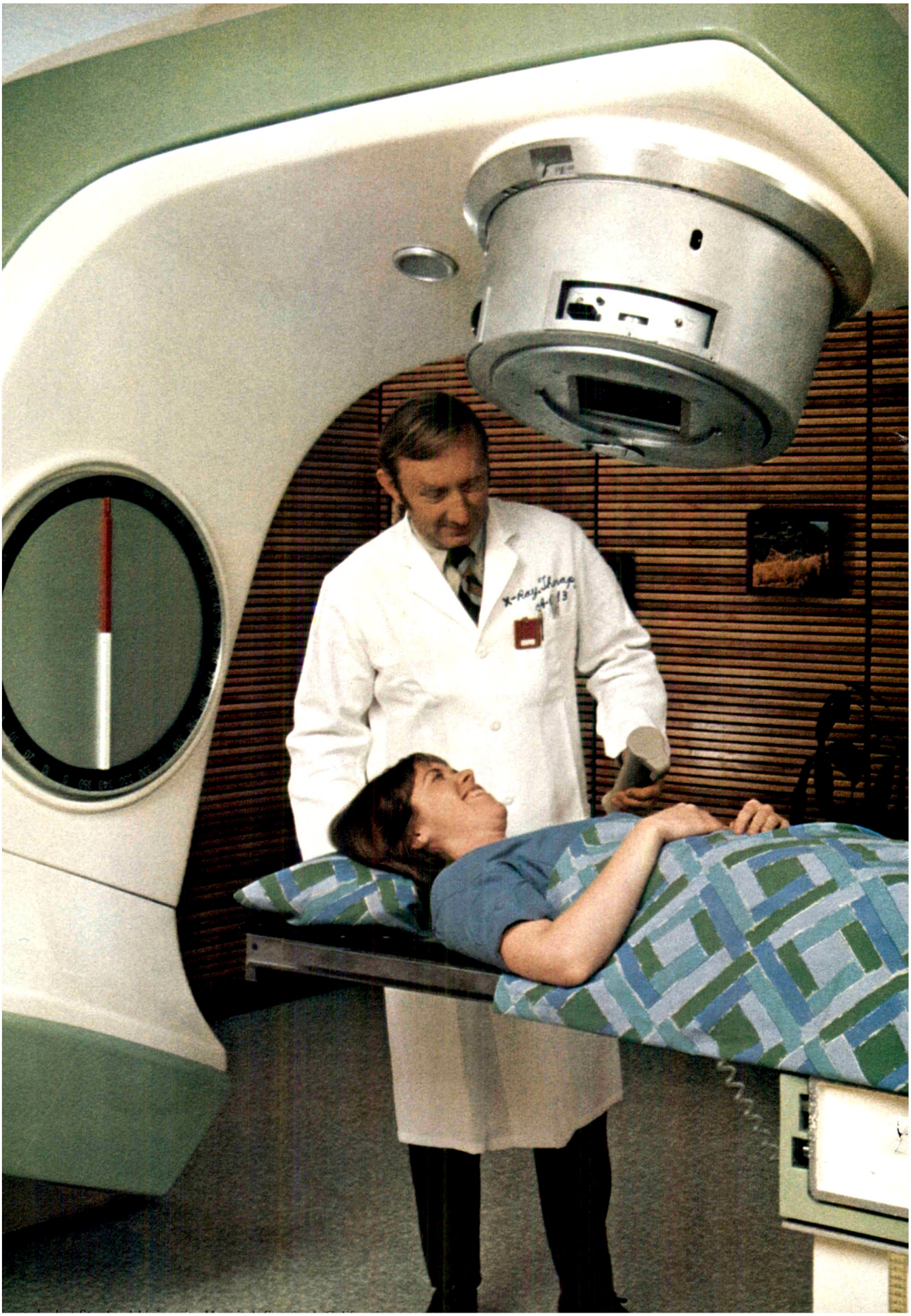
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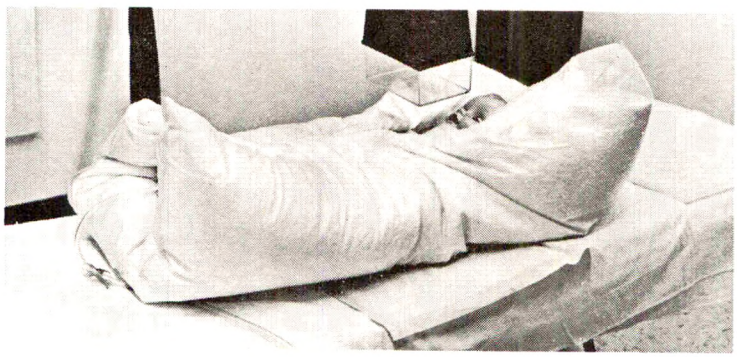
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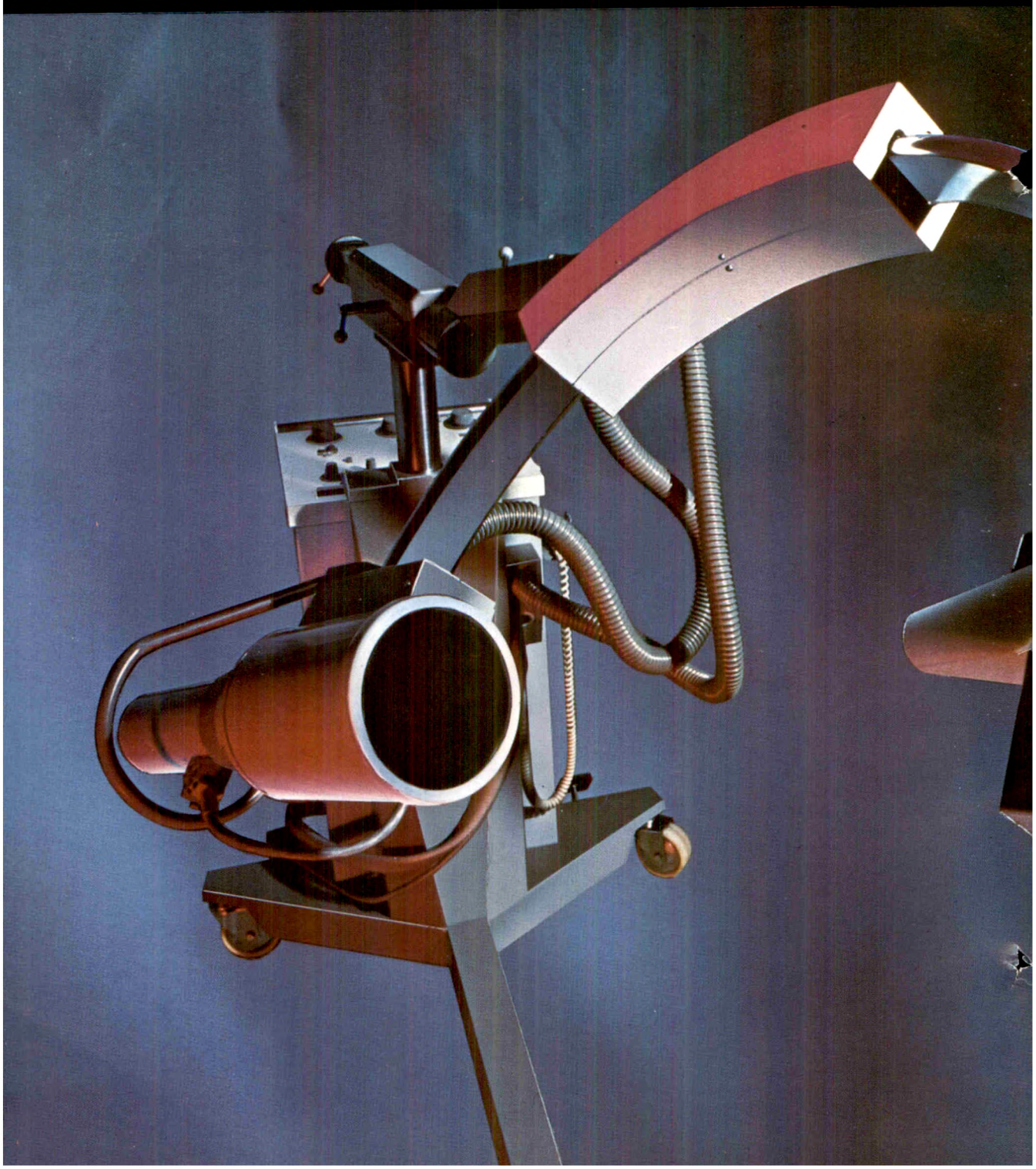
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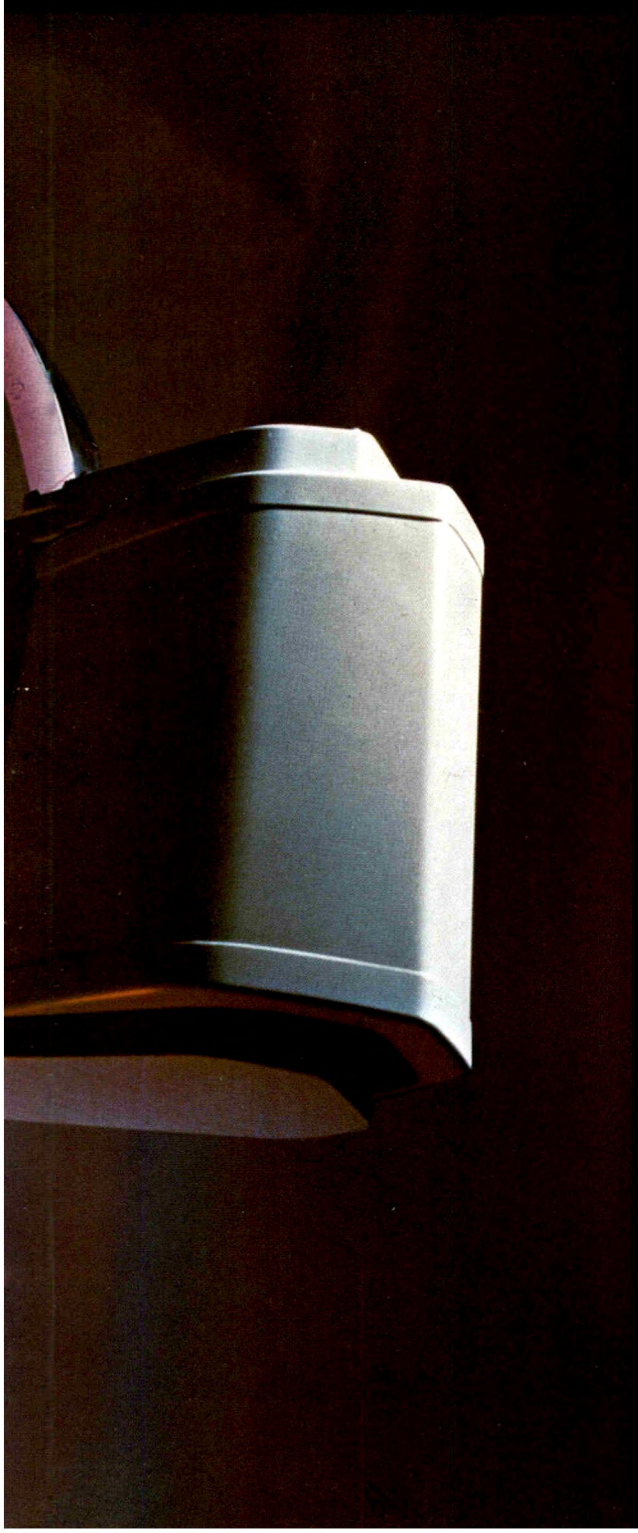
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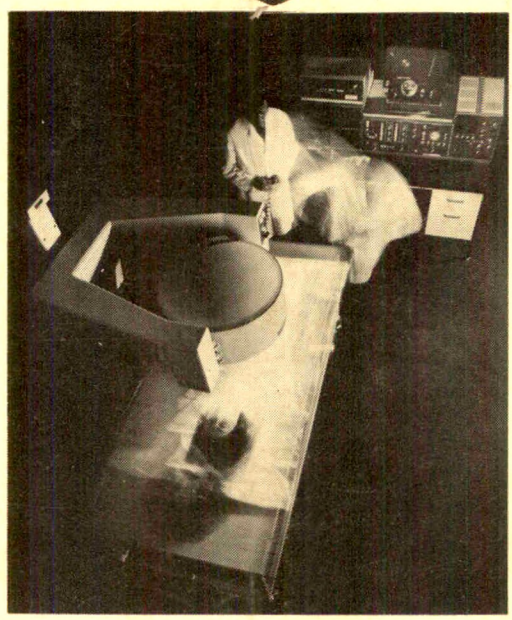
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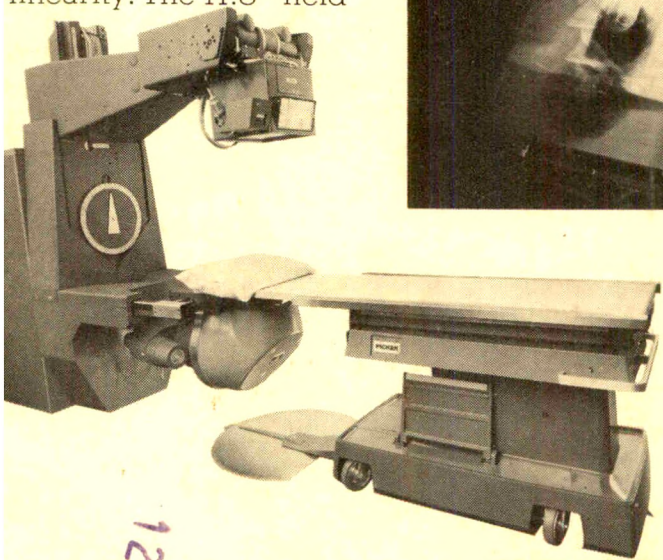
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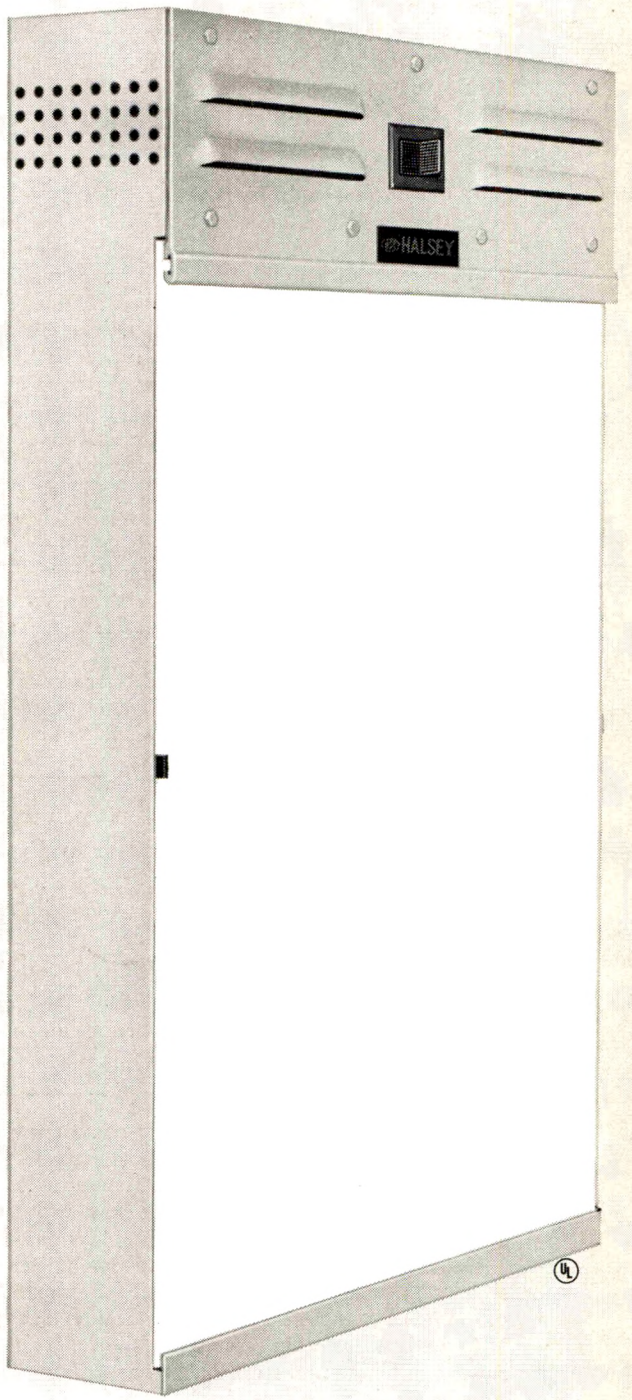
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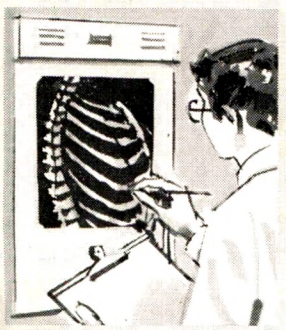
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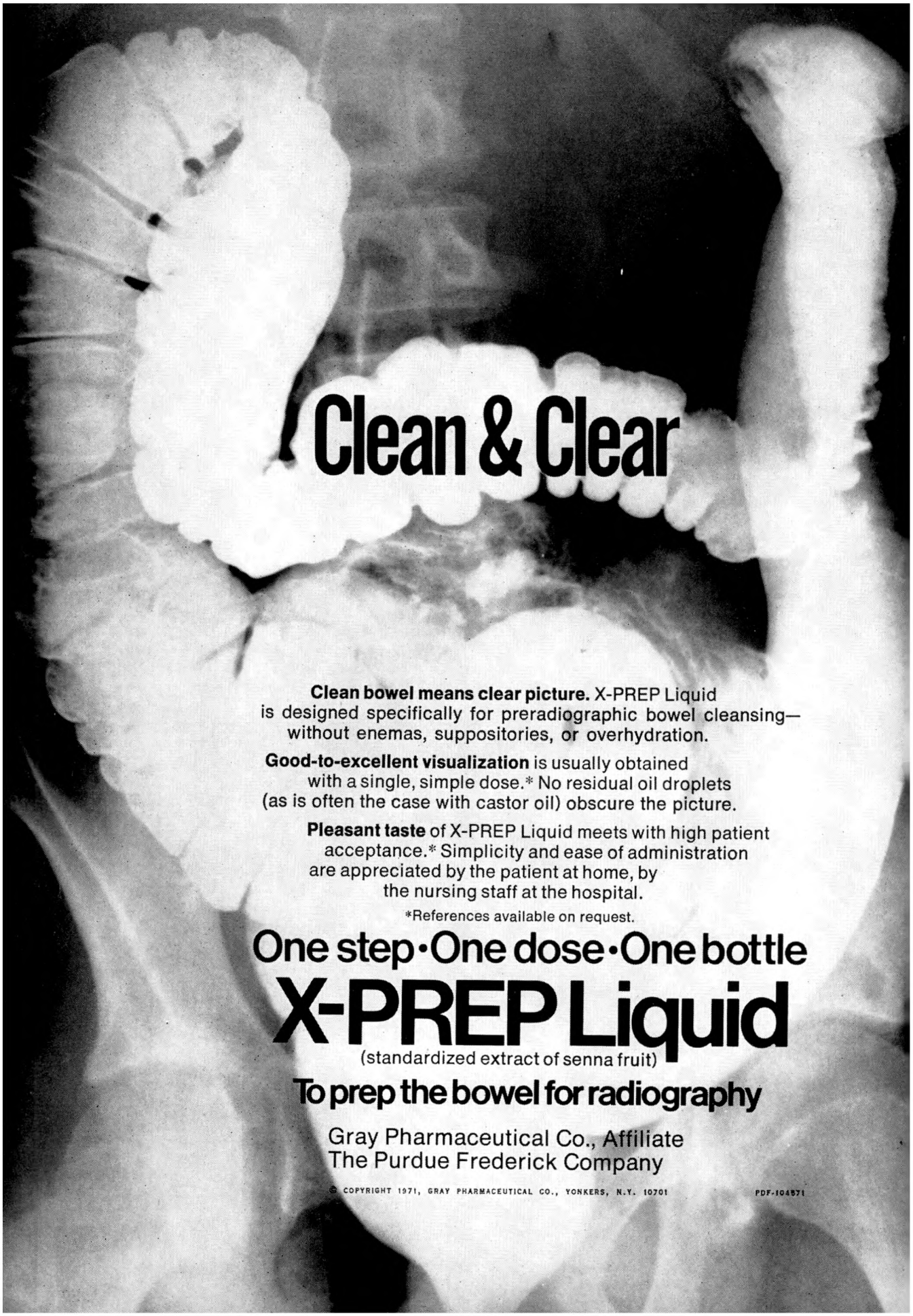
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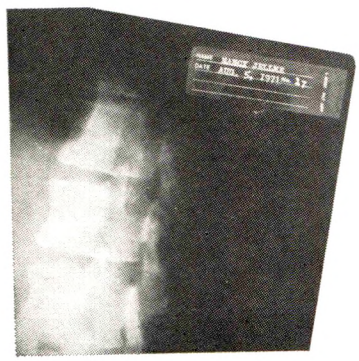
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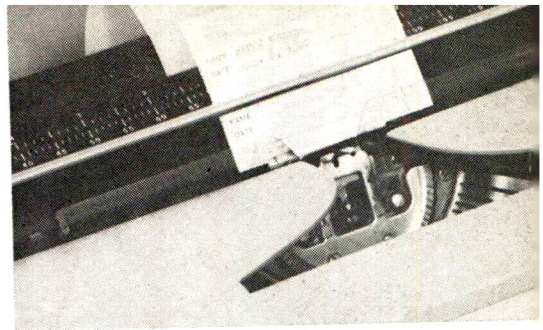
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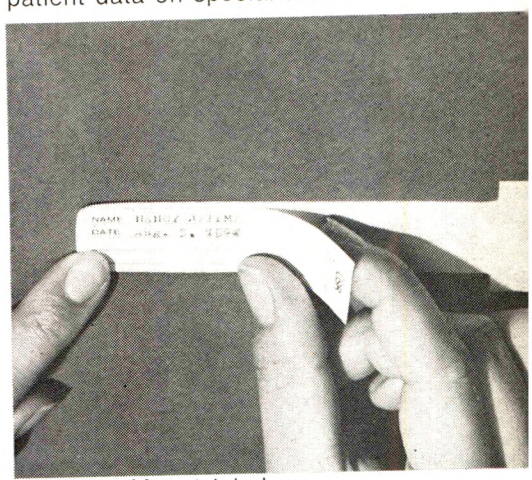


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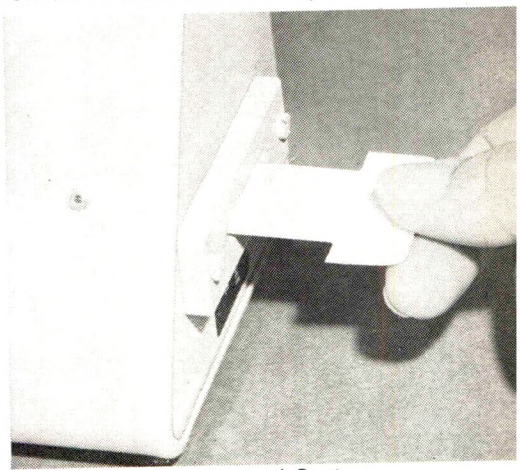
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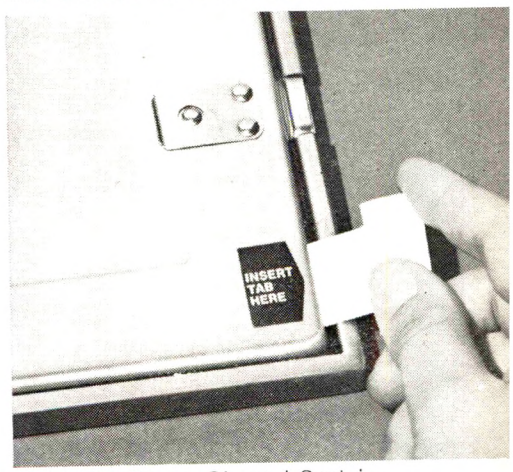
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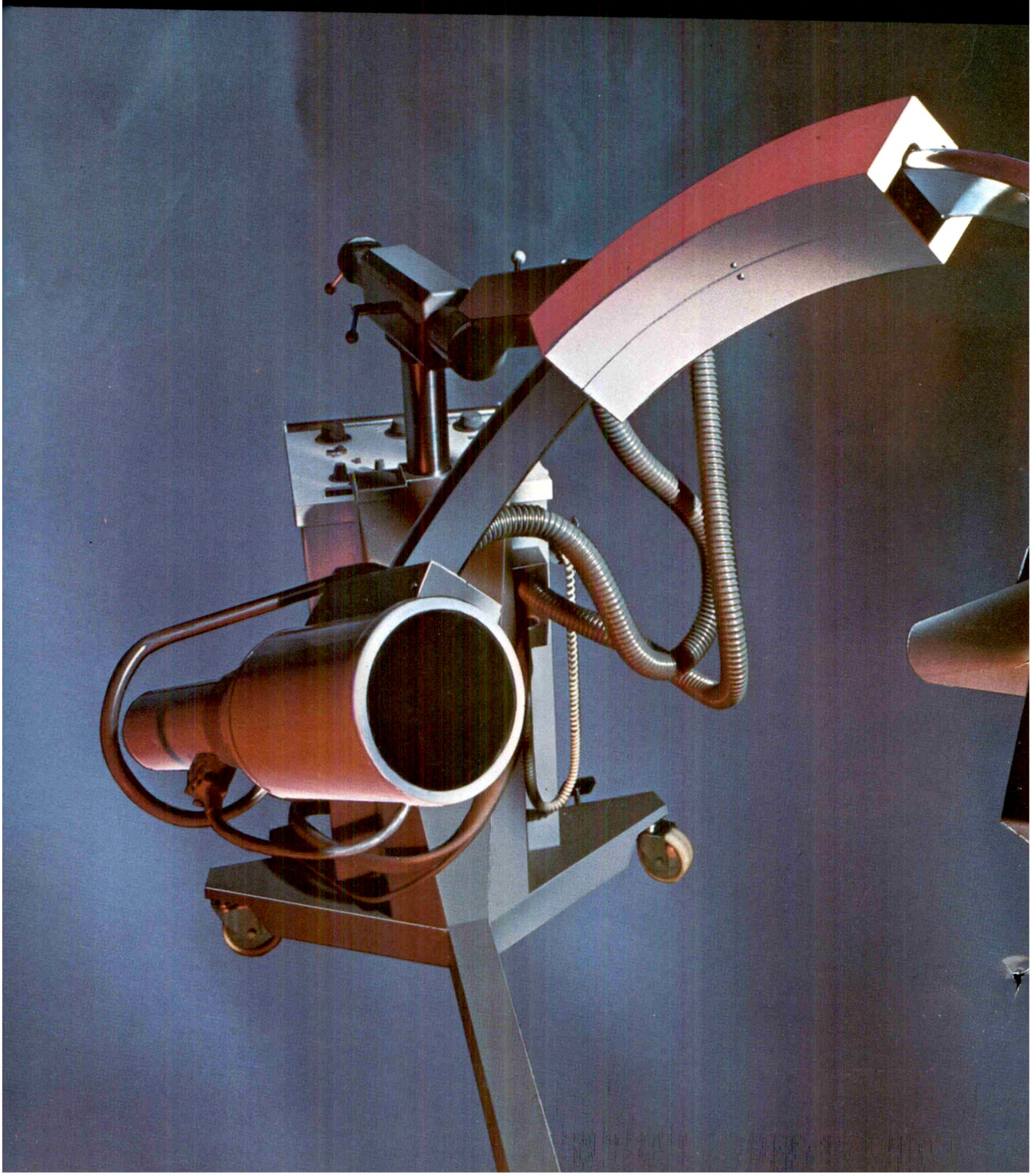


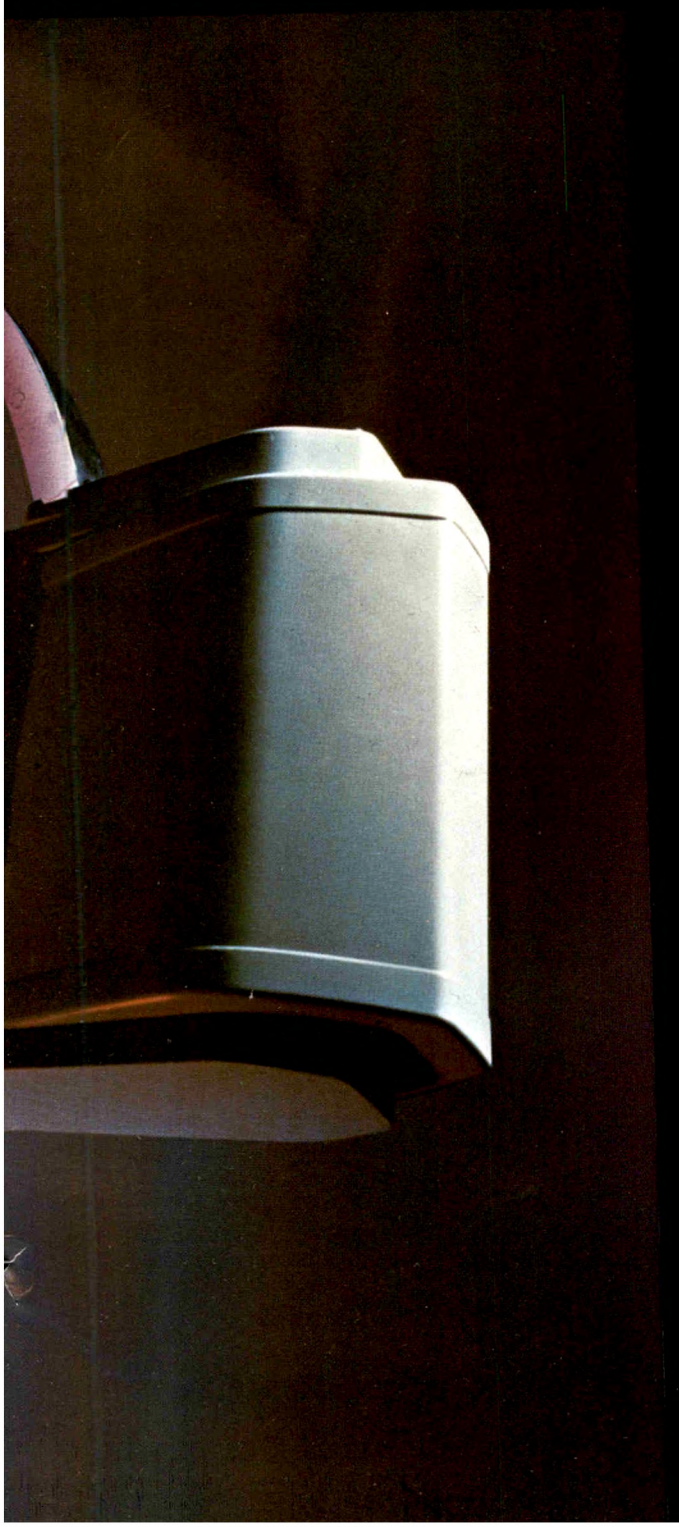
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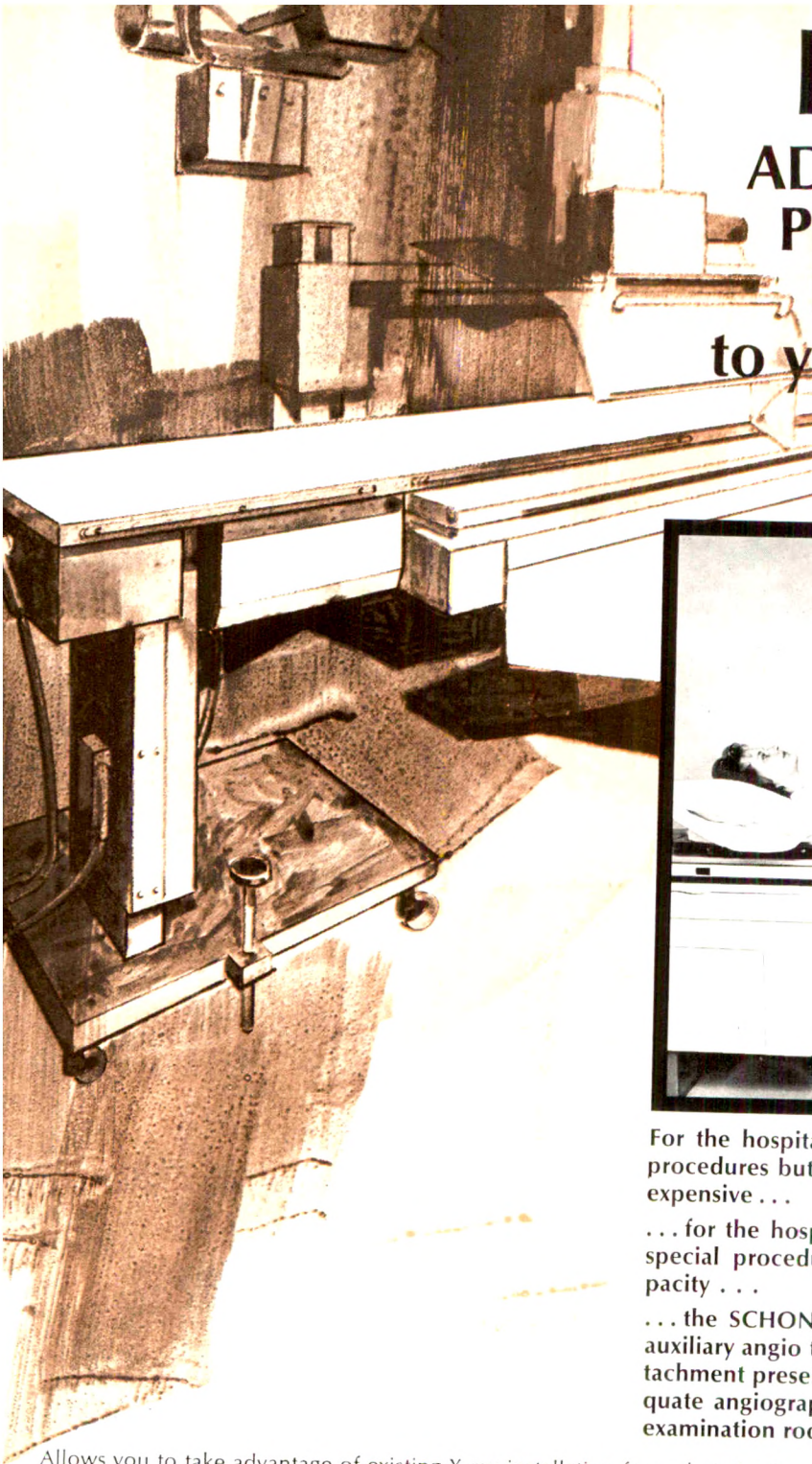
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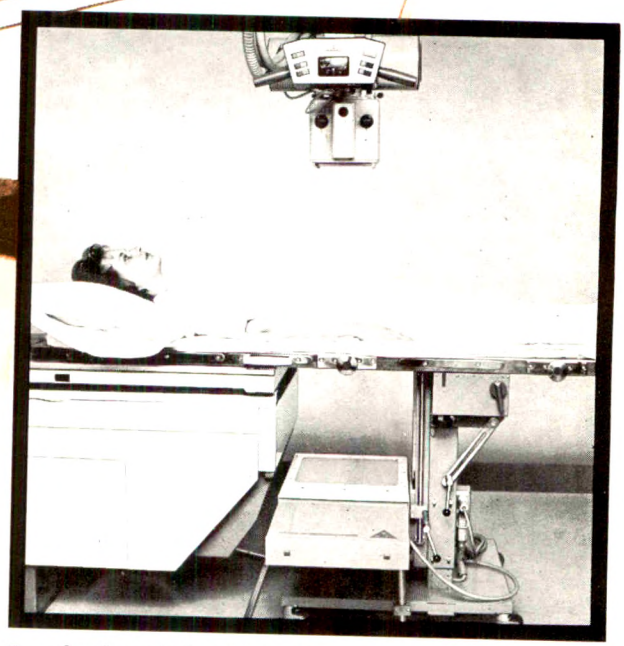
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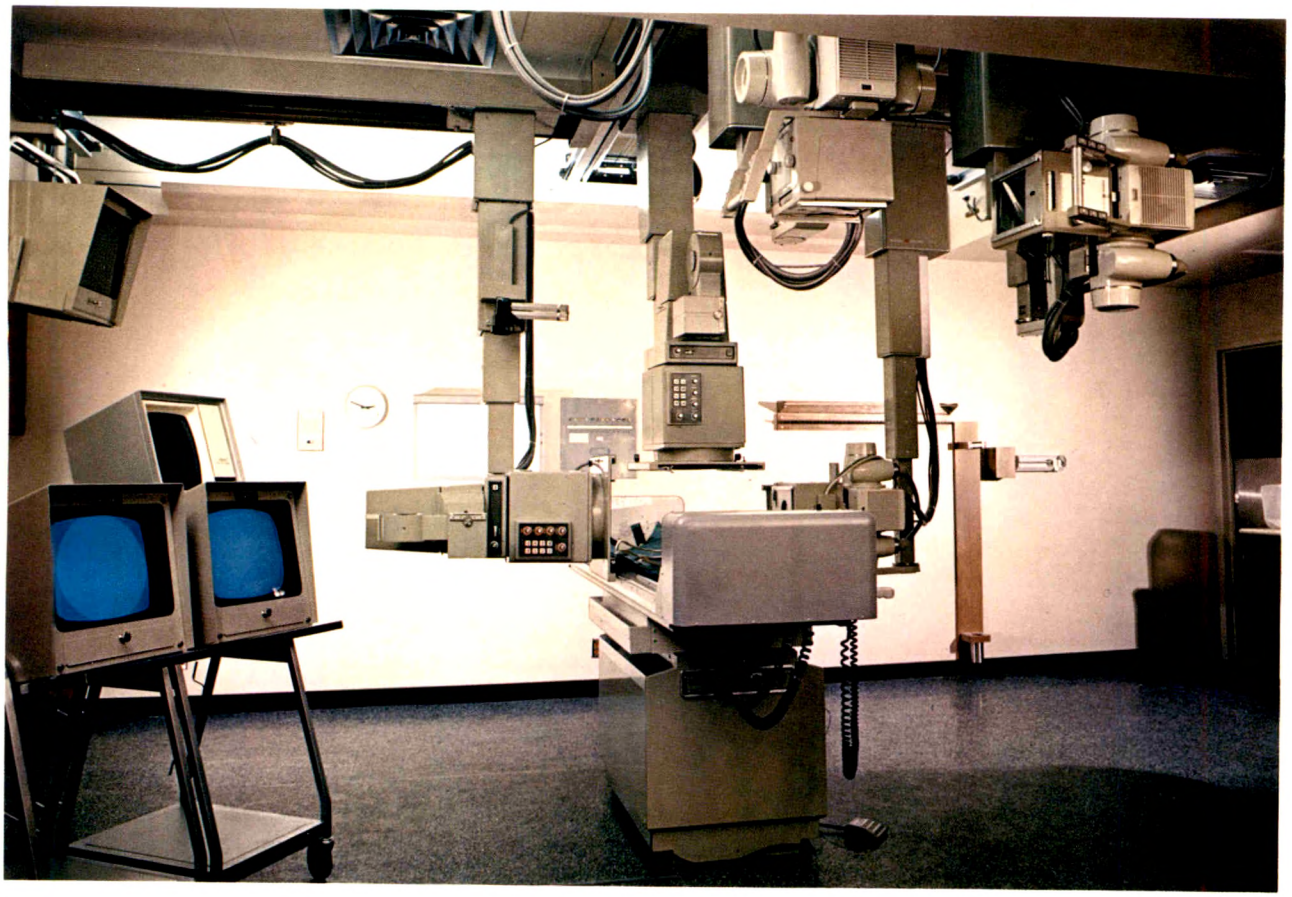
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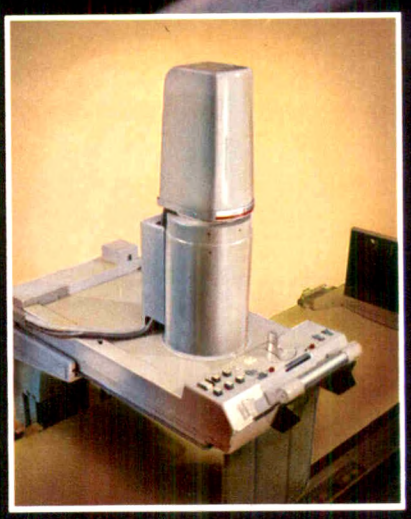
Still more GE differences: Innovation; several examples are described inside. And service; ask any hospital we serve about our capability for keeping procedure schedules on schedule. And broad range of equipment; designed to be integrally combined to maximize the potential of each component.

Here's specific GE equipment that proves it...

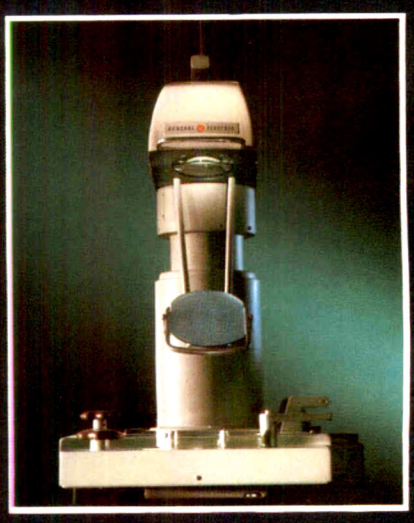
Fluoricon 300 image intensifier



Fluoricon Compact,
folded optics



Fluoricon Compact,
coaxial optics



Fluoricon Compact,
mirror optics

Image intensifiers that coordinate with any room's schedule

All things considered—degree of technic sophistication desired, visualization and recording preferences, your profile of patients and the economic facts of life—General Electric can match your image intensification demands.

Fluoricon® 300

This top-of-the-line image intensifier assures line pair and image resolution never before possible; permits the realization of new fluoro technics.

One reason: a new solid state TV camera, coaxially coupled to a new lens system, minimizes light loss. Before installation, you can select from a wide range of single, dual and triple image fields in 4, 6 and 9 inch sizes. Special circuitry, too, permits you to pan a patient and get consistent images, without blooming, regardless of localized excess brightness; equalizes brightness across the monitor; and enhances contours for crisper images.

Video tape recording is integral at enhanced levels; also at normal fluoro levels for back up recording. Further recording capability includes a choice of 35 mm cine cameras; Cine 180™; and GE's new 105 mm photospot camera, with frame rates up to 12 per second.

Fluoricon® 200

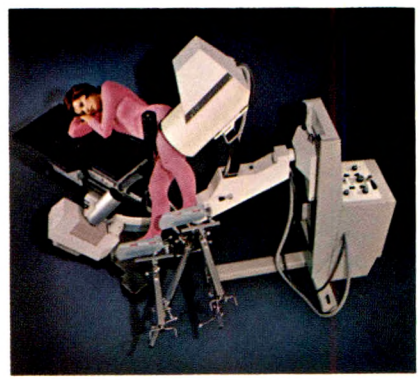
In basic R & F procedure rooms, the Fluoricon 200 image intensifier features TV fluoroscopy for dynamic studies. And, integrates video tape recording. A coaxially-coupled TV camera and lens system maximizes light transfer for every procedure.

This 9 inch single field system assures the optimum line pair and image resolution needed; maintains a consistent image even during panning. An optical viewer is interchangeable with the TV camera. Or, select a Fluoricon 200 with mirror optics only.

Compact intensifiers

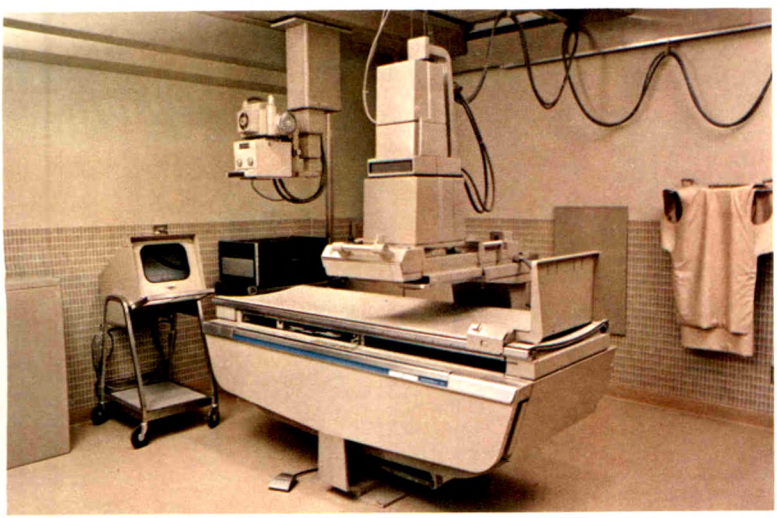
A choice of three Fluoricon Compact 6 inch image intensifiers combines lightweight efficiency with a quality image for the many routine assignments in your daily schedule. Diagnostic capability that's neither too costly nor too specialized.

The first, with folded optics, accommodates a 70 mm photospot camera and provides video presentation. A unit with coaxial optics provides video presentation only. And, an intensifier with mirror optics fits wherever you need an economical general fluoro unit.



Mobile intensifier

The Mobile Fluoricon image intensifier extends bright image visualization to bedside, to orthopedics, surgery, cast rooms, emergency and ICU/CCU wards. It moves and positions with ease around patients for real-time images from any point of view.



Basic R & F room featuring Fluoricon 200 image intensifier

GENERAL ELECTRIC

X-ray tubes and generators:

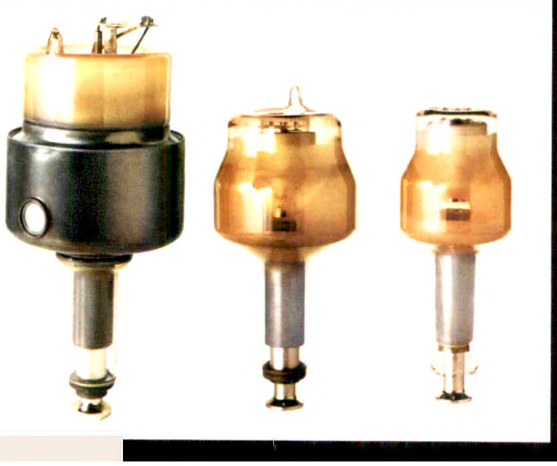
Output-matched for any procedure room.

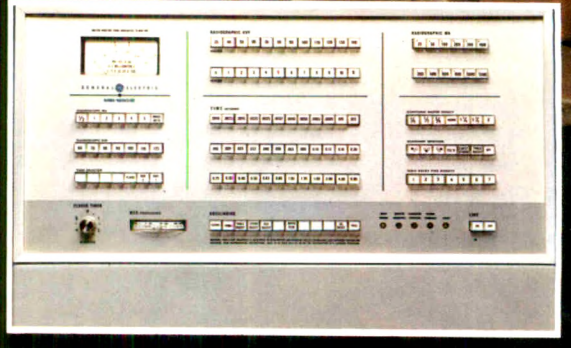
High-energy, high-volume radiographic procedures demand a tube/generator team that can maintain the pace. GE satisfies that demand.

Three Maxiray™ x-ray tubes, with anode diameters up to five inches, take on and dissipate the big heat that's generated during high energy procedures and extended schedules. You get the high instantaneous exposure ratings you need; so, more radiation can be generated through a smaller focal spot for better detail and resolution.

And, there's a GE generator powered to any system. Like the MSI™ line. Three-phase, 12-pulse generators with consistent power that meets full-rated capacity both in theory and in fact. Millisecond interrogation means more useable time per exposure. And, there's the MST™ 3-phase, 6-pulse generator line. All MSI and MST generators feature Quantamat™ phototiming that automatically and accurately times exposures down to two milliseconds. And, tube protection circuitry that conforms to the wide range of tubes available today.

Maxiray x-ray tube and Sentry collimator





MSI-1250 generator

Automatic collimator:

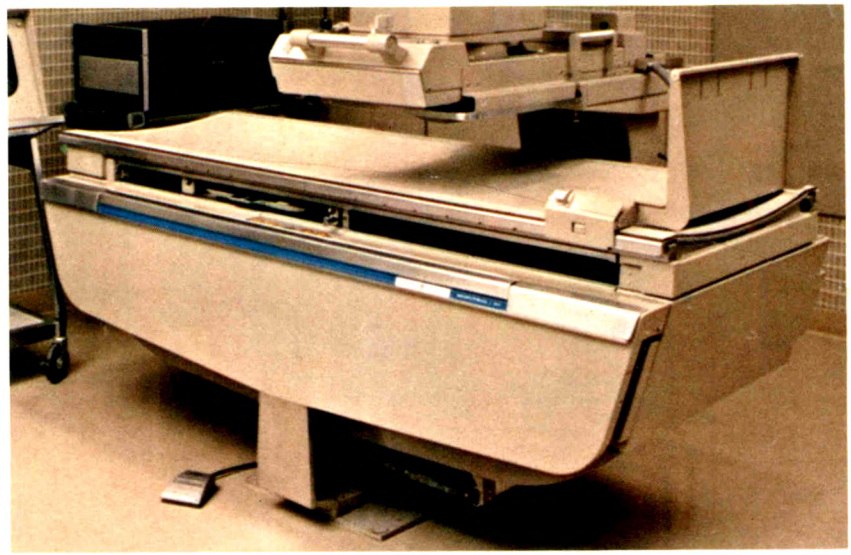
Integrates safety into any x-ray system.

Sentry™, an automatic beam limiting system, reduces off-focal radiation; assures minimum exposure; reduces patient set-up time. With three levels of shutters, Sentry conforms to HEW specifications.

In horizontal table positions, Sentry senses cassette size and focal distance. Then, automatically maintains the radiation field size equal to or less than film size, even as the tube unit is raised and lowered. Automatic collimation is also provided, at standard distances, for vertical table technics and for off-table chest work.



Sentry collimator



Monitrol/90 table

Radiographic tables:

One table line permits multiple equipment combinations.

Monitrol™. You could build most of your R & F procedure rooms around it.

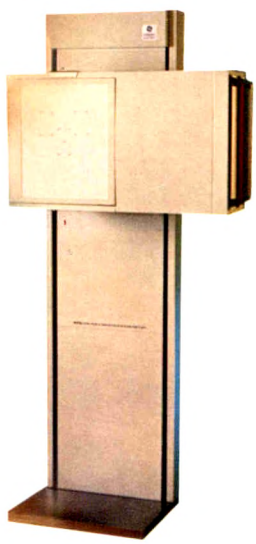
Monitrol/90, with 90°-90° table angulations and four-way top motions, facilitates views from any desired position, including steep-angle technics. All movements are safety interlocked.

Monitrol/15 provides 90°-15° angulations and a four-way shift top. Equip it to handle a variety of routine R & F procedures. Or, without the fluoroscopic carriage and tower, you have a versatile table for conventional radiography only.

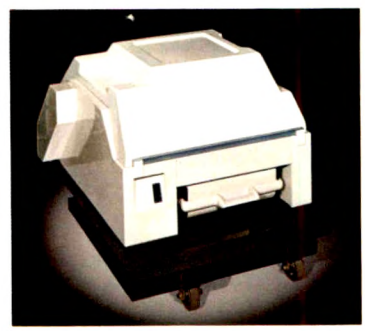
GENERAL ELECTRIC

Take the load off your chest x-ray schedule.

Changex™ chest unit keeps the patient load flowing smoothly. Features a 125-film storage magazine, with automatic advance, that ends the need for technologists to handle individual 14 x 17 inch cassettes. Positions at a touch. Assures optimum film/screen compression for sharp, informative radiographs.



Changex chest unit



Q-82 changer

Rapid film changer:

Runs on air to produce superior 12-per-second films.

The Q-82™ changer virtually eliminates jamming and vibration; delivers superior resolution at speeds up to 12 frames per second.

This self-threading changer uses air to advance the film and to compress intensifying screens during exposures. Features a wide selection of frame rates in each of three different phases, plus an adjustable pause. The magazine is automatically loaded, in daylight, with your choice of up to 39 frames.

Multiple procedure diagnostic x-ray system:

Proves its credentials in busy hospitals.

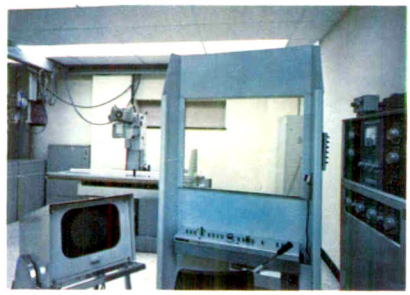
Some procedures no radiologist wants to repeat. Such as those requiring the use of opaque materials. Or with pediatric and geriatric patients. The first film, ideally, should be a good film; and provide the needed diagnostic information.

General Electric's Telegem™ 5 diagnostic x-ray system is designed for times like these. Situations complicated by crowded schedules, a premium on time and shortages of trained professional personnel.

Telegem 15 integrates fluoroscopy, radiography and linear tomography in one room. All controlled from a shielded remote console. So, there's no need to wear heavy lead aprons or gloves.

All programming, changeover between technics, table positioning—and more—are performed from remote with the turn of a dial or press of a switch.

TV fluoroscopy lets you confirm patient positions. Phototimed







exposures assure consistent film density. And you can program up to six uniform exposures on a single film. Even intermix Bucky radiography and linear tomography or zonography to provide a complete examination on one film.

This integrated system includes a choice of two 3-phase, 12-pulse generators; choice of 6, 9 or 6/9 dual field image sizes; cine and 70 mm photospot recording capability; a GE Maxiray x-ray tube and fully-powered 90°-15° table.

Because it's a compact system, Telegem 15 makes efficient use of small space. It's entirely floor supported and easy to install. Flexibility of component placement permits ready adaptation to a variety of existing room layouts and sizes.

Details about specific Telegem 15 hospital installations are available on request.

GENERAL ELECTRIC



Illuminators:

Fluoroline Rotoview delivers the big revolution in high capacity illuminators.

With a full capacity of 376, 14 x 17 inch films, the General Electric Fluoroline® Rotoview Illuminator provides more available information in a minimum of floor space. Or, choose the 208 film capacity model. Either one assures that the radiologists' viewing time remains undisturbed by film-loading interruptions.

Fluoroline Rotoview is designed for easy operation. Panel drive mechanism is uncommonly quiet and smooth. Each of the four-place film panels glides into position at the push of a button or floor switch. Panels can be powered upward or downward.

Viewing area is uniformly lighted and independently controlled. Detail presentation of any area of a radiograph is increased by a high intensity, movable scanning spotlight.

All controls are recessed and integrated in the 15 inch work shelf. A 2-way floor switch provides hands-off control. And, a unique full-length tray at the bottom of the unit slides out to allow easy retrieval of dropped items, such as films or pens. Fluoroline Rotoview is paneled with textured oak laminate. Operates on 115 V, 60 Hz, 15 amp current.

Best Buy Program :

GE's extra step that makes the difference in medical systems.

Value is an elusive concept. So, to make it more meaningful to you, General Electric has initiated what we call the Best Buy Program. Meaning, a program to make GE medical systems the best you can buy.

This involves a variety of internal incentives among GE employees to continually remind them of their responsibility to maintain GE's reputation as the industry leader in quality and service. The program's goal is to have each employee add a touch of value to every system he touches.

Specific examples of this extra effort are described throughout this Compendium. Best Buys like the Raport™ radiographic reporting system. This GE innovation can increase department efficiency, shorten the patient's stay, and reduce the cost per report. Another Best Buy product, not described in this Compendium, is GE's AMX - 110., a cordless auto/mobile x-ray unit. Everything needed for routine radiography is integrated, including the power to conduct the procedures and for driving the unit to wherever the patient is located. Your GE representative can add more reasons why GE is your best buy.

Radiographic reporting system:

Transforms radiologist's diagnosis into typewritten reports in seconds.



Now only a few seconds from the time a patient's radiographs are interpreted, a hard copy report can be automatically typed, ready for signature and delivery to the referring physician. Ready too for clinician conferences and for posting in the wards.

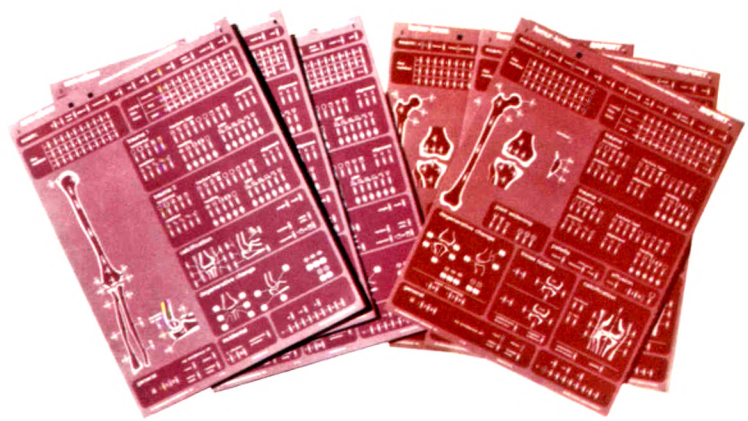
The Raport™ radiographic reporting system from General Electric provides this dramatic time decrease in a process that used to take hours. Hours of work. Hours of waiting. Helps reduce a major problem resulting from growing patient loads while it expedites availability of vitally needed diagnostic information to the referring physician.

The system features a series of mark-sense cards, which cover examinations of the entire body. Used in conjunction with dictating equipment, they provide reporting capability for all of the department's case load. Of this total, approximately 80 percent of the reports can be produced using only the input from mark-sense cards. The remainder can use either a combination of the card and dictation or dictation only, at the radiologist's option.

While reading the films, the radiologist simply checks symbols representing his diagnosis. Marked information is then quickly scanned, instantly interpreted, converted into complete-yet-varied, fully-structured sentences, and automatically typed. One simple press of a button produces a complete, typewritten report, including patient and radiologist identification.

X-ray reports are stored in the Raport control unit's memory. This provides query capability by patient name, name of the referring physician, patient location, or type of examination requested. In addition, the system can provide work lists covering daily department activity.

Raport permits the department to increase the number of examinations on which it can report each day; increases the efficiency of the clerical staff; and, can reduce the cost per report.



R&T procedure room:

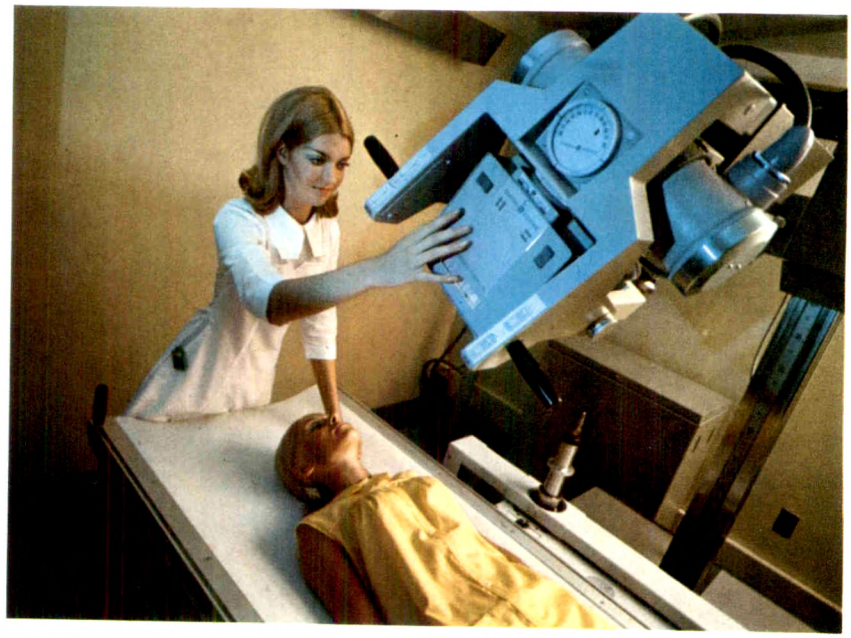
Combines radiography and tomography into one versatile-technic system.

General Electric's Maxitome™ tomo/radiographic system assures dual procedure capability in one room. You can program radiographic and tomographic technics, with the turn of a dial, from the system's remotely-located controls. Capability that can help relieve the pressures of growing patient loads.

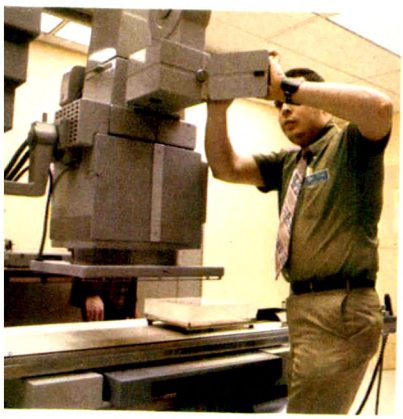
Select any of Maxitome's 11 tomographic patterns, from linear to pluridirectional. And, adjust the plane, mm by mm, from zero to 240 mm above the floating top table.

Dial again for Bucky-quality conventional radiography. Collimation is automatic, manual or controlled from remote. And, manual masking is integrated into the table. Each procedure permits multiple film formats up to 9 on 1.

You can also do off-table radiography with a vertical Bucky. Angulate the tube unit to perform oblique view technics. And, confirm the patient position, to reduce retakes, with intensified TV fluoroscopy.







Service capability:

Helps keep your procedure schedules on schedule.

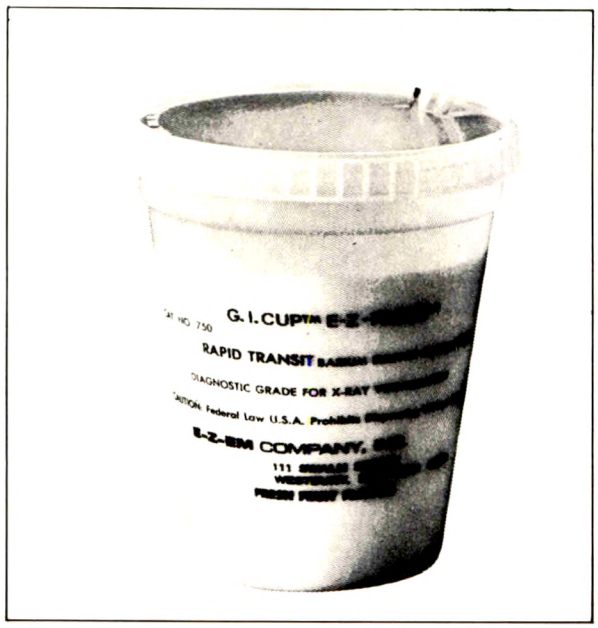
First, because of numbers. More than 700 GE field service technicians comprise the largest medical service organization in North America. And, they're strategically located to assure help fast when needed.

Secondly, training. At the Medical Systems Institute, GE's unique service education center, continual programs keep seasoned sales and service representatives technologically up-to-date and bring new men rapidly up to full service proficiency. Ask your GE representative to tailor a service plan to your individual requirements.

GENERAL ELECTRIC

General Electric Medical Systems, Milwaukee, Toronto, Liege

PATENT APPLIED FOR



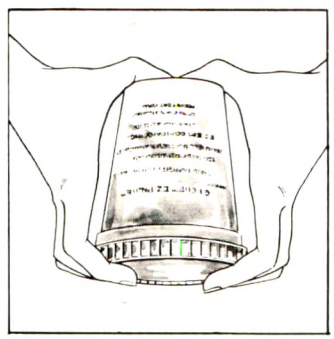
E-Z-EM's practical answer to waste, spillage and mess. The GI cup contains E-Z-PAQUE™, a rapid transit barium formulation — passes through the small bowel in half the time.

THE 45¢ CUP

Fill



Mix



Ready-to-use



IT'S ECONOMICAL

It costs no more to use the E-Z-EM GI Cup™ than to measure, pour and mix barium from a bulk container. We would be happy to supply you with free samples. In stock at your local dealer or contact:

E-Z-EM **"System of your choice"**

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How can the most reliable linear accelerator also be the least expensive?

It's simple when it's the rugged new SHM/Picker Therapi™ 4 linear accelerator. A true second generation instrument designed and built to achieve three major goals: simplicity, reliability, and low cost.

Even the smallest medical facility can now proudly provide the advantages of 4 MeV high output therapy. This versatile machine provides the broad spectrum of single and multiple field fixed beam techniques, and the large field techniques. At a price almost $35,000 under what you think you'd have to spend.

Advanced engineering knowhow resulted in truly remarkable simplicity. The reliability had to go up; initial and subsequent costs, down. And simplicity of design led to simplicity in use, too.

Then there's Picker service. Your local serviceman is factory-trained to master the specific machine that is subsequently delivered to you. He's your local expert and he knows how to keep you functioning.

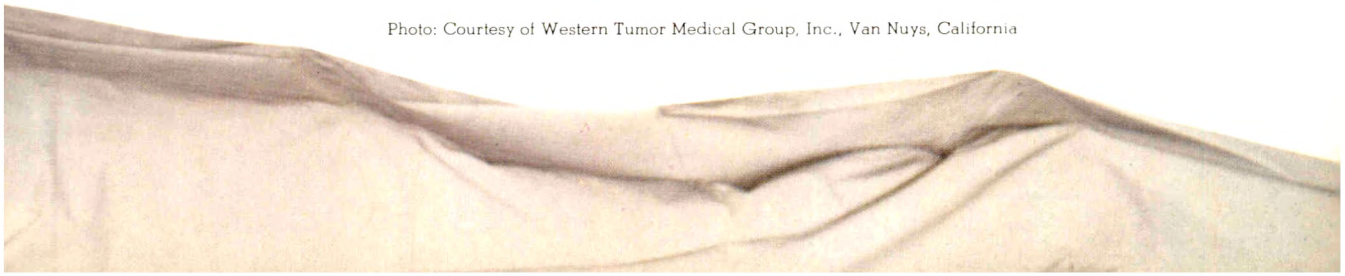
This is the kind of tough brute of a machine that can go all day at full output.

The SHM/Picker Therapi 4—practical for the smallest facility, perfect as a back-up unit in larger institutions.

Speak to your local Picker representative for more details, or write Picker Corporation, 595 Miner Road, Cleveland, Ohio 44143. Images for health.

PICKER®
ONE OF THE C.I.T. COMPANIES

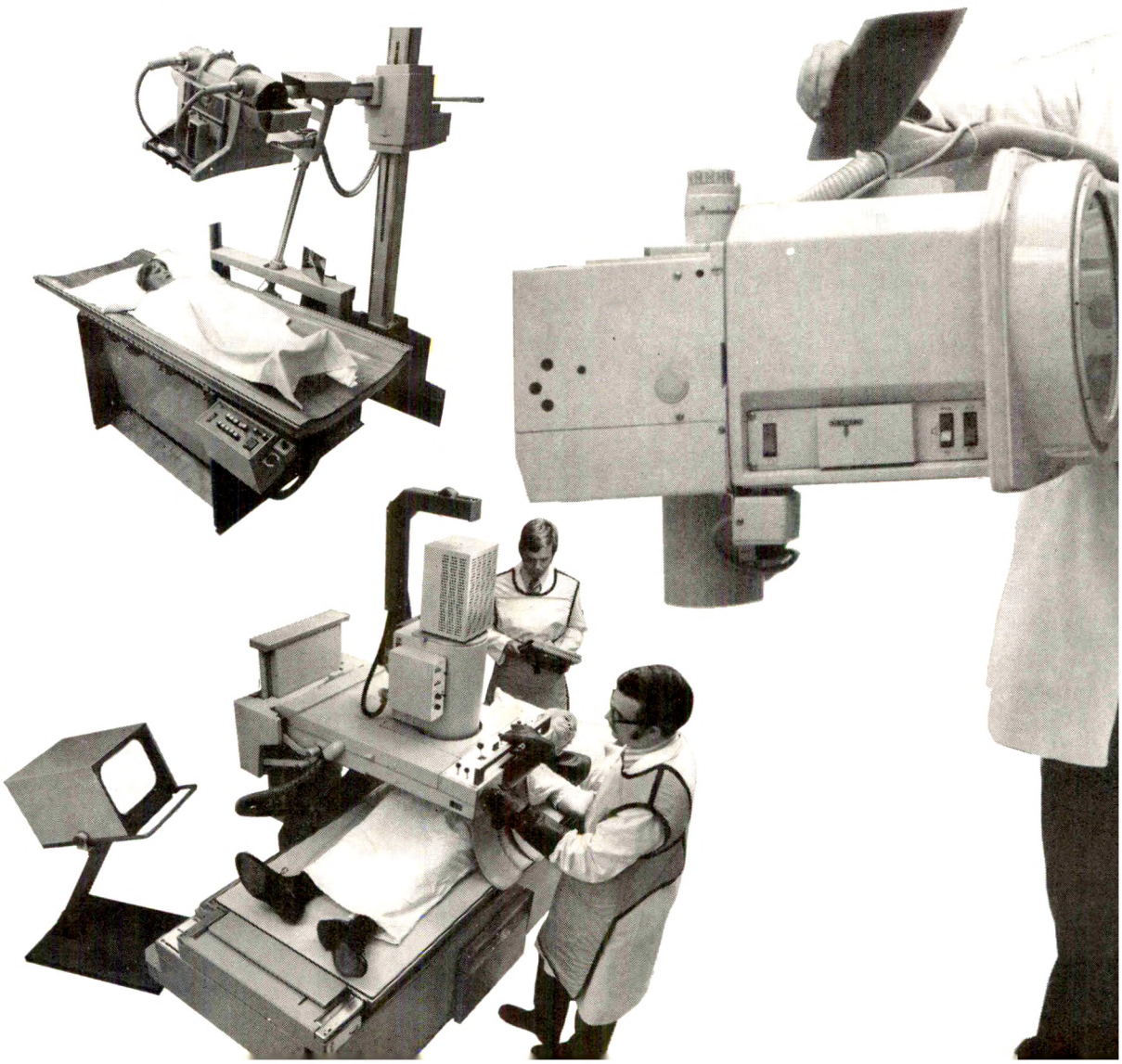
Photo: Courtesy of Western Tumor Medical Group, Inc., Van Nuys, California

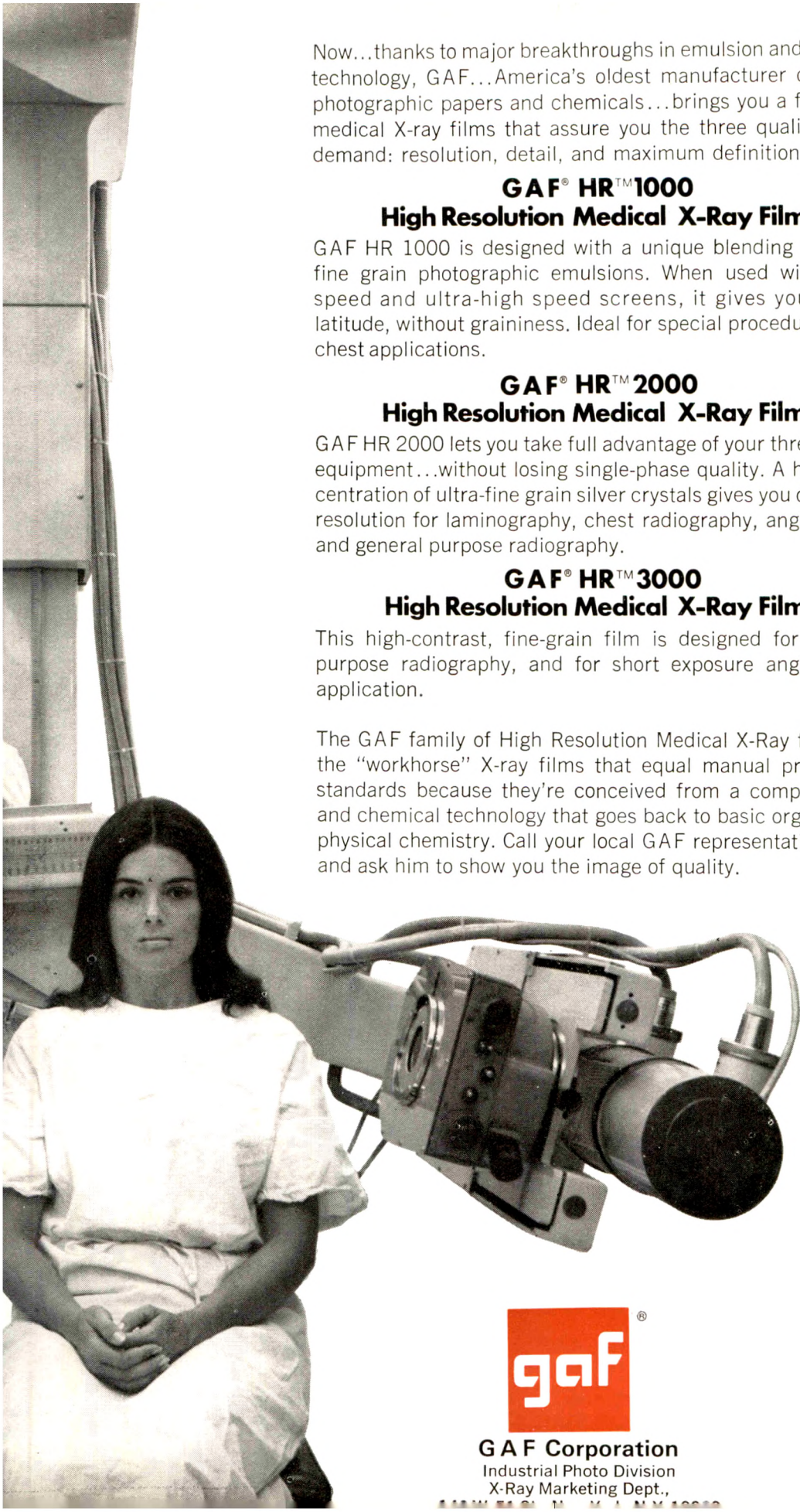


GAF has three 90-second X-ray film that don't look like 90 second X-ray films.

They look like manually-processed films!

And that's the standard of your profession.





Now...thanks to major breakthroughs in emulsion and coating technology, GAF...America's oldest manufacturer of films, photographic papers and chemicals...brings you a family of medical X-ray films that assure you the three qualities you demand: resolution, detail, and maximum definition.

GAF® HR™1000
High Resolution Medical X-Ray Film.

GAF HR 1000 is designed with a unique blending of ultra-fine grain photographic emulsions. When used with high-speed and ultra-high speed screens, it gives you image latitude, without graininess. Ideal for special procedures, and chest applications.

GAF® HR™2000
High Resolution Medical X-Ray Film.

GAF HR 2000 lets you take full advantage of your three-phase equipment...without losing single-phase quality. A high concentration of ultra-fine grain silver crystals gives you optimum resolution for laminography, chest radiography, angiography and general purpose radiography.

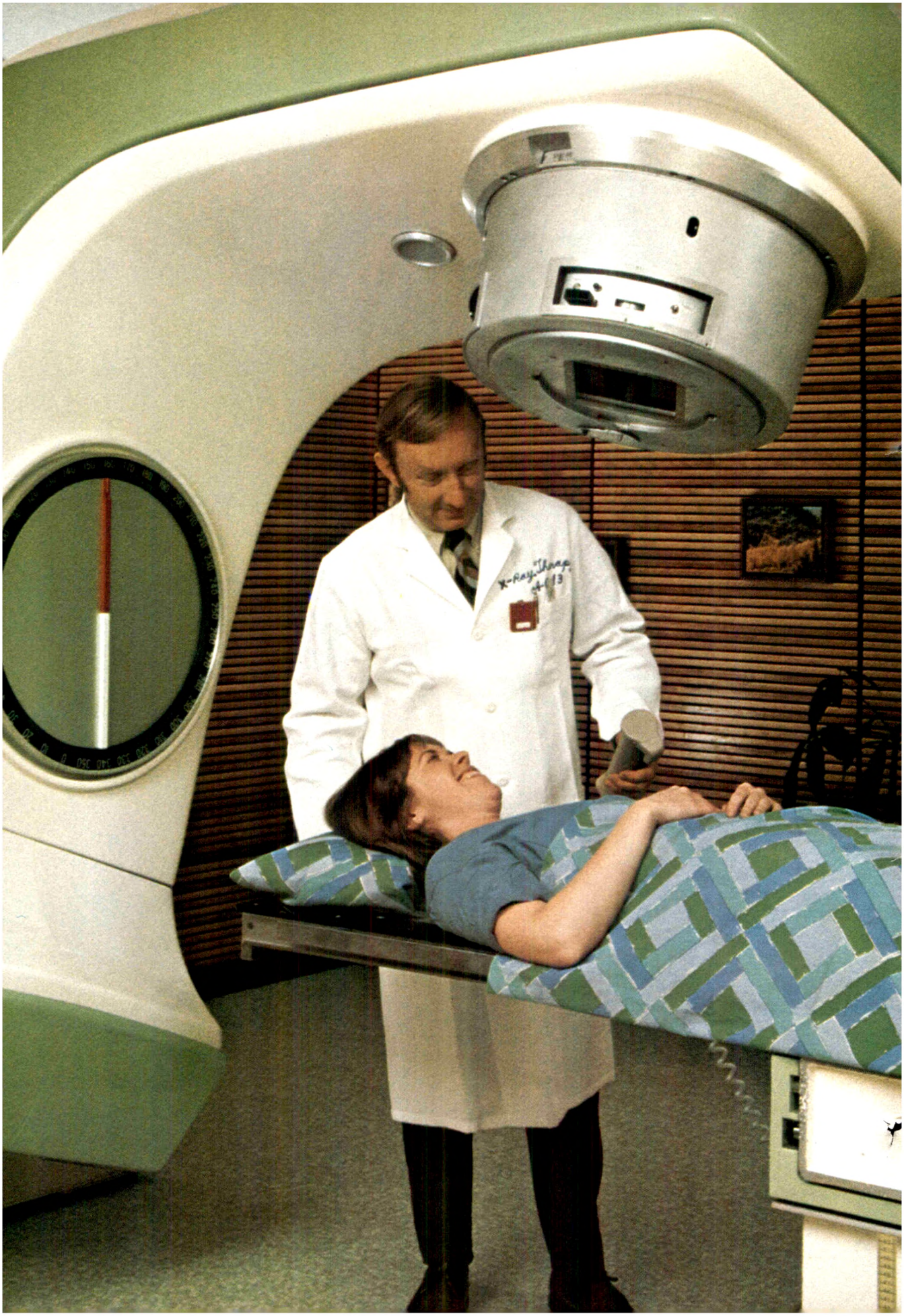
GAF® HR™3000
High Resolution Medical X-Ray Film.

This high-contrast, fine-grain film is designed for general purpose radiography, and for short exposure angiography application.

The GAF family of High Resolution Medical X-Ray films are the "workhorse" X-ray films that equal manual processing standards because they're conceived from a complete film and chemical technology that goes back to basic organic and physical chemistry. Call your local GAF representative today and ask him to show you the image of quality.

gaf®

GAF Corporation
Industrial Photo Division
X-Ray Marketing Dept.,



The Clinac® 4.

We gave it reliability for the most demanding radiotherapy schedules Plus the power and precision for special, aggressive treatment.

You won't find a more reliable radiotherapy linear accelerator than the Clinac 4. In designing it Varian's engineers knew the demanding workloads many radiotherapy machines must handle. At least 8 hours per day of constant, on-off use, month after month. The Clinac 4's unsurpassed record of reliability has now been demonstrated in over 500,000 treatments at more than 80 hospitals.

But reliability is just one part of the story. What about those cases requiring painstaking, precise, aggressive radiotherapy treatments? The retinoblastomas, the pituitaries, the prostates, the Hodgkins. The blocked fields, the wedged fields, the multi-port treatments, the abutting fields. This is where the Clinac 4 really shines.

The Clinac 4 has exceptional precision. The 3 mm spot size,

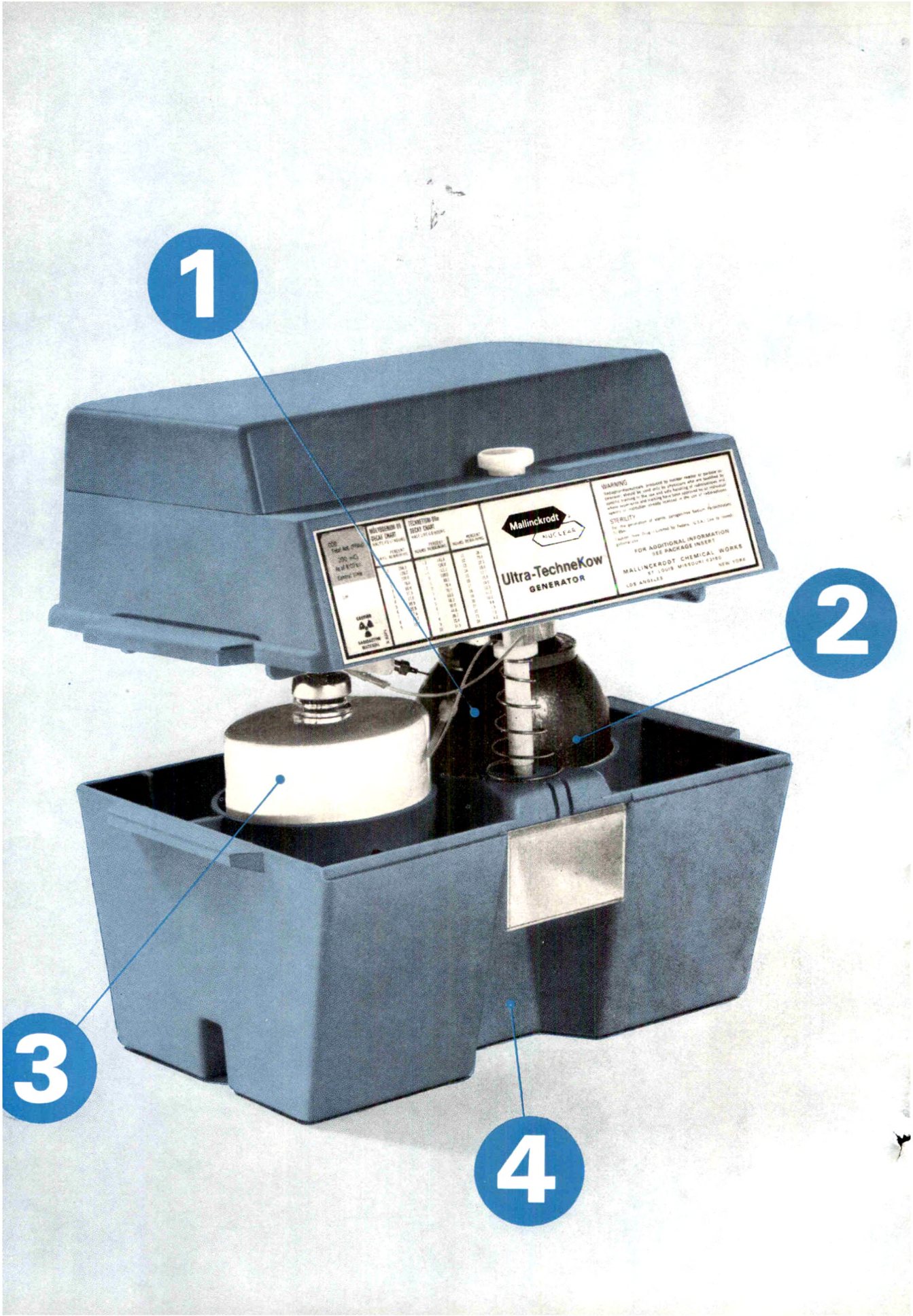


the isocentric precision of ±1 mm, the small penumbra and set-up aids—such as laser backpointer, and variable speed couch and gantry—allow extremely exact machine positioning and delivery of dose.

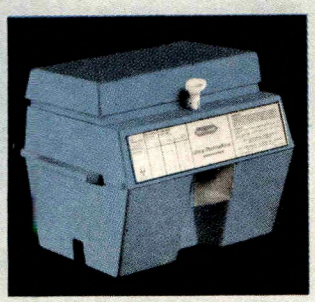
The flatness of the beam and the small penumbra are key features of the Clinac 4. You can treat both large and small volumes cleanly, homogeneously. *With an intensity capability of 350 rads/min at the isocenter,* the short treatment time minimizes the opportunity for patient movement, and the unit's millimeter tolerances can be used to maximum advantage.

If you're thinking about your radiotherapy department's need for a linear accelerator offering both workhorse reliability and power/precision for aggressive treatment, learn more about Clinac 4. Call or write Varian Radiation Division, 611 Hansen Way, Palo Alto, California 94303, (415) 493-4000; Springfield, N.J., (201) 376-6633; Detroit (Plymouth), Michigan (313) 455-1570; Chicago (Park Ridge), Illinois, (312) 825-6232; Atlanta, Georgia (404) 292-0570. In Europe: Varian A.G., Steinhausestrasse, 6300, Zug, Switzerland.

varian
radiation division


1
2
3
4
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LOS ANGELES

Introducing the New Ultra-TechneKow® Technetium Generator



with 4 New Features

1. New enlarged lead shield reduces radiation exposure to the operator. With at least 1½ inches of lead all around the generator column this is one of the best shielded generators available today.

2. New "Ion Control" Process (patent applied for) reduces aluminum level to a point where it is virtually undetectable by normal laboratory test methods. The eluate may be used with any of the currently available sulfur colloid kits or with other tagging procedures requiring low aluminum levels

3. New 500-ml saline supply allows as many as 15 or 16 elutions per week. The saline supply is built in and factory sealed, an exclusive feature of the new **Ultra-TechneKow**

4. New self-aligning milking station makes the elution process simpler than ever. When the "Sight Glass" elution shield with evacuated vial is placed into the milking station the needle is automatically centered over the evacuated vial. Press plunger down, turn slightly to lock into position, and elution proceeds automatically.

It's the most advanced concept in technetium-99m generators.

This all-new, redesigned version of our **Ultra-TechneKow** series is carefully engineered into an attractive, pre-assembled, completely self-contained unit. This model is the culmination of seven years of experience making technetium-99m generators. The **Ultra-TechneKow** Generator is shipped each week complete with evacuated elution vials, needle pack with labels, molybdenum-99 and technetium-99m reference tables needle guard for operator safety, convenient carrying handles, and package insert with complete information.

Contact your Mallinckrodt/Nuclear representative now for detailed information on this unique new product of Mallinckrodt/Nuclear research.

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RADIOPHARMACEUTICALS
Mallinckrodt Chemical Works
St. Louis, Missouri 63160

Siemens introduces a self-contained system for mammography.



SIEMENS

The Mammomat features a three-phase generator with automatic programmed exposure technique and electronic kV stabilization.

The Siemens Mammomat is a dependable, easy-to-use system for producing radiographs of the entire breast using cranial-caudal, lateral-medial, or axillary projection.

The entire system can be vertically adjusted by means of a counterpoised supporting arm so that the patient can be in a standing, sitting, or supine position. Beam projection can be rotated through 270°. Optimum positioning is facilitated with a transparent adjustable compression cone and base plate.

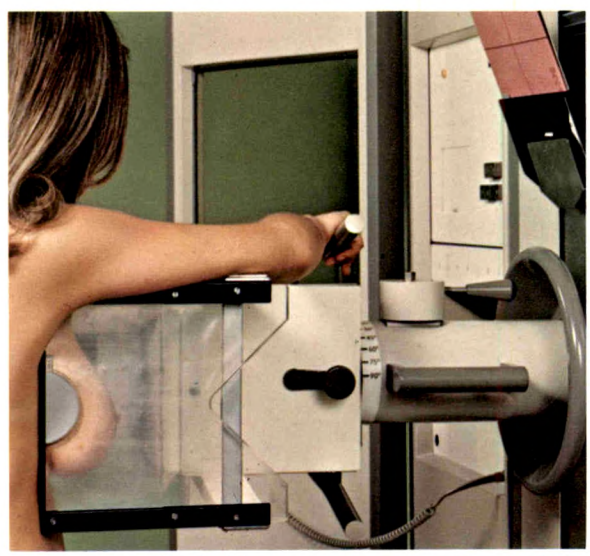
The programmed exposure of the Mammomat is controlled with push-buttons that select four different combinations of kV with the highest possible tube current and proper film density. The Iontomat automatic exposure timer provides consistent film blackening.

A new type of rotating anode tube combined with a high-power three-phase generator makes possible exposure times as short as 0.5 second.

For more information on the Mammomat, contact Siemens Corporation, Medical Systems, 186 Wood Avenue South, Iselin, New Jersey 08830.



Patient is shown here in sitting position. Standing or supine positioning is also easily accomplished.



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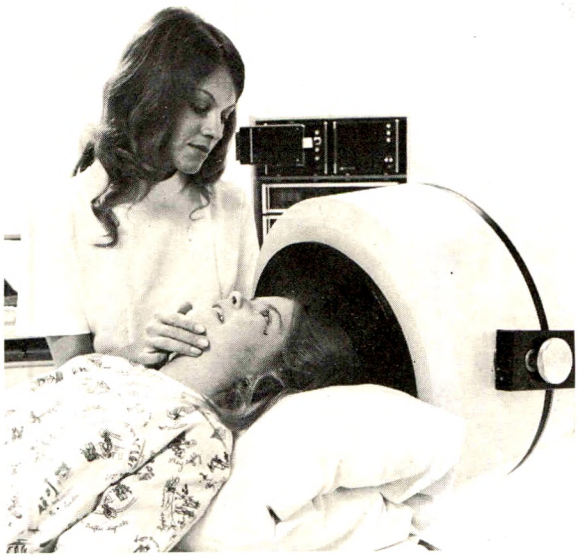


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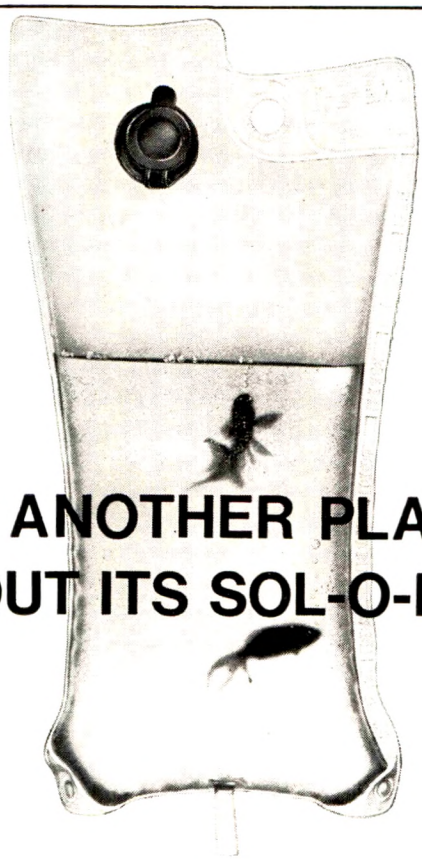
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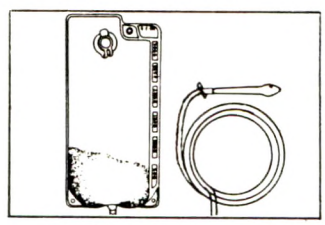
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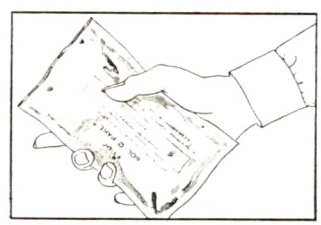
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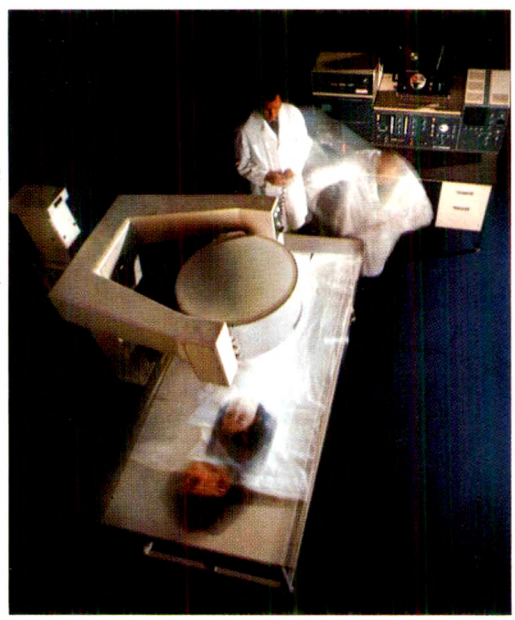
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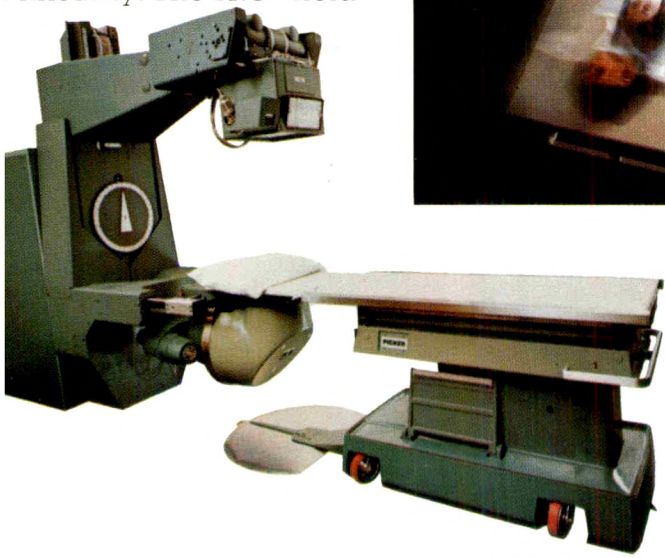
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VASCULAR THROMBOEMBOLISM COMPLICATING UMBILICAL ARTERY CATHETERIZATION*

By HUGH J. WILLIAMS, M.D., CHARLES W. JARVIS, M.D., WILLIAM A. NEAL, M.D., *and* JOHN W. REYNOLDS, M.D.
ST. PAUL AND MINNEAPOLIS, MINNESOTA

THE catheterization of umbilical vessels in the ill or potentially ill neonate has become a valuable and widely utilized procedure in the past decade. In addition to providing a convenient means of sampling blood chemistries and gases, umbilical catheters also serve as a convenient route for the administration of fluids and medication.

Thrombosis has been the most commonly reported complication of catheterization of umbilical vessels, but embolization, vascular perforation, hemorrhage, infection, blanching of limbs, and both ischemic and chemical necrosis of abdominal viscera have also been described.[12] The reported incidence of thrombosis in infants with umbilical venous catheters has ranged from 3 per cent[4] to at least 33 per cent.[17] Clinical evidence of vascular complications related to umbilical artery catheterization was noted in 3.3 per cent of 387 infants by Cochran *et al.*[2] Other investigators have suggested, however, that clinically unrecognizable thrombosis probably occurs in a significant number of such infants.[9,16] Arterial thrombosis and embolism are also occasionally encountered in newborn infants not subjected to umbilical vessel catheterization.[1] Despite the reported complications, most neonatologists believe that the potential benefits of properly performed umbilical vessel catheterization outweigh the risks of the procedure.

The purpose of this report is to document the unexpectedly high incidence of occult arterial thrombosis encountered in neonates harboring umbilical artery catheters, utilizing a prospective study and the technique of pull-out aortography.

MATERIAL AND METHOD

The study encompassed 100 consecutive infants referred to the neonatal intensive care unit of the Children's Hospital of St. Paul, between January 1 and April 15, 1971. The majority of these were premature infants with hyaline membrane dis-

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Presented also at the Seventy-second Annual Meeting of the American Roentgen Ray Society as the Award Paper of the Society for Pediatric Radiology, Boston, Massachusetts, September 28–October 1, 1971.
From the Children's Hospital, St. Paul, Minnesota; and the Department of Pediatrics, University of Minnesota, Minneapolis, Minnesota.

TABLE I

DISTRIBUTION OF CATHETERS (100 PATIENTS)

Umbilical artery	64
Umbilical vein	11
Artery and vein	4
No catheter	21

ease, but other conditions were encountered, including congenital heart disease, sepsis, seizure disorders, hypoglycemia, hypocalcemia, renal vein thrombosis and meconium aspiration syndrome. In infants who required frequent blood gas monitoring or who were at risk for serious complications, a No. 5 French Argyle polyvinyl umbilical catheter was inserted shortly after admission, according to previously described methods.[11] In all cases the insertion of an arterial catheter was the procedure of choice, and only when this failed was a venous catheter employed. Seventy-nine of the 100 infants had umbilical catheters inserted, 68 of these in an umbilical artery and 11 in the umbilical vein (Table I). A maintenance solution, usually 10 per cent dextrose with 200 units heparin/l. was constantly infused through the catheter, except during blood withdrawal. Irritating agents such as concentrated sodium bicarbonate, THAM or 50 per cent dextrose were administered slowly, unless emergency situations necessitated rapid infusion.

In 21 randomly selected infants, or approximately one-third of those with umbilical artery catheters, aortography was performed at the time of catheter removal. The aorta and subsequent run-off into the iliac and femoral vessels were demonstrated by the hand injection of renografin 60 through the umbilical artery catheter as it was withdrawn slowly over a period of several seconds. The dosage of renografin employed was 1 cc./lb. body weight, and there were no adverse reactions. The pull-out aortogram was filmed by means of a 70 mm. camera at a speed of 3 films per second with simultaneous videotape recording. Every attempt was made to expedite the procedure to prevent chilling of the infant, and in most cases performance of the examination required removal of the infant from the isolette for less than 3 minutes. In addition, the study included careful observation for thrombus formation in 12 of the 16 infants who died and were autopsied.

RESULTS

ROENTGENOGRAPHIC FINDINGS

Pull-out aortograms were obtained in 21 infants, and the roentgenologic findings are summarized in Table II. Thrombus formation was demonstrable in 18 cases. In 1 infant the aortogram was normal, and in the 2 remaining cases the study was technically unsatisfactory. The clots, which were demonstrable in 18 of the 19 infants with a satisfactory examination, assumed 4 types of configuration roentgenographically and pathologically (Fig. 1). In some infants the clot formed a circumferential fibrin sheath of varying thickness around the catheter, a phenomenon demonstrated to rapidly occur around indwelling venous catheters by Hoshal *et al.*[10] In some infants a large conventional type of pericatheteric blood clot was encountered. In other cases a mural clot occurred and produced an irregularity of the aorta, often with extension into an iliac vessel. In a few instances combinations of the preceding types of thrombi were observed in the same patient. In several infants a long clot was visualized, apparently attached proximally to the wall of the upper lumbar aorta and extending distally within the blood column like a piece of seaweed to the aortic bifurcation.

Fragmentation of the thrombus with migration of pieces of clot down the iliac and femoral vessels was observed during catheter withdrawal in several cases. In none of these infants, however, was there any subsequent clinical evidence for circulatory impairment of the lower extremities. In 1 infant (Case 15) with a large distal aortic clot, complete thrombotic occlusion of the right common iliac artery and migration of clot fragments down the left femoral

TABLE II

ROENTGENOGRAPHIC FINDINGS DURING PULL-OUT AORTOGRAPHY

Patient	Admission Diagnosis	Birth Weight (gm.)	Catheter Duration (da.)	Roentgenographic Findings
1	Neonatal seizures	4,374	1	Normal aortography
2	Pneumonitis	2,470	4	Technically unsatisfactory
3	Meconium aspiration syndrome	2,854	5	Clot in Ao and both CIA with migration down RHA
4	Meconium aspiration syndrome	3,522	3	Clot in distal Ao, RCIA and RHA
5	HMD	1,895	2	Large clot in Ao with partial occlusion of SMA
6	HMD	1,647	2	Clot in distal Ao and LCIA with complete occlusion of LFA
7	HMD	1,846	8	Clot in Ao with migration down LCIA
8	HMD	2,470	5	Clot in Ao with migration down RFA
9	HMD	1,306	11	Large clot in Ao
10*	HMD	1,136	11	Large clot in Ao extending above renal arteries
11	Congenital heart block, CHF	1,810	3	Clot in distal Ao and RCIA
12	Neonatal seizures	2,861	3	Clot in distal Ao
13	HMD	2,380	8	Technically unsatisfactory
14	HMD	2,698	8	Clot in distal Ao
15	Hypoglycemia	4,545	3	Clot in distal Ao with migration down and complete occlusion of LFA
16	HMD	3,692	12	Clot in Ao and both CIA with migration of clot down RFA
17	HMD	1,888	5	Large clot in Ao and both CIA
18	Renal vein thrombosis	2,800	14	Large clot in Ao
19	Sepsis	4,104	2	Clot in distal Ao
20	Atelectasis	2,918	1	Clot in distal Ao and LCIA
21	HMD	1,320	4	Clot in distal Ao

* Expired; HMD = Hyaline membrane disease; CHF = Congenital heart failure; Ao = Aorta; CIA = Common iliac artery; R = Right; L = Left; FA = Femoral artery; HA = Hypogastric artery; SMA = Superior mesenteric artery.

artery were noted during the pull-out procedure (Fig. 2, *A* and *B*). However, there were no clinical signs of ischemia in either leg during or following the aortogram in this infant. Only 1 infant (Case 6) had clinical signs of circulatory impairment prior to the procedure, as evidenced by blanching, coolness and diminished pulse in the left leg. Pull-out aortography in this infant demonstrated complete occlusion of the left superficial femoral artery. Following removal of the catheter and heparinization for 48 hours, the clinical evidence for ischemia disappeared. Clinical and roentgenographic evidence for bowel infarction developed in 1 infant (Case 10) with a demonstrable aortic thrombus some 4 hours following removal of the catheter (Fig. 3). At autopsy complete thrombotic occlusion of the superior mesenteric artery with massive small bowel necrosis was demonstrated. Since there were no signs of bowel ischemia prior to the withdrawal of the catheter, it seemed probable that embolization of a thrombus arising from the catheter tip or wall of the aorta occurred at the time of catheter withdrawal. In another infant (Case 5) with a large seaweed type of clot, complete occlusion of the superior mesenteric artery developed

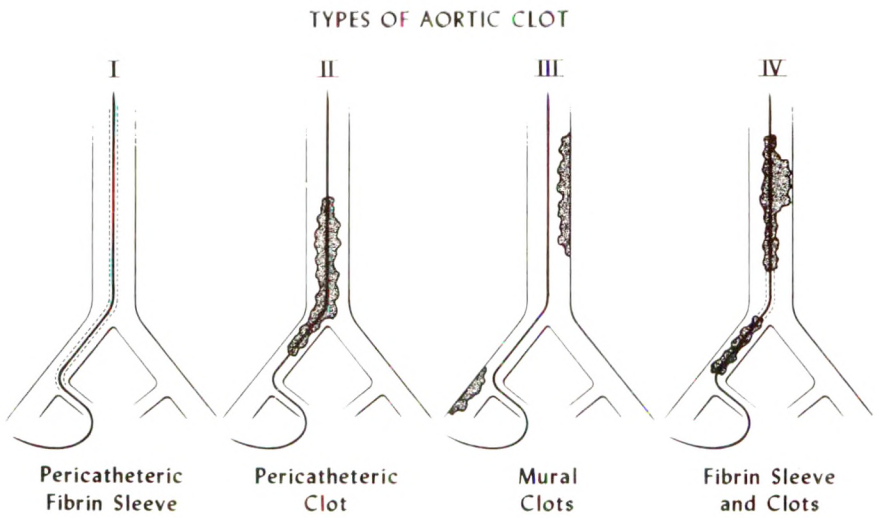


FIG. 1. The 4 types of clots encountered roentgenologically and pathologically in this prospective study are illustrated.

during the pull-out procedure (Fig. 4, *A* and *B*). Subsequently, either fragmentation or dissolution of the clot must have rapidly occurred or the collateral circulation proved adequate, since no evidence of small bowel ischemia became evident.

In many infants the catheter-bearing hypogastric and common iliac arteries were of considerably larger caliber than the corresponding vessels on the opposite side. Presumably this represented compensatory enlargement due to a relative reduction of the vessel lumen secondary to the presence of the catheter.

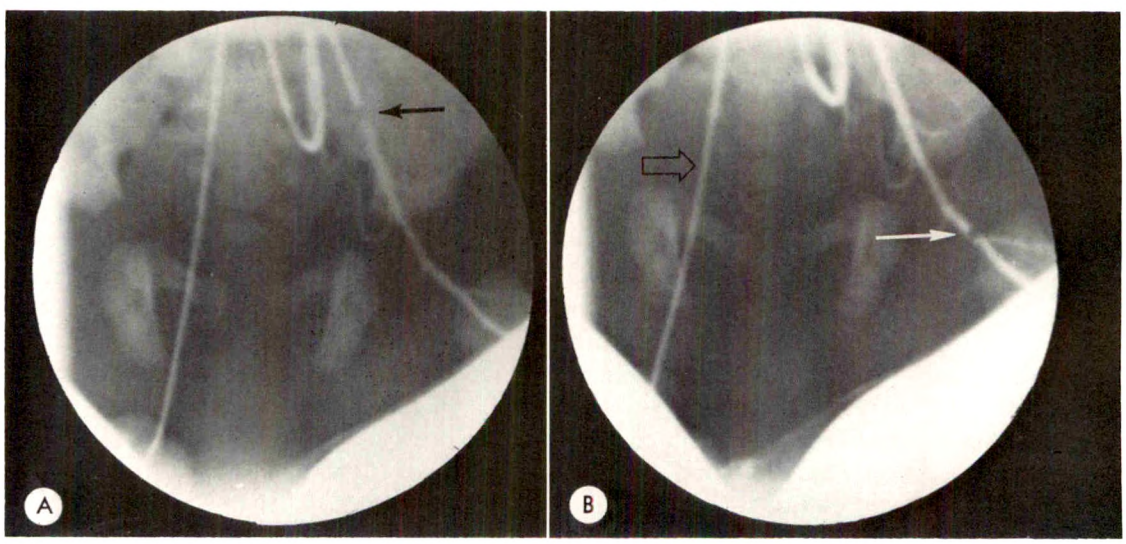


FIG. 2. Case 15. (*A* and *B*) In these exposures taken one-third of a second apart, migration of a small piece of clot down the left common femoral artery (black arrow in *A*) into the superficial femoral artery (white arrow in *B*) is demonstrated. The right common iliac artery is not filled because of a large occluding clot (not evident in this portion of the study) which extended into its proximal portion from the aorta. The opacified umbilical artery catheter (open arrow in *B*) is projected over the right pelvis and sacrum. (Reprinted with permission of *Pediatrics*.)

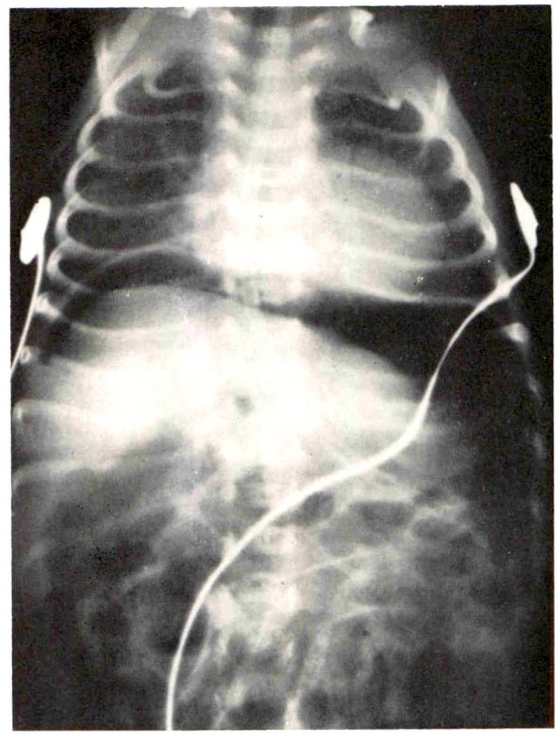


Fig. 3. Case 10. A large amount of free air is present under both diaphragms of this infant who developed necrotizing enterocolitis 4 hours after removal of the umbilical artery catheter. A large clot was noted in the abdominal aorta during pull-out aortography. At autopsy the clot occluded the superior mesenteric artery and extended into the right common iliac artery.

»»»→

NECROPSY FINDINGS

Thrombotic complications including various visceral infarcts were encountered at autopsy. These will not be emphasized since their occurrence has been previously well documented. The pathologic findings will be largely restricted to the presence or absence of catheter-associated thrombus formation.

Sixteen infants expired, 12 of whom had autopsies performed. The significant data in the autopsied cases are recorded in Table III. In all 16 infants umbilical artery catheters had been employed. Two of these patients underwent aortography prior to death, and in both instances autopsy confirmed the presence of the aortic clot demonstrated roentgenographically. Thrombus

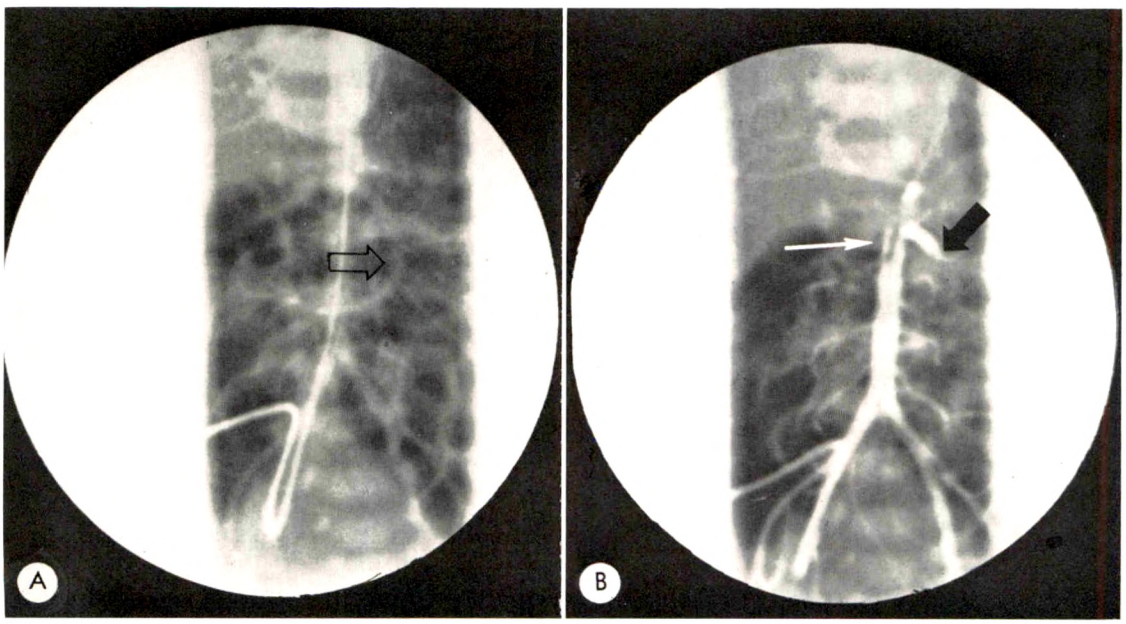


Fig. 4. Case 5. (*A*) Early filling of the superior mesenteric artery is visible (open arrow). (*B*) In an angiogram taken one-third of a second later, the superior mesenteric artery is occluded just distal to its origin (closed arrow). A long seaweed type of clot extends to the aortic bifurcation (white arrow). Despite occlusion of the superior mesenteric artery, no subsequent evidence of bowel ischemia developed.

TABLE III

INCIDENCE OF THROMBOSES AT AUTOPSY

Patient	Primary Diagnosis	Birth Weight (gm.)	Catheter Duration (da.)	Arterial Thrombus at Necropsy
A1	Atelectasis, polycystic kidneys	2,240	1	Absent
A2	Hyaline membrane disease	1,228	3	Absent
A3	Hyaline membrane disease	1,205	1	Thin pericatheteric fibrin sheath
A4	Hyaline membrane disease	1,000	1	Absent
A5	Thromboembolism fistula	2,080	7	Clot in left common iliac artery
A6*	Hyaline membrane disease	1,136	11	Large clot in aortic and right iliac artery; thrombotic occlusion of superior mesenteric artery
A7	Hyaline membrane disease	1,580	1	Pericatheteric fibrin sleeve
A8	Bowel necrosis	1,220	15	Absent
A9	Hyaline membrane disease	1,595	3	Clot in superior mesenteric artery; pericatheteric fibrin sleeve
A10	Hyaline membrane disease	950	1	Absent
A11*	Renal vein thrombosis	2,800	14	Clot in aorta with extension into right renal artery
A12	Sepsis	1,030	5	Large conventional clot around aortic catheter

* Antemortem aortography performed (Cases 10 and 18, Table 11).

formation in the vicinity of the aortic catheter was noted in 7 of the 12 necropsies. In several patients this consisted simply of a fibrin sheath surrounding the catheter, (Fig. 5, *A* and *B*) but in others the clot was of a much more extensive nature (Fig. 6; 7; and 8).

It is interesting to speculate concerning the relatively higher incidence of aortic thrombosis encountered at autopsy in our series (7/12) as compared to other investigators. Numerous factors, including the type of catheter used and the concentration of heparin in the maintenance solution administered through it could play a role. There is no question, however, that our recent interest in the problem prompted a more careful search for thrombi and fibrin sleeves at autopsy than hitherto accorded, resulting in a considerably higher incidence of clots than we ourselves had previously noted. Fibrin sleeves, in particular, may be filmy and evade detection during a casual study. The lower incidence of thrombus formation present at necropsy (58 per cent) as compared to that demonstrated angiographically is probably, at least in part, due to an inherently greater sensitivity of clot detection by angiography performed during life. Some of the clots noted during the pull-out procedure were in the process of fragmentation and embolization and would thus be expected to escape detection at autopsy.

In 7 of the 12 infants autopsied, a clot was detected, including 2 infants in whom the catheter had been present for less than 24 hours. In 5 of the 12 infants no clot was present. In 3 of these the catheter had been in position for 24 hours or less. In the other 2 infants the catheters had been inserted for periods of 3 and 15 days, respectively. The absence of detectable clot in these 2 infants is difficult to explain, since most observers have stressed that clots are almost always encountered around catheters inserted for periods exceeding 24 hours. In this regard, differences in the clotting mechanism or degrees of platelet adhesiveness between infants and adults or in infants themselves may be areas for speculation.

The microscopic findings of a typical circumferential fibrin sleeve are illustrated in

Figure 5*B*. Another fibrin sleeve, attached to the aortic wall by a clot, is illustrated in Figure 6. The clot results from intimal damage of the aorta at the site of catheter contact.

DISCUSSION

The reader is referred to the comprehensive reviews of Kitterman *et al.*[12] concerning the complications of umbilical vessel catheterization. Gupta *et al.*[9] reported a 6 per cent incidence of arterial thrombosis evident clinically or at autopsy in infants harboring umbilical artery catheters and concluded that the presence of umbilical artery catheters must increase the likelihood of actual thrombosis many times. These authors postulated that infants with no signs or symptoms of vascular occlusion might well have sustained occult injury to both the aorta and iliac vessels secondary to the catheter. Wigger *et al.*[17] reported visible clots around vascular catheters in 12.5 per cent of autopsied neonates, a 3.5 fold increase in thrombus formation as compared to autopsied infants not harboring an umbilical catheter. They also stated that the incidence of clots in survivors was unknown. Formanek and co-workers[5] reported a 54 per cent incidence of thrombus formation around the percutaneous femoral catheter, utilizing the technique of pull-out aortography, in a series of 93 diagnostic catheterizations. They found catheter as-

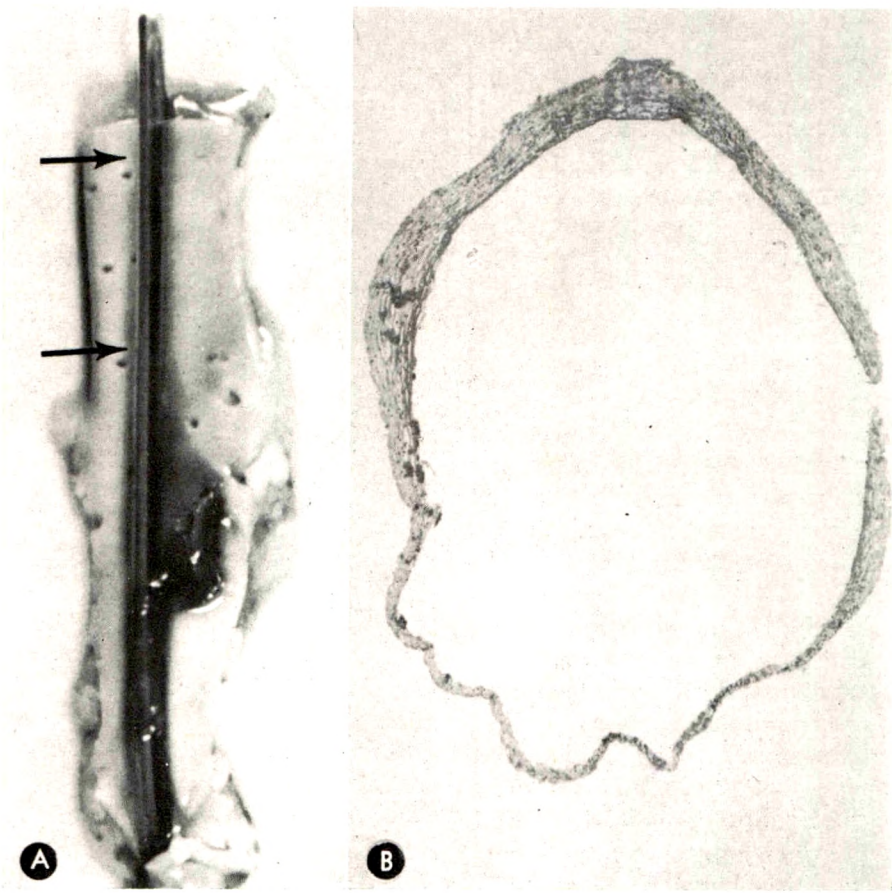


FIG. 5. (*A*) A pericatheteric fibrin sleeve (between arrows) is visible as a filmy radiolucent stripe. It extended the entire length of the catheter but only the section where it reflected light is visible in this photograph. (*B*) Cross section of the fibrin sleeve shown in *A*. In some areas the wall is only a few cells thick. The defect in the sleeve wall occurred when the catheter was removed.



FIG. 6. Cross section of another fibrin sheath. There is a broad surface where the fibrin sheath is attached to the intima of the aorta by a larger, conventional type of clot.

sociated thrombus formation present in 100 per cent of patients in whom the catheter had been present for a period exceeding 24 hours. More recently, Hoshal *et al.*[10] described the rapid encasement of indwelling subclavian vein catheters by a totally circumferential fibrin sleeve in a study utilizing vein dissection and cinefluoroscopy. In 1 of these cases an internal jugular vein clot was demonstrated by the injection of contrast medium during pull-out venography.

All the studies to date regarding arterial thrombus formation complicating umbilical artery catheterization have been retrospective analyses of autopsy cases or reports of recognizable vascular complications such as leg blanching or gangrene of an extremity. To our knowledge, this is the first prospective study conducted in neonates harboring an umbilical artery catheter in order to determine the incidence of occult thrombus formation. Only 1 infant (Case 6) demonstrated clinical evidence of ischemia which prompted the procedure, and this disappeared after 48 hours of heparin therapy. The demonstration of thrombi in 18 of the 19 patients with a technically satisfactory pull-out aortogram was surprising and rather disturbing. Despite the high incidence of clinically occult but roentgenographically demonstrable thromboembolism, there were no serious sequelae with the exception of 1 infant (Case 10) who died from occlusion of the superior mesenteric artery. In 1 infant (Case 15) with complete thrombotic occlusion of the right common iliac artery demonstrable at the time of pull-out aortography, there was no evidence of peripheral ischemia, indicating that conventional clinical criteria for predicting vascular occlusion are not always reliable (Fig. 2, *A* and *B*). The conclusion that the arterial catheter played a direct causative

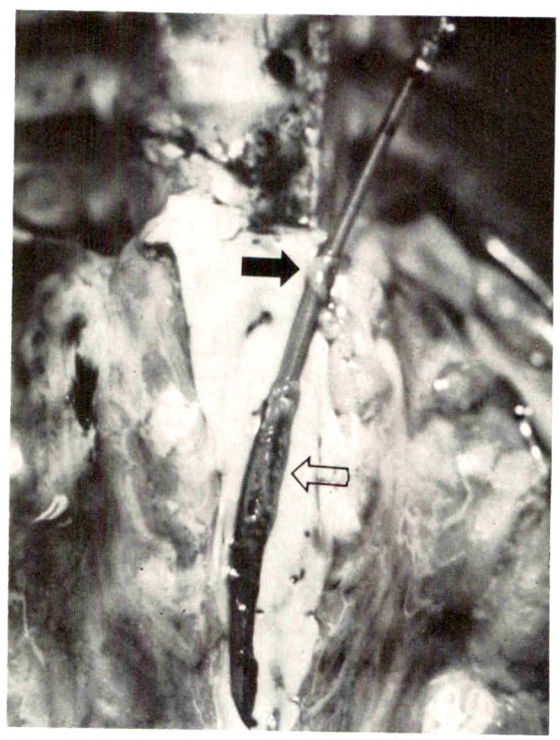


FIG. 7. The upper arrow (solid) indicates a fibrin pericatheteric sleeve attached to the adjacent wall of the aorta. The lower arrow (open) demonstrates a conventional type of clot surrounding the lower portion of the aortic catheter.

role in the strikingly high incidence of thrombosis encountered in this study is inescapable, since the incidence of antemortem thrombosis in neonates without umbilical catheters is reportedly only between 1 and 5 per cent.[8,15] Indeed, the highest rate of vascular thrombosis unassociated with catheterization that has been reported is only approximately 16 per cent and occurred in infants of diabetic mothers.[15]

The pathogenesis of thrombosis has been aptly described by Deykin.[3] Venous thrombi form in a relatively slow flowing vascular stream and resemble a blood clot, with the red cells held together by an extensive fibrin network. Therefore, heparin and oral anticoagulants which block fibrin formation are effective in preventing formation of venous thrombi. In contradistinction, in arterial thrombosis the earliest visible phenomenon is the adherence of platelets to an abnormal surface of the arterial wall or to the surface of a catheter. Fibrin formation plays a later and less prominent role in arterial thrombosis than in venous thrombosis. For this reason agents which inhibit platelet adhesiveness, such as aspirin or glyceryl guiacolate, may be more effective in preventing arterial thrombosis than heparin and oral anticoagulants. The danger of aggravating the pre-existent intracranial hemorrhage through such measures, however, cannot be ignored.

Fibrin sleeve formation around indwelling catheters originates from 2 sites of intimal injury, namely, where the catheter enters the vessel wall and where its tip contacts the adjacent vessel wall.[10] Fibrin deposition advances in both directions from these points and rapidly encases the catheter with a circumferential sheath (Fig. 5, *A* and *B*; and 8).

Most patients had coagulation studies performed, although not as a part of the study protocol. The fibrinogen levels were normal in all patients, but the platelet counts were transiently depressed in 8 patients. A relationship between the duration

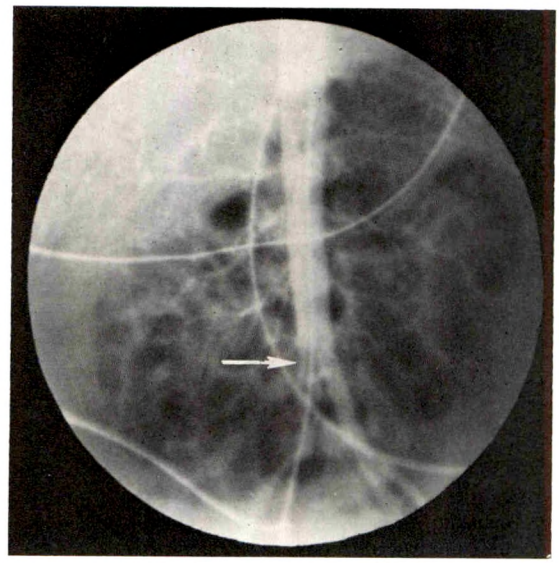


FIG. 8. Case 11. A circumferential fibrin sleeve (arrow) forms a radiolucent halo around the aortic catheter. The sleeve extends into the right iliac artery, where it is greater in diameter and causes considerable diminution in the lumen of this vessel.

of catheter placement or type of drugs administered through the catheter and clot formation was not decisively demonstrated in this study. However, thrombus or fibrin sleeve formation was roentgenologically demonstrable in every infant in whom the catheter had been *in situ* for 2 days or more. The incidence of arterial thromboses demonstrated at autopsy (7/12) was less than in our aortographic studies (18/19), possibly because in 5 of the 12 infants undergoing necropsy, the catheter had been present for 24 hours or less. We do not have an explanation for the absence of clot formation at autopsy in 2 infants harboring an arterial catheter for periods of 3 and 15 days, respectively, and in whom pull-out aortography was not performed. The fate of the clots is uncertain, but it is presumed that activation of the fibrinolytic system aids in their dissolution. With time, formation of an endothelial layer over the exposed surface of the clot occurs and the clot shrinks in size (Fig. 9). We have observed recanalization of both an occluded renal artery and opposite renal vein in 1 infant not included in this study. Although

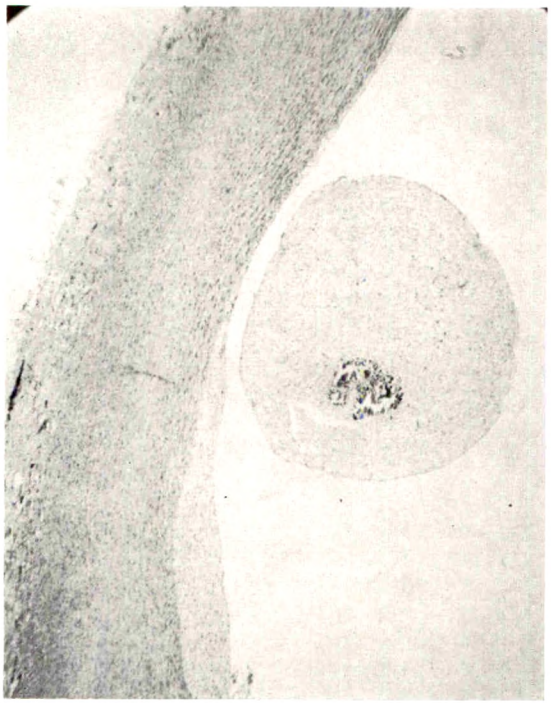


FIG. 9. Case 18. Cross section of a partially calcified organizing thrombus near its attachment to the aorta. This infant died of bronchopulmonary dysplasia 6 weeks after the catheter was removed. The aortic thrombus extended into the right renal artery and this kidney demonstrated several infarcts.

no immediate complications were noted in those surviving infants with roentgenographically demonstrated clots, close follow-up studies are warranted to check for the possibility of long term sequelae such as limb shortening. Intermittent claudication and retarded limb growth have been reported following cardiac catheterization.[13,14]

Umbilical artery catheterization has become a valuable and firmly established procedure in the management of the critically ill neonate. The unexpectedly high incidence (95 per cent) of arterial thrombus formation complicating the use of such catheters, as demonstrated in this prospective study, emphasizes the need for careful selection of patients for catheterization. It also focuses on the need for improved anticoagulant techniques and for the development of a reliable nonthrombogenic catheter. Catheters vary in their thrombogenic properties, with polyethylene and siliconized polyurethane catheters less thrombogenic than teflon.[5] Several compounds, including graphite-benzalkonium chloride-heparin sodium and cetyltrimethyl ammonium bromide have been used to coat arterial catheters, and reportedly will diminish the incidence of thrombus formation for short periods of insertion.[6,7] Although these compounds will temporarily reduce the thrombogenic characteristics of plastic catheters, a completely satisfactory nonthrombogenic material is not presently available for umbilical vessel catheterization. Further investigation of compounds which reduce platelet adhesiveness seems justified since this phenomenon plays a major role in arterial thrombosis. The high occurrence of aortic clots demonstrated in this study has prompted us to preferentially position arterial catheters in the distal abdominal aorta in order to diminish the length of intra-arterial catheter, and thus the extent of secondary thrombus formation. Such a position also reduces the likelihood of thromboembolism to the vital visceral vessels. Because of the demonstrated frequency of aortic clots, the rapid administration of solutions or employment of the "push technique" through an aortic catheter which has been in position for several days should be avoided. Administration of an exchange transfusion through an aortic catheter in 1 infant not included in this study resulted in dislodgement of an existing aortic clot and immediate embolic occlusion of the left femoral artery with clinical evidence of sudden ipsilateral ischemia (Fig. 10).

SUMMARY

1. A prospective study to determine the frequency of clinically occult arterial thromboembolism in neonates harboring an umbilical artery catheter was conducted, utilizing 100 consecutive infants admitted to our neonatal intensive care unit. Autopsies were also performed in 12 of the 16 infants who expired during the study.

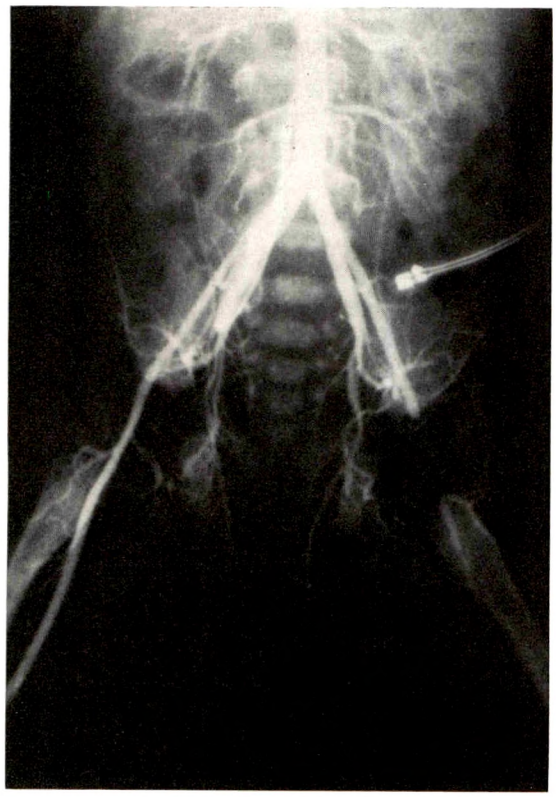


FIG. 10. There is complete occlusion of the left femoral artery in this infant by an embolus which was dislodged from the catheter tip during rapid administration of a blood transfusion. The clinical evidence of ischemia disappeared several days following removal of the catheter.

2. Pull-out aortography was randomly done in 21 infants, or approximately one-third of those in whom an arterial catheter had been inserted. Significant thrombus formation was demonstrated in 18 of the 19 infants with a technically satisfactory aortogram. One infant died as a direct result of the intravascular thrombosis.

3. Thromboembolism may occur in newborn infants and produces little or no clinical evidence of its presence. Some of the ischemic signs and symptoms encountered in neonates harboring umbilical artery catheters and attributed to vascular spasm are probably manifestations of thromboembolism.

4. The potential hazard of clot formation following umbilical artery catheterization should prompt efforts to minimize its occurrence, including careful selection of patients, development of nonthrombogenic catheters and the employment of improved anticoagulation techniques.

5. The high incidence of occult thrombosis demonstrated in this study has prompted us to position the umbilical artery catheter at the aortic bifurcation to minimize the extent of thrombus formation and the likelihood of thromboembolism to vital visceral vessels.

6. Although no immediate complications were noted in those surviving infants with roentgenologically demonstrable clots in this study, close follow-up observation is warranted to exclude the possibility of long term sequelae.

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REFERENCES

1. BRALY, B. D. Neonatal arterial thrombosis and embolism. *Surgery*, 1965, *58*, 869–873.
2. COCHRAN, W. D., DAVIS, H. T., and SMITH, C. A. Advantages and complications of umbilical artery catheterization in newborn. *Pediatrics*, 1968, *42*, 769–777.
3. DEYKIN, D. Thrombogenesis. *New England J. Med.*, 1967, *276*, 622–628.
4. ERKAN, V., BLANKENSHIP, W., and STAHLMAN, M. T. Complications of chronic umbilical vessel catheterization. *Pediat. Res.*, 1968, *2*, 317.
5. FORMANEK, G., FRECH, R. S., and AMPLATZ, K. Arterial thrombus formation during clinical percutaneous catheterization. *Circulation*, 1970, *61*, 833–839.
6. GLANCY, J. J., FISHBONE, G., and HEINZ, E. R. Nonthrombogenic arterial catheters. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *108*, 716–723.
7. GOTT, V. L., WHIFFEN, J. D., and DUTTON, R. C. Heparin bonding on colloidal graphite surfaces. *Science*, 1963, *142*, 1297–1298.
8. GROSS, R. E. Arterial embolism and thrombosis in infancy. *Am. J. Dis. Child.*, 1945, *70*, 61–73.
9. GUPTA, J. M., ROBERTSON, N. R., and WIGGLESWORTH, J. S. Umbilical artery catheterization in newborn. *Arch. Dis. Childhood*, 1968, *43*, 382–387.

10. Hoshal, V. L., Ause, R. G., and Hoskins, P. A. Fibrin sleeve formation on indwelling subclavian central venous catheters. *A.M.A. Arch. Surg.*, 1971, *102*, 353–358.

11. James, L. S. Complications arising from catheterization of umbilical vessels. In: Problems of Neonatal Intensive Care Units. Report of the Fifty-ninth Ross Conference on Pediatric Research, Stowe, Vermont, 1968.

12. Kitterman, J. A., Phibbs, R. H., and Tooley, W. H. Catheterization of umbilical vessels in newborn infants. *Pediat. Clin. North America*, 1970, *17*, 895–912.

13. Mansfield, P. B., Gazzaniga, A. B., and Litwin, S. B. Management of arterial injuries related to cardiac catheterization in children and young adults. *Circulation*, 1970, *52*, 501–507.

14. Mortensen, J. D. Clinical sequelae from arterial needle puncture, cannulation, and incision. *Circulation*, 1967, *35*, 1118–1123.

15. Oppenheimer, E. H., and Esterly, J. R. Thrombosis in newborn: comparison between infants of diabetic and nondiabetic mothers. *J. Pediat.*, 1965, *67*, 549–556.

16. Scott, J. M. Iatrogenic lesions in babies following umbilical vein catheterization. *Arch. Dis. Childhood*, 1965, *40*, 426–429.

17. Wigger, H. J., Bransilver, B. R., and Blanc, W. A. Thromboses due to catheterization in infants and children. *J. Pediat.*, 1970, *76*, 1–11.

JET COLLAPSE WITH AORTIC RUPTURE— A COMPLICATION OF UMBILICAL CATHETER ANGIOGRAPHY*

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IT IS the purpose of this communication to stress the potential hazards of excessive injection pressures using end-open umbilical catheters in newborns. The forceful, forward jet from the catheter may cause angiographic jet collapse and rupture of the aorta involving all layers, as demonstrated in one of our cases who came to autopsy.

Although vascular jet collapse and the mechanism of aortic rupture during angiography were studied in animals by Doumanian and Amplatz,[2] to our knowledge this represents the first human case with such a complication that has come to autopsy.

REPORT OF A CASE

C.M., a newborn male, was the product of an uncomplicated pregnancy and normal delivery. The patient was felt to have congenital heart disease and was sent to the University of Minnesota for further evaluation. An Argyle No. 5 French umbilical artery catheter was placed into the descending thoracic aorta; 2 cc./kg. 50 per cent diatrizoate contrast medium was injected with an effective pressure of 495 PSI; the gauge pressure was 90 PSI using an Amplatz injector with a 30 cc. syringe.

Anteroposterior and lateral roentgenograms demonstrated jet collapse of the abdominal aorta on the early studies, and immediately thereafter periaortic extravasation of contrast medium (Fig. 1, *A* and *B*).

The baby expired 48 hours later due to congestive heart failure and pulmonary congestion. The cause of death was thought to be due to hypoplastic left heart syndrome, and not related to the angiography.

At autopsy the thoracic aorta showed complete rupture, with a hematoma being confined by the adventitia. Histologic examination demonstrated disruption of the media of the aorta apparently caused by the jet from the catheter.

DISCUSSION

Since the advent of careful monitoring of blood gases through indwelling umbilical catheters, more and more infants are undergoing angiography for suspected congenital heart disease. As most of these patients in the newborn period are suffering from left-sided hypoplastic syndromes or coarctation, aortography is usually performed through the present end-hole plastic catheter. Even with low injection pressures, corresponding to 220 PSI effective pressure, the walls of the pliable aorta may be pulled into the jet resulting in angiographic jet collapse. Figure 2, *A* and *B*, shows another patient, C.W., where this phenomenon is well demonstrated with this pressure. Doumanian and Amplatz[2] have convincingly shown in canine and *in vitro* experiments that angiography through end-open catheters does cause markedly negative forces in the forward jet of injected contrast medium which may result in collapse of the vascular wall. This is more often seen in veins than in arteries, and depends largely on the injection pressure. If the effective injection pressure is increased to 800 PSI, the collapsed canine aortic wall may be cut by the jet, and an extravascular hematoma may occur (Fig. 3). Histologic examination of the damaged canine aorta shows disruption of all layers except the adventitia. Our case, contrary to the experience in animals, shows the dissection almost exclusively in the media, in the same plane as noted in dissecting aneurysms (Fig. 4).

The reported case seems to indicate that the aortic wall in newborns is very pliable and even more friable than the canine aorta, since jet collapse may be noticed at

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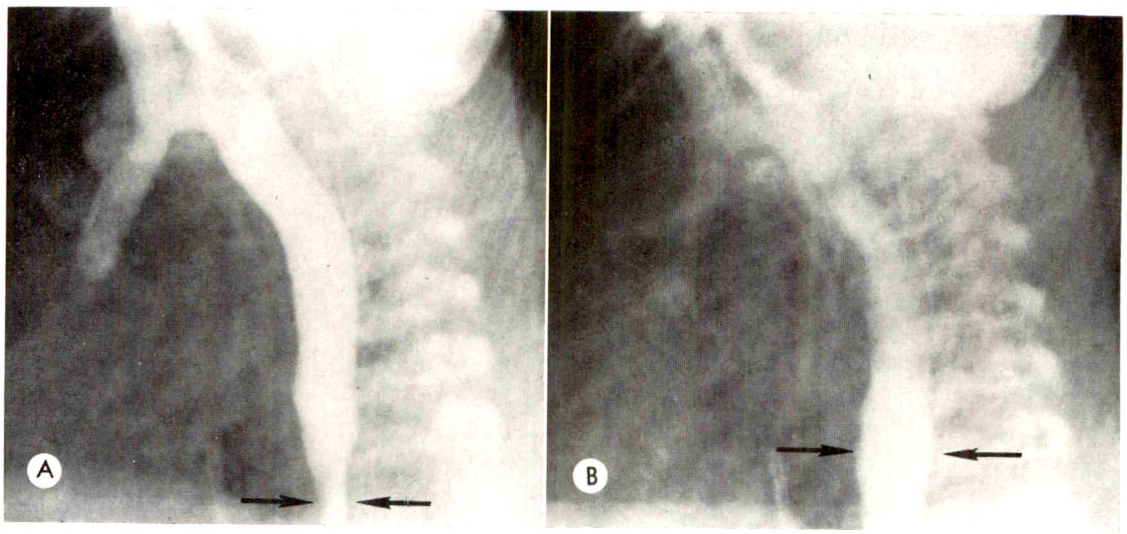


FIG. 1. (*A*) Jet collapse of the aorta during injection phase (arrows). (*B*) Extravasation of contrast medium due to aortic rupture (arrows).

pressures as low as 220 PSI effective pressure, and aortic rupture may occur at an effective pressure of 500 PSI. Jet collapse is also related to blood pressure which is much lower in critically ill newborns with congenital heart disease than in healthy dogs.

The ultimate outcome of such a vascular damage, consisting of disruption of the 2 main layers of aortic wall, is not known, but it is probable that this represents a persistent weakness, possibly resulting in a false aneurysm similar to traumatic rupture of the aorta. It can, therefore, be assumed that this represents a serious, perhaps even life-threatening complication of angiography later on in life.

The experience of this case seems to suggest the danger of high pressure angiography through end-open umbilical catheters. Experience in a large number of cases, however, has shown that effective pressures

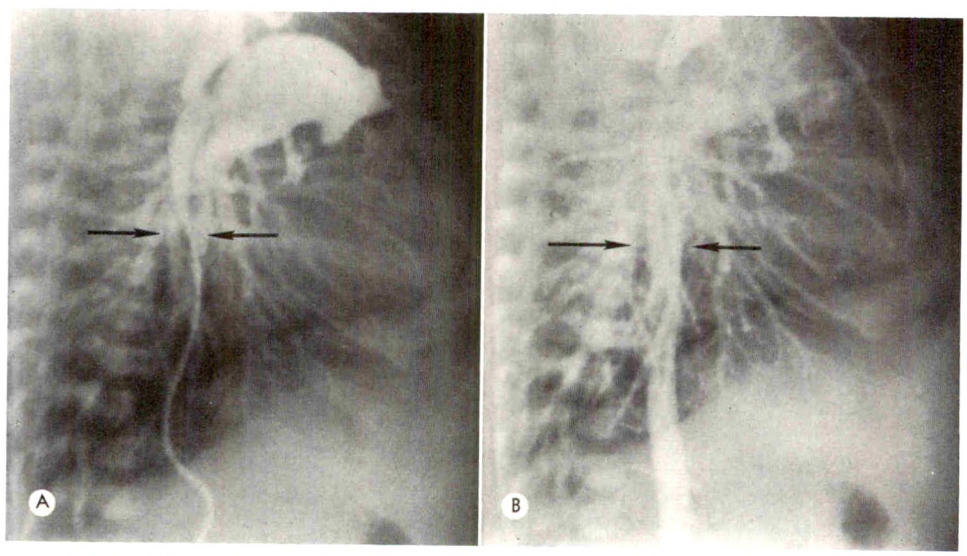


FIG. 2. (*A*) Jet collapse (arrows) with recoil of catheter but no extravasation of contrast medium. (*B*) Contrast medium clears normally from the aorta (arrows).

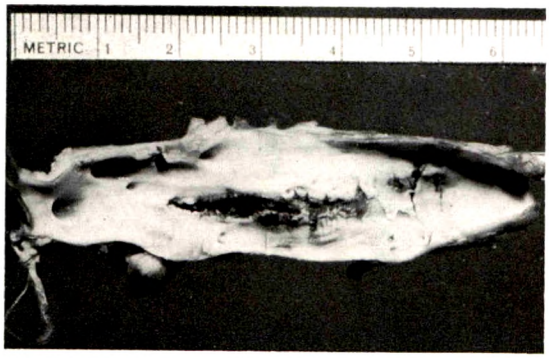


FIG. 3. Disruption of canine aorta by forward catheter jet. Rupture involves all layers except adventitia.

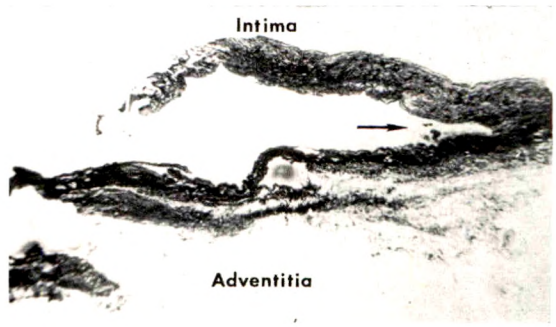


FIG. 4. Histologic section of aorta showing disruption of the media (arrow).

up to 275 PSI (50 PSI gauge pressure with a 30 cc. syringe on Amplatz injector) can be safely used. Under these circumstances the aorta may collapse, but the forward jet is not forceful enough to cause disruption and extravasation of the contrast medium. Since injection pressures cannot be determined on some flow control injectors, a maximum injection rate of approximately 6 cc. per second should not be exceeded.

SUMMARY

A case of aortic jet collapse and rupture which came to postmortem examination is reported.

The complication occurred in a newborn undergoing angiography with an end-open, indwelling umbilical catheter and an effective injection pressure of 500 PSI. At the time of autopsy, a complete rupture of the aortic wall through all layers, a small hematoma confined by adventitia, and a dissection of the media were found.

It is concluded that the aorta in newborns is easily collapsible and easily damaged by the forward jet from a high pressure injection of contrast medium. For this type of angiography, maximal effective injection pressures of 275 PSI or flow rates not exceeding 6 cc. per second are suggested.

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REFERENCES

1. AMPLATZ, K. Cardiovascular injectors. In: Vascular Roentgenology. Edited by Schobinger. The Macmillan Company, New York, 1964, pp. 91–95.
2. DOUMANIAN, H. O., and AMPLATZ, K. Vascular jet collapse in selective angiocardiography. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1967, *100*, 344–352.

SEVERE CONGENITAL HEART DISEASE IN THE NEONATAL PERIOD*

A FUNCTIONAL APPROACH TO EMERGENCY DIAGNOSIS

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SEVERE, life-threatening congenital heart disease is an important cause of infant mortality in the United States. The purpose of this paper is to present a coordinated physiologic-radiologic approach to the emergency diagnosis of cardiac lesions in the neonatal period. Emphasis is placed on the lesions most frequently encountered under 2 weeks of age. In our experience, 9 conditions account for approximately 75 per cent of children with a cardiac basis for cyanosis or respiratory distress in this age group. The methodology and logic of the diagnostic approach reflect our conviction that it is the immediate survival of the infant rather than an exhaustive anatomic and physiologic diagnosis which is important during this precarious period. This approach is designed to obtain information sufficient to allow a *functional diagnosis* and thereby indicate the correct course of action, such as a physiologically appropriate emergency surgical procedure, an initial trial of intense medical therapy or, equally important, no treatment beyond supportive measures. Since clinical diagnosis is relatively inaccurate in this age group, cardiac catheterization and angiocardiography are considered essential in the management of these patients.

MATERIAL AND METHOD

The principles described in this article have been derived from day-to-day practices of the Pediatric Cardiopulmonary Laboratory at UCLA. This catheterization laboratory is operated jointly by Pediatric Cardiology and Pediatric Radiology. Approximately 240 pediatric cardiac catheterizations are performed yearly. From 10 to 15 per cent of these investigations are in children 14 days of age or less, where the need for diagnosis is considered to be urgent and the procedure is usually carried out on an emergency basis.

PATHOPHYSIOLOGY

Approximately 75 per cent of all congenital heart lesions either remain undetected or do not produce signs or symptoms significant enough to warrant complete investigation in the neonatal period.[3,15,16,22] Infants with systemic to pulmonary shunts, even with large communications, rarely get into respiratory or cardiac difficulty in the first months of life, because of the relatively high postnatal pulmonary vascular resistance which limits left-to-right shunt volume during the neonatal period. With the exception of those infants with transposition of the great arteries, patients with cyanotic congenital heart lesions and increased pulmonary blood flow likewise rarely get into difficulty in the first weeks of life. Infants with tetralogy of Fallot, aortic stenosis or pulmonic stenosis rarely have a lesion of sufficient severity to present indications for emergency study in the neonatal period.

The 9 congenital heart lesions which commonly produce cyanosis or distress in the first 2 weeks of life and frequently require emergency diagnostic work-up are: (1) pulmonary atresia (or severe pulmonic stenosis) with intact ventricular septum (PA/PS); (2) pulmonary atresia (or severe

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pulmonary stenosis) with ventricular septal defect (PA/PS+VSD); (3) Ebstein's malformation of the tricuspid valve; (4) tricuspid atresia (TA); (5) transposition of the great arteries (TGA); (6) syndrome of coarctation of the aorta (CS); (7) total anomalous pulmonary venous return with obstruction (TAPVR); (8) hypoplastic left heart syndrome (HLH); and (9) severe coarctation of the aorta. These 9 lesions can be grouped into 3 pathophysiologic categories:

GROUP 1

Lesions with decreased pulmonary blood flow: PA/PS, TA, Ebstein's malformation, and PA/PS+VSD.[6–8,13,24] Infants with PA/PS become intensely cyanotic early because of profound underperfusion of the lungs. The majority of these patients have a severely hypoplastic right ventricle which is incapable of dilatation. About 20 per cent of the patients with PA/PS have a normally formed right ventricle which is dilated. In these cases the right atrium may also be dilated, especially if there is associated tricuspid regurgitation.

Severe reduction of pulmonary blood flow is also a principal problem in most cases of TA. In the neonate with Ebstein's malformation, the main hemodynamic abnormality is extensive tricuspid regurgitation due to a misplaced and incompetent tricuspid valve. Ebstein's malformation is functionally similar to PA/PS and TA, since pulmonary blood flow is reduced and a significant portion of systemic venous return is shunted from right to left atrium.

In contrast to the defects already mentioned, infants with PA/PS+VSD, or the extreme form of tetralogy of Fallot or pseudotruncus arteriosus, usually do not develop systemic venous congestion since they have a large ventricular defect which allows venting of the obstructed right ventricle to the systemic circulation. However, the pulmonary oligemia may be so severe that they may also require emergency intervention due to the extreme hypoxemia and resultant metabolic acidosis.

GROUP 2

Lesions with increased pulmonary blood flow: TGA and CS.[2,10] In TGA, the pulmonary and systemic circulations are parallel rather than in series. In those infants who are recognized to be cyanotic soon after birth, there is usually only a small communication between the systemic and the pulmonary circulations at the foramen ovale. Because of inadequate mixing between the circulations, these infants have severe systemic hypoxemia despite increased pulmonary blood flow.

In CS there is a ventricular septal defect (VSD) associated with a severe coarctation of the thoracic aorta. There is often an associated patent ductus arteriosus as well. This combination of the VSD and coarctation leads to massive left-to-right ventricular shunting and early congestive heart failure. Due to the coarctation, systemic vascular resistance is higher than normal and the ventricular shunt volume becomes greater earlier in these patients than in neonates with isolated VSD. Failure typically occurs under 2 weeks of age in the coarctation syndrome compared to 6 to 12 weeks with isolated VSD.

GROUP 3

Lesions with severe pulmonary venous congestion: TAPVR, HLH, and severe coarctation of the aorta with intact ventricular septum.[5,9,10,12,20] The functional common denominator in these anatomically diverse lesions is pulmonary venous hypertension and pulmonary venous congestion. Almost all infants with TAPVR who are ill soon after birth have severe obstruction to the pulmonary venous drainage along the course of the anomalous channel or at the site of entrance into the systemic venous circulation. Most of these patients have anomalous drainage below the diaphragm into the portal venous system.

In the HLH syndrome there may be stenosis or atresia of the mitral valve, severe hypoplasia of the left ventricle, atresia of the aortic valve and tubular hypoplasia of the ascending aorta. These

lesions may occur alone or in any combination. Depending upon the anatomy in a given case, all or most of the pulmonary venous return may have to find egress from the left atrium to the right atrium across the foramen ovale. In most cases, this opening is insufficient in size to accommodate the flow without left atrial and pulmonary venous hypertension.

In severe isolated coarctation of the aorta, the obstructive lesion is distal to the heart, but the principal cause of symptoms is the elevation of pulmonary venous pressure due to marked elevation of the left ventricular filling pressure with failure.

DIAGNOSTIC APPROACH

PHYSICAL EXAMINATION

All of the lesions discussed in this paper may cause cyanosis, respiratory distress or both in the first days of life. There are, nonetheless, several characteristic clinical profiles which may be helpful in differential diagnosis. Patients with TGA, PA/PS with or without VSD and TA often have marked cyanosis with little or no respiratory distress for a number of days after birth. Infants with TAPVR with obstruction and HLH syndrome characteristically have marked tachypnea and a dyspneic appearance, but only slight or questionable cyanosis. A unique and changing pattern of findings is characteristic of Ebstein's malformation. Immediately after birth the infant has cyanosis, tachypnea, a loud systolic murmur and hepatomegaly, all of which undergo spontaneous improvement over the first few weeks of life.

Most neonates with TGA and HLH syndrome have little or no murmur, whereas patients with CS, Ebstein's malformation and TA have a prominent systolic murmur along the lower left sternal edge. Unless there is an associated patent ductus arteriosus which provides flow to the lower extremities from the right ventricle, patients with both isolated coarctation and CS have a diagnostic discrepancy in brachial and femoral pulses and pressures.

ELECTROCARDIOGRAM

The electrocardiogram (ECG) shows a normal frontal plane axis and right ventricular preponderance expected at this age in the majority of the lesions which present under 2 weeks of age. With Ebstein's malformation, first degree heart block and prolonged right ventricular conduction may be present from birth or appear later. Marked right ventricular hypertrophy is typical of TAPVR with obstruction, reflecting severe right ventricular hypertension. In about 80 per cent of patients with PA/PS the right ventricle is hypoplastic and this is reflected in the ECG as a relative absence of right ventricular forces and a normal frontal plane axis. The diagnosis of TA is virtually assured in a cyanotic infant whose ECG shows left axis deviation, right atrial enlargement and left ventricular hypertrophy. Combined ventricular hypertrophy and normal axis is the most common electrocardiographic pattern in patients with CS, while the ECG in infants with isolated coarctation shows right ventricular hypertrophy.

CHEST ROENTGENOGRAM

The upright posteroanterior roentgenogram of the chest is very important in the diagnosis of congenital heart lesions in the neonatal period.[3,14,18] The lateral roentgenogram is only occasionally helpful. It is rarely necessary to undertake a cardiac series with barium in the esophagus. The critical information to be obtained from the chest roentgenogram is evaluation of cardiac size and pulmonary vasculature. The cardiac configuration is rarely helpful in this age group. On the basis of the chest roentgenogram, the 9 lesions can usually be divided into 3 categories. Within each group there are helpful differentiating features.

It must be realized that while in the majority of cases the sharp differentiations outlined below are applicable, there will be cases where this differentiation will prove

to be difficult primarily because of uncertainty about the pulmonary vascularity.

Group 1

Infants with chest roentgenogram showing decreased pulmonary vascularity: If the heart is large in this group, the likelihood is that the patient has PA/PS (Fig. 1),[6] or Ebstein's malformation.[1] Gross enlargement of the cardiac silhouette, particularly of the right atrium, is somewhat more characteristic of Ebstein's malformation than PA/PS. If the chest roentgenogram shows a normal or only moderately enlarged heart, the likelihood is that the infant has PA/PS+VSD (Fig. 2), or PA/PS with an intact ventricular septum and a hypoplastic right ventricle. In TA the heart is either normal in size or slightly enlarged (Fig. 3). Discrete enlargement of the right atrium is found in some but not all cases of TA.[7,24]

Group 2

Infants with chest roentgenogram showing normal or moderately increased pulmonary vascularity of the arterial type: These infants probably have TGA (Fig. 4),[2] or CS (Fig. 5).[16] Most commonly infants with TGA have the transposition of the great vessels without inversion of the ventricles. TGA is one of the few lesions which has a characteristic cardiac contour in the neonatal period. The familiar "egg-shaped" heart is due to a narrow cardiac waist, convexity of the entire left heart border and modest prominence of the right atrium. Infants with CS have a large heart, increased pulmonary blood flow, plus some degree of pulmonary venous congestion. Identification of the coarctation on the chest roentgenogram is usually not possible in this age group.[9]

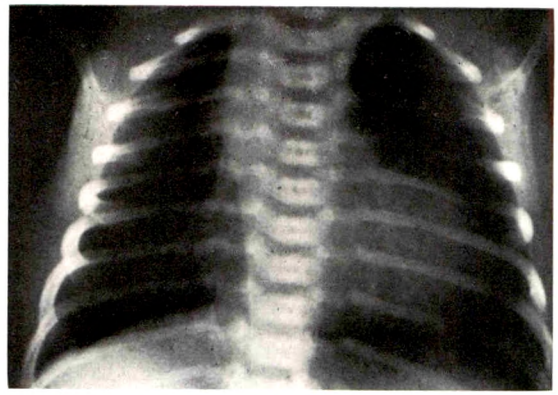


Fig. 2. *Severe tetralogy of Fallot.* The heart is slightly enlarged with an elevated apex, shallow cardiac waist and right aortic arch. The pulmonary vascularity is decreased.

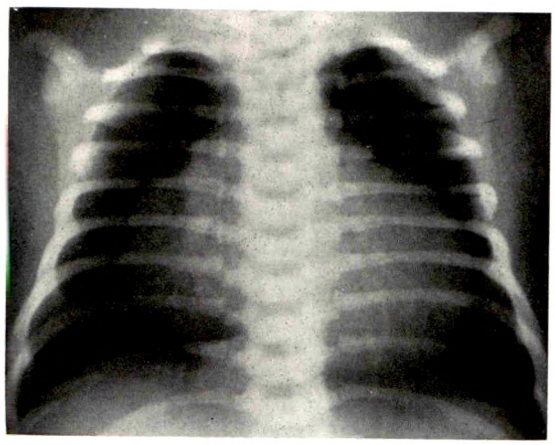


Fig. 1. *Severe pulmonic stenosis.* The heart is markedly enlarged with decreased pulmonary vascularity. The prominence of the right inferior heart border is due to moderate enlargement of the right atrium secondary to tricuspid insufficiency.

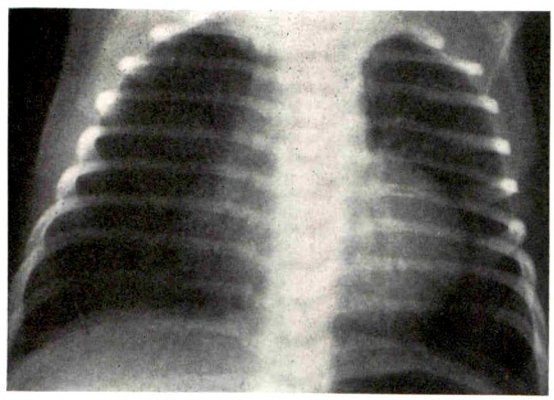


Fig. 3. *Tricuspid atresia.* The heart is slightly enlarged with an elevated apex, shallow cardiac waist and left aortic arch. The prominence of the right inferior heart border is due to moderate enlargement of the right atrium. The pulmonary vascularity is decreased.

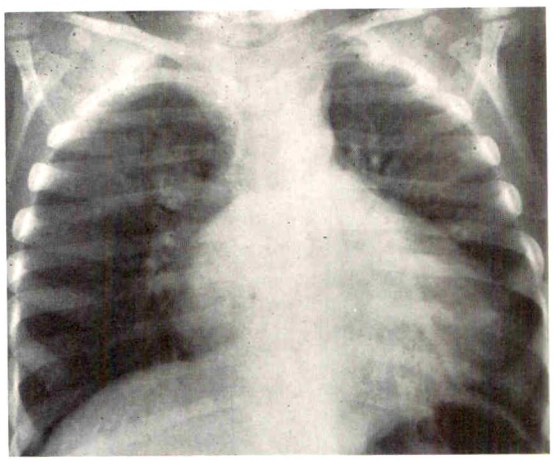


FIG. 4. *Transposition of the great arteries.* The heart is enlarged with narrow cardiac waist. There is increased pulmonary vascularity of the arterial type.

Group 3

Infants with chest roentgenogram showing pulmonary venous congestion: If the chest roentgenogram suggests venous congestion and also shows cardiomegaly, the likelihood is that the infant has either severe isolated coarctation of the aorta,[9] or some form of HLH syndrome.[10] Heart size may be normal with HLH syndrome in the first day of life (Fig. 6). If the chest roentgenogram reveals severe congestive changes with normal or slightly enlarged heart, the infant is more likely to have TAPVR with obstruction (Fig. 7).[12,20]

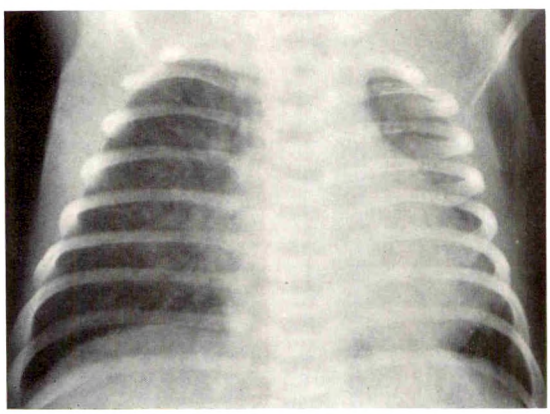


FIG. 5. *Coarctation syndrome.* The heart is moderately enlarged. The cardiac waist is normal in size and configuration. There is increased pulmonary vascularity of the arterial type.

CARDIAC CATHETERIZATION AND ANGIOGRAPHY

Cardiac catheterization and angiocardiography are so closely related in the emergency work-up of congenital heart lesions that they must be considered part of the same diagnostic procedure. Unlike elective cardiac catheterization and angiography in which it is customary to collect all of the physiologic data and then proceed to selective angiocardiography, in emergency situations the physiologic and angiographic procedures need not follow each other in any prearranged order.

A functional and, if possible, exact anatomic diagnosis needs to be established in a minimum of time with minimal amounts of contrast medium.[17,23] It is desirable to limit the total dose of contrast material to 2 cc./kg.–3 cc./kg. whenever possible and 4 cc./kg. is considered by ourselves and others to be an arbitrary upper limit of safety in the sick neonate. To accomplish this goal, the potential value of each angiocardiogram must be carefully weighed before they are actually performed. It must be remembered that cardiac catheterization and angiocardiography represent additional stresses to the already precarious status of the profoundly ill infant.

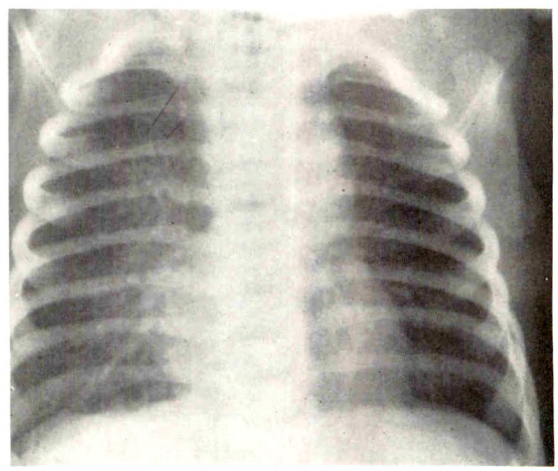


FIG. 6. *Hypoplastic left heart syndrome.* The heart is not enlarged. There is moderate pulmonary venous congestion. The heart may be of normal size in the hypoplastic left heart syndrome in the first few days of life, although it usually becomes larger as the congestive heart failure increases.

Therefore, the diagnostic procedure must be as brief as possible and the patient adequately monitored. *The need to measure and support the infant's temperature and repeatedly assess and correct acid-base balance during the procedure cannot be overemphasized.*

A formal and schematic approach to emergency cardiac catheterization and angiography is outlined below. This scheme is particularly applicable when the diagnosis is quite uncertain prior to cardiac catheterization and although it is presented in steps, it can and should be deviated from depending on circumstances.

The use of a biplane cine system reduces the amount of contrast material and the number of injections needed and shortens the diagnostic procedure. Its use is most advantageous and highly recommended for studies in infants. In most of these problems, posteroanterior and lateral projections are best.

Emergency cardiac catheterization in this age group is usually best begun by the umbilical approach using a soft polyvinyl catheter.[19] It is wise to begin by catheterizing an umbilical artery and to immediately obtain an arterial sample for pH, pCO_2 and pO_2 *regardless* of the suspected diagnosis.

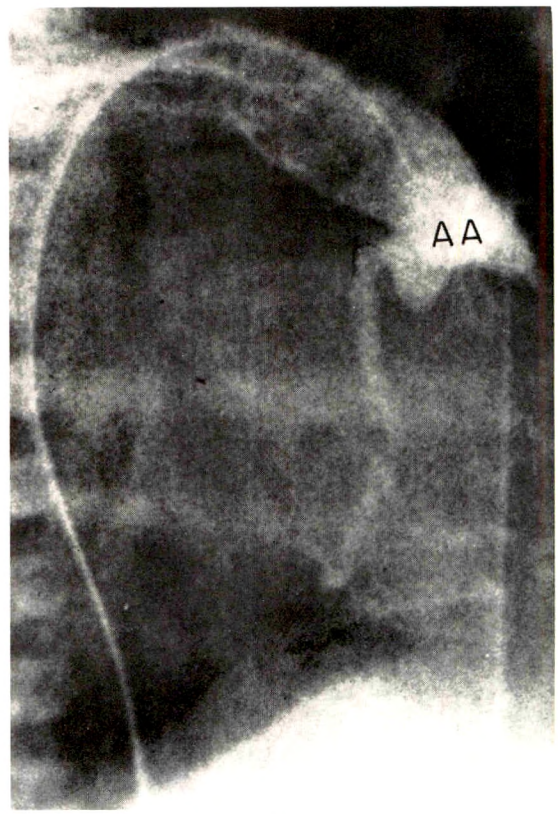


FIG. 8. *Transposition of the great arteries.* The catheter curves anteriorly. The aortic arch is "open" and the aortic valve ring is high in position. AA = Ascending Aorta.

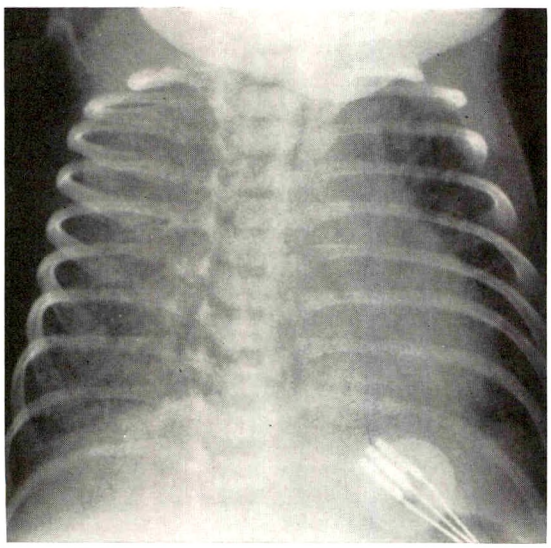


FIG. 7. *Total anomalous pulmonary venous return* below the diaphragm with obstruction. The heart is slightly enlarged with considerable pulmonary venous congestion.

Step 1. The left heart is approached in a retrograde manner through an umbilical artery. As the catheter is advanced into the ascending aorta an anterior or retrosternal position of the catheter tip viewed in the lateral plane strongly suggests the diagnosis of transposition (Fig. 8). This diagnosis should be confirmed by an aortogram because the catheter can readily pass anteriorly through a patent ductus arteriosus into a normally placed pulmonary artery. An aortogram is also necessary to rule out associated aortic arch anomalies. If the diagnosis is transposition, one should proceed directly to balloon atrial septostomy. If the patient remains stable after these steps have been carried out, maneu-

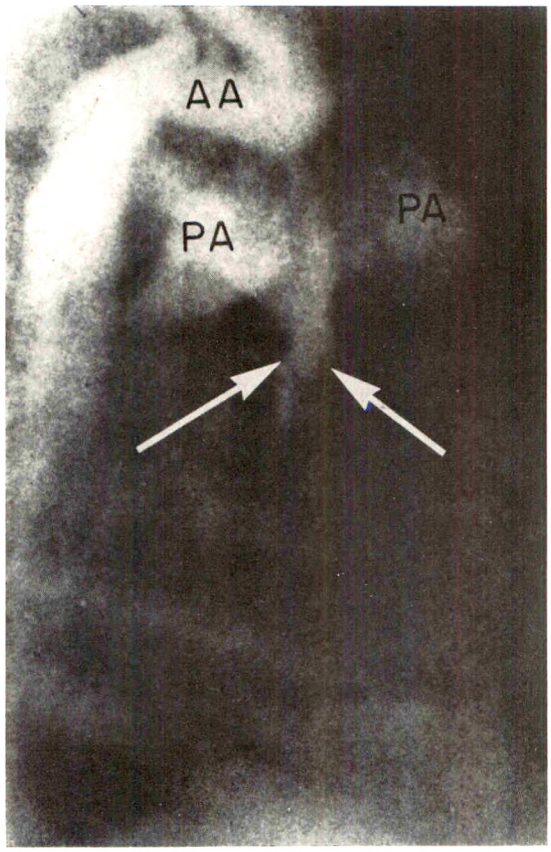


FIG. 9. *Hypoplastic left heart syndrome.* Lateral view. Injection into the descending aorta shows a normal arch and descending aorta, and retrograde filling of the small, hypoplastic ascending aorta (arrows). There is also filling of the rather large pulmonary artery through a patent ductus arteriosus which is not clearly seen. AA = Aortic Arch; PA = Pulmonary Artery.

vers to evaluate the ventricular septum, pulmonary outflow tract and pulmonary pressures should be undertaken.

If the catheter cannot be advanced around the arch of the aorta or if it has a tendency to go repeatedly into the ductus arteriosus, then a second catheter inserted into the other umbilical artery may allow passage around the arch of the aorta. If the ascending aorta still cannot be entered, a small volume "sighting" injection should be performed just distal to the point of apparent obstruction. In this fashion a coarctation or other arch anomaly may be diagnosed. If there is minimal or no antegrade blood flow in the ascending aorta as with atresia of the aortic valve or hypoplasia of the left ventricle, this injection will readily fill the aortic arch, hypoplastic ascending aorta and coronary arteries in a retrograde fashion (Fig. 9).

If no arch anomaly or transposition is present, the tip of the arterial catheter is left in the ascending aorta, since it subsequently may be desirable to manipulate it across the aortic valve or record simultaneous aortic and ventricular pressures following venous catheterization.

Step 2. Following umbilical artery catheterization the umbilical vein should be catheterized. In the first few days of life, and especially in hypoxic or distressed infants, it is usually possible to enter the right atrium through the ductus venosus. If the ductus venosus has closed, the saphenous vein is the entry of choice. The pressure in the right atrium and even more importantly, the size of the right atrium are often revealing.[8] The giant right atrium and displacement of the tricuspid ring to the left can frequently give a clue to the diagnosis of Ebstein's malformation prior to injection of contrast material. *However, it must be emphasized that every effort should be made to enter the right ventricle even if it is difficult to do so, or difficult to maintain the catheter in it.* The reason for this is that PA/PS, PA/PS with hypoplastic right ventricle and Ebstein's malformation may all present with a large right atrium. Injection into the right ventricle will usually reveal regurgitation into the right atrium in all of these lesions, but only a right ventricular injection will demonstrate the size of the cavity and confirm the presence or absence of pulmonary atresia or severe pulmonic stenosis (Fig. 10). In PA/PS + VSD, the right atrial size will be normal and injection into the right ventricle will reveal immediate filling of a large ascending aorta through the ventricular defect.[11,13] The pulmonary outflow tract is frequently poorly seen because of its small size (Fig. 11, *A* and *B*), and it is rarely possible to clearly define the status of the pulmonary valve in these patients due to the marked

decrease in pulmonary flow. *In general, in lesions with decreased pulmonary blood flow, the right ventricular injection is the most critical one.*

If the right ventricle cannot be entered, after repeated attempts, then the possibility of TA is very real. However, inability to enter the right ventricle cannot be taken as proof of this diagnosis. In these cases, we prefer injection at the junction of the right atrium and superior vena cava to show absence of early filling of the right ventricle and immediate shunting of contrast medium from the right atrium into the left atrium, left ventricle and ascending aorta. Delayed opacification of a small right ventricle and pulmonary artery with contrast medium from the left ventricle will substantiate the diagnosis of TA.

Step 3. If the preceding steps yield no results, then the catheter should be advanced into the pulmonary artery. If pulmonary arterial pressure is at or above aortic pressure, severe obstruction beyond the pulmonary capillaries should be suspected. In this case the pulmonary circulation time may be quite prolonged. For this reason the filming of the pulmonary injection should continue until the site of return from the pulmonary veins can be ascertained. Since anomalous return with obstruction is frequently below the diaphragm, the angiocardiographic field should include the area of the liver as well as the thorax (Fig. 12).

Step 4. At this point there may still be insufficient information to adequately establish a diagnosis, as tends to be the case in some forms of the HLH syndrome. In these instances, the left atrium should be catheterized through the foramen ovale, which usually presents no difficulty in the neonatal period. Likewise, it is usually possible to advance the catheter from the left atrium into the left ventricle. Injection into the left atrium or ventricle will help in fully assessing the size and character of the left ventricle in the HLH syndrome, and it will often assist in fully visualizing severe coarctation of the aorta (Fig. 13, *A* and *B*). In instances of left-to-right shunting, a left ventricular injection may allow differentiation of ventricular from aorticopulmonary communications.

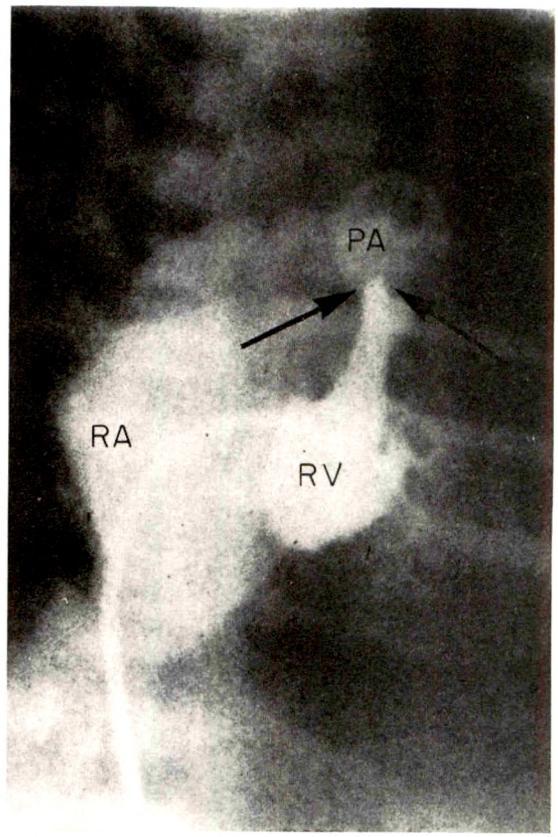


Fig. 10. *Severe pulmonic stenosis with hypoplastic right ventricle.* The injection was made into the small right ventricle. The arrows point to the thickened valve leaflets. RA = Right Atrium; RV = Right Ventricle; PA = Pulmonary Artery.

Principles of Emergency Therapeutic Management

Group I

Lesions with decreased pulmonary vascularity: With the important exception of patients with Ebstein's malformation, infants in this group are candidates for an emergency aorticopulmonary shunt (Waterston or Potts procedures), if they are hypoxic enough to have metabolic acidosis or hypoxic episodes, or if they have a patent ductus arteriosus, as the primary source of pulmonary blood flow. In the patient with pulmonary atresia and a normal right

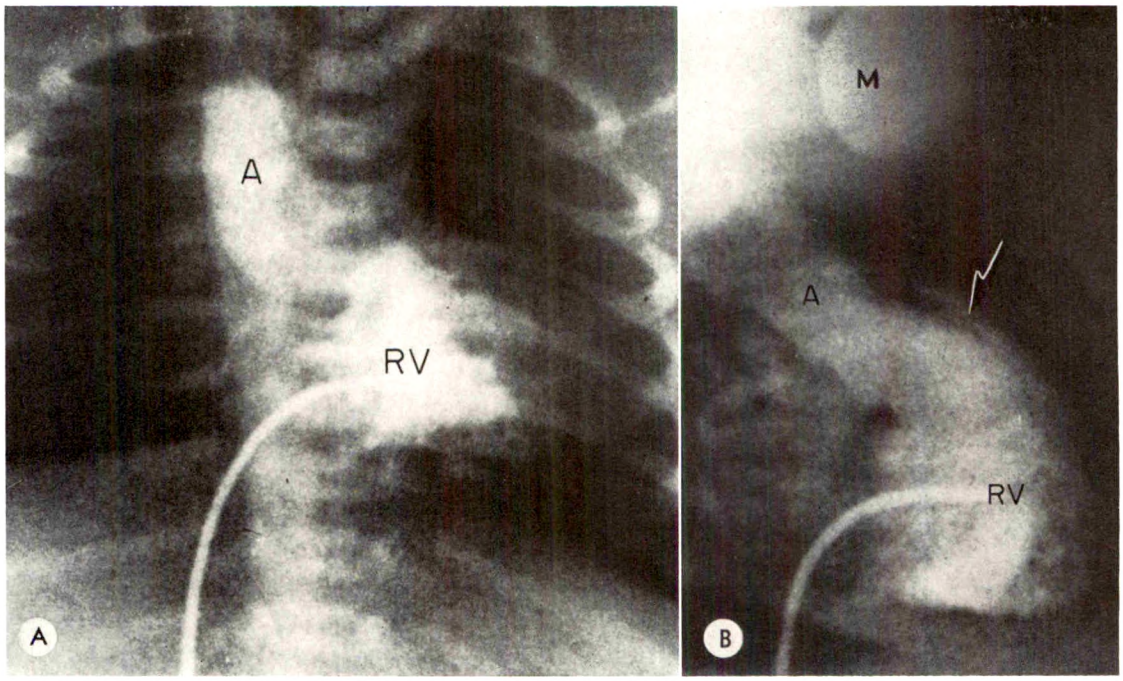


FIG. 11. (*A* and *B*) *Severe tetralogy of Fallot.* Posteroanterior and lateral views. The injection was made into the right ventricle. There is immediate filling of the right-sided aorta through a high ventricular septal defect. The small pulmonary outflow tract is not seen in the posteroanterior view and the arrow points to it in the lateral view. RV = Right Ventricle; A = Aorta; M = Monitor.

ventricle, an attempt may be made to open the atretic pulmonary valve instead of performing a shunt procedure. The severity of tricuspid regurgitation and right-to-left atrial shunting in Ebstein's malformation is largely dependent upon the level of pulmonary vascular resistance. Since patients with this lesion usually show spontaneous improvement as pulmonary vascular resistance falls, shunt surgery is not indicated.

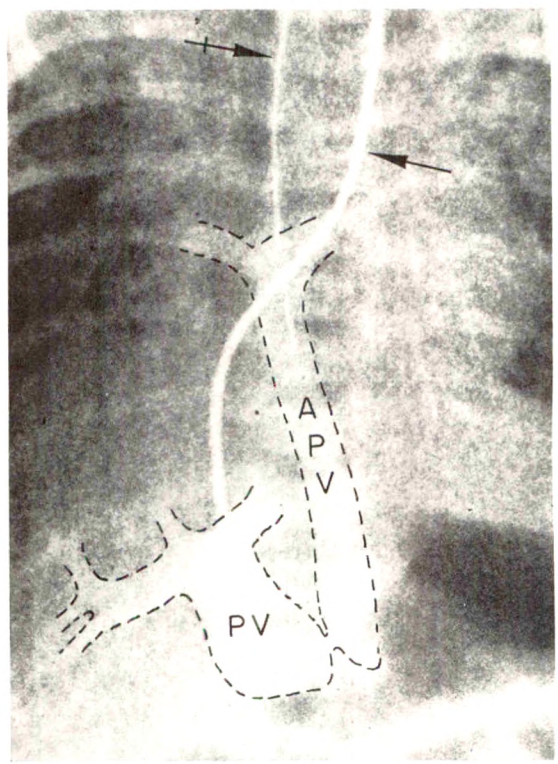


FIG. 12. *Total anomalous pulmonary venous return.* The injection was made into the pulmonary artery. The arrow points to the pulmonary artery catheter. The pulmonary veins collect into 2 anomalous channels which join and cross the diaphragm and feed into the portal vein (___). The crossed arrow points to a feeding tube. APV = Anomalous Pulmonary Vein; PV = Portal Vein.

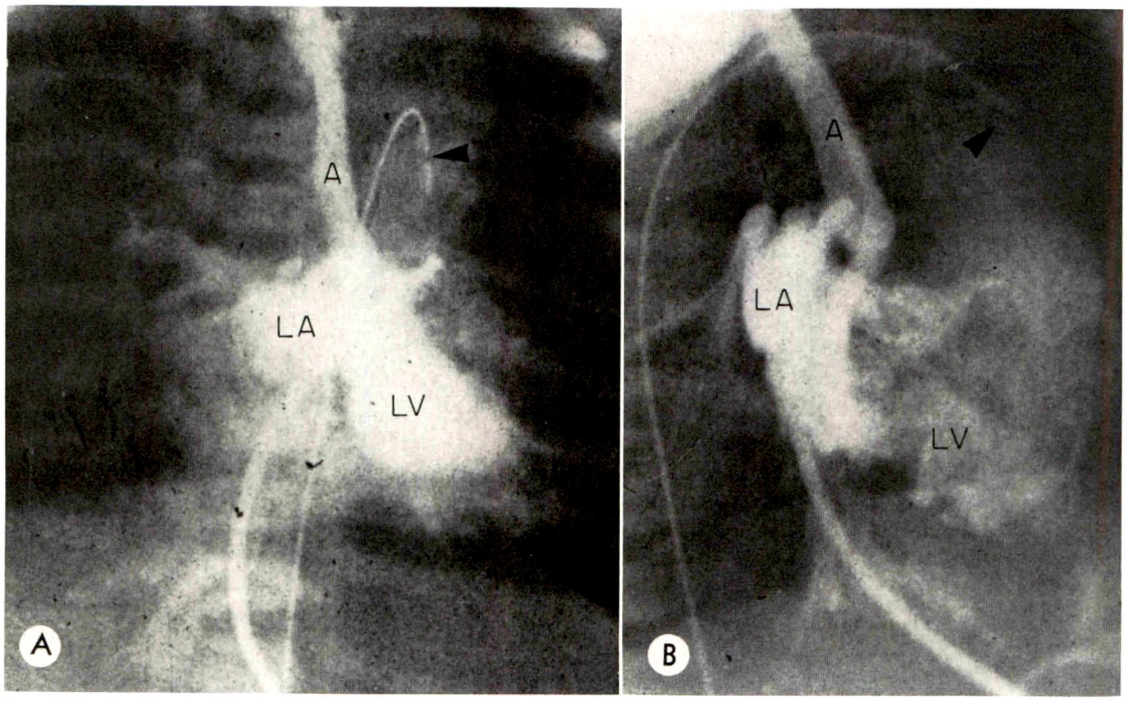


FIG. 13. (*A* and *B*) *Hypoplastic left heart syndrome.* Injection into the left atrium reveals it to be small. The left ventricle is also small. The ascending aorta is hypoplastic. The thin umbilical catheter is in the thoracic aorta crossing the ductus arteriosus into the pulmonary artery. The arrow points to the tip of the catheter which is in the main pulmonary artery. A = Aorta; LA = Left Atrium; LV = Left Ventricle.

GROUP 2

Lesions with normal or increased pulmonary blood flow: In patients with TGA a balloon atrial septostomy (Rashkind procedure)[21] is performed at the time of the diagnostic study. Atrial septectomy (Blalock-Hanlon procedure)[4] may be done, if balloon septostomy is unsuccessful. In most cases of CS, resection of the coarctation, usually with concomitant banding of the pulmonary artery, is urgently needed to relieve severe left ventricular failure and pulmonary venous congestion.

GROUP 3

Lesions with severe pulmonary venous congestion: Emergency repair with deep hypothermia or cardiopulmonary bypass is indicated in patients with TAPVR with obstruction. In infants with congestive heart failure due to an isolated coarctation, an initial trial of intensive medical therapy is indicated. Some of these children will respond adequately to this approach and allow postponement of corrective surgery until a more optimal age (3 to 4 years). In the HLH syndrome there is currently some interest in surgically creating an aorticopulmonary communication to allow unobstructed flow from the pulmonary artery to the aorta, and concomitant balloon septostomy or surgical atrial septectomy to decompress the left atrium.[5]

SUMMARY

A coordinated physiologic-radiologic approach is presented to the emergency diagnosis of congenital heart lesions in the first 2 weeks of life.

The pathophysiology and roentgenologic findings of the 9 lesions primarily responsible for respiratory distress and cyanosis on a cardiac basis in this age group are discussed in detail.

A logical, schematic approach to cardiac

catheterization and angiocardiography is proposed.

The principles of emergency therapeutic management are briefly outlined.

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REFERENCES

1. Amplatz, K., Lester, R. G., Schiebler, G. L., Adams, P., Jr., and Anderson, R. C. Features of Ebstein's anomaly of tricuspid valve. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1959, *81*, 788–794.
2. Barcia, A., Kincaid, O. W., Davis, G. D., Kirklin, J. W., and Ongley, P. A. Transposition of great arteries: angiocardiographic study. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *100*, 249–283.
3. Bessolo, R. J., and Vincent, W. R. Diagnosis of congenital heart disease in first two weeks of life. *California Med.*, 1969, *110*, 200–206.
4. Blalock, A., and Hanlon, C. R. Interatrial septal defect—its experimental production under direct vision without interruption of circulation. *Surg., Gynec. & Obst.*, 1948, *87*, 183–187.
5. Cayler, G. G., and Smeloff, E. A. Surgical palliation of hypoplastic left side of heart. *New England J. Med.*, 1970, *282*, 780–783.
6. Desilets, D. T., Marcano, B. A., Emmanouilides, G. C., and Gyepes, M. T. Severe pulmonary valve stenosis and atresia. *Radiol. Clin. North America*, 1968, *6*, 367–382.
7. Elliott, L. P., Van Mierop, L. H., and Gleason, D. C. Roentgenology of tricuspid atresia. *Seminars in Roentgenol.*, 1968, *3*, 399–409.
8. Ellis, K., Griffiths, S. P., Burris, J. O., Ramsay, G. C., and Fleming, R. J. Ebstein's anomaly of tricuspid valve: angiocardiographic considerations. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1964, *92*, 1338–1352.
9. Garman, J. E., Hinson, R. E., and Eyler, W. R. Coarctation of aorta in infancy: detection on chest radiographs. *Radiology*, 1965, *85*, 418–422.
10. Gerald, B. Combined aortic and mitral valve obstruction in early infancy. *Radiology*, 1967, *88*, 1100–1104.
11. Gleason, D. C., Ferguson, T., and Hartmann, A. F., Jr. Angiocardiographic differentiation between Fallot's pulmonary atresia and Type IV persistent truncus arteriosus. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *100*, 312–317.
12. Harris, G. B., Neuhauser, E. B., and Giedion, A. Total anomalous pulmonary venous return below diaphragm: roentgen appearances in three patients diagnosed during life. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1960, *84*, 436–441.
13. Kirklin, J. Tetralogy of Fallot. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *102*, 253–266.
14. Kurlander, G. J., Petry, E. L., and Girod, D. Plain film diagnosis of congenital heart disease in newborn period. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *103*, 66–77.
15. Lambert, E. C., Canent, R. V., and Hohn, A. R. Congenital cardiac anomalies in newborn: review of conditions causing death or severe distress in first month of life. *Pediatrics*, 1966, *37*, 343–351.
16. Lambert, E. C., Tingelstad, J. B., and Hohn, A. R. Diagnosis and management of congenital heart disease in first week of life. *Pediat. Clin. North America*, 1966, *13*, 943–982.
17. Levin, A. R., Grossman, H., Schubert, E. T., Winchester, P., and Gilladoga, A. Effect of angiocardiography on fluid and electrolyte balance. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *105*, 777–783.
18. Lieber, A., Rosenbaum, H. D., Hanson, D. J., and Kwann, H. M. Accuracy of predicting pulmonary blood flow, pulmonary arteriolar resistance and pulmonary venous pressure from chest roentgenograms. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *103*, 577–582.
19. Linde, L. M., Higashino, S. M., and Berman, G. Umbilical vessel cardiac catheterization and angiocardiography. *Circulation*, 1966, *34*, 984–988.
20. Lucas, R. V., Jr., Adams, P., Jr., and Anderson, R. C. Total anomalous pulmonary venous connection to portal venous system: cause of pulmonary venous obstruction. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1961, *86*, 561–575.
21. Rashkind, W. J., and Miller, W. W. Creation of atrial septal defect without thoracotomy: palliative approach to complete transposition of great arteries. *J.A.M.A.*, 1966, *196*, 991–992.
22. Rowe, R. D., and Mehrizi, A. The Neonate with Congenital Heart Disease. W. B. Saunders Company, Philadelphia, 1968.
23. Rudolph, A. Complications occurring in infants and children. *Circulation*, 1968, *37*, Suppl. 3, 111–59; 111–66.
24. Wittenborg, M. H., Neuhauser, E. B. D., and Sprunt, W. H. Roentgenographic findings in congenital tricuspid atresia with hypoplasia of right ventricle. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1951, *66*, 712–727.

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PULMONARY ATRESIA WITH INTACT VENTRICULAR SEPTUM*

NEW DEVELOPMENTS IN DIAGNOSIS AND TREATMENT

By KENT ELLIS, M.D., WILLIAM J. CASARELLA, M.D., CONSTANCE J. HAYES, M.D., WELTON M. GERSONY, M.D., FREDERICK O. BOWMAN, Jr., M.D., *and* JAMES R. MALM, M.D.
NEW YORK, NEW YORK

PULMONARY atresia with intact ventricular septum is an uncommon congenital cardiac malformation which has nearly always been fatal in early infancy.[3,4,9,10,13] Pulmonary valvulotomy alone has been successful in only a small minority of cases. Recently, however, much improved infant survival has been reported following prompt diagnosis and emergency surgical augmentation of pulmonary blood flow by aorta to pulmonary artery shunts (Waterston or Potts types).[5,9,19] Among these reports, our group[1] obtained especially promising results using a combination of pulmonary valvulotomy and an aorta to pulmonary artery shunt as the initial surgical treatment. Also reported was the subsequent successful surgical reconstruction of the right ventricle and pulmonary outflow tract using unicusp aortic homografts in 2 babies who initially had small right ventricles.

The importance of a prompt, precise angiocardiographic diagnosis is greatly increased by these remarkable improvements in the management of this condition.

The purpose of this paper is to briefly present the clinical and roentgenographic features of pulmonary atresia with intact ventricular septum (PA-IVS), emphasizing the findings and value of selective angiocardiographic studies. In addition, our experience involving 27 babies with this anomaly seen since 1960 will be briefly reviewed with respect to survival after surgical treatment.

PATHOLOGY

The occluding diaphragm (arrows in Fig. 1 *A*) in PA-IVS is formed by fused pulmonary valve tissue. Both the right ventricular infundibulum and the main pulmonary trunk are patent, and the latter often of near normal size but smaller than the enlarged aorta. The right ventricular walls are thickened by marked hypertrophy.

In about 80 per cent of cases the cavity of the right ventricle is small (sometimes miniscule). In the other 20 per cent the right ventricular lumen is normal or even enlarged. Actually a continuous spectrum in right ventricular sizes exists. Tricuspid valve development is related to the ventricular size; therefore, the valve is often small or hypoplastic. Frequently, the tricuspid leaflets are partially adherent to the ventricular wall or displaced into the ventricular cavity in a fashion resembling a mild Ebstein's anomaly.[1,9] In the larger right ventricles, the valve leaflets are more often deformed and incompetent.[8] In many cases abnormal sinusoids directly connect the right ventricle to the coronary arteries[8,17] (Fig. 13, *A* and *B*). The right atrium, the left atrium, the left ventricle and aorta are larger than normal. The aortic arch is virtually always on the left. Additional cardiovascular or other system anomalies are not common.

PATHOLOGIC PHYSIOLOGY

During fetal life occlusion of the right

* Presented at the Seventy-second Annual Meeting of the American Roentgen Ray Society, Boston, Massachusetts, September 28–October 1, 1971.

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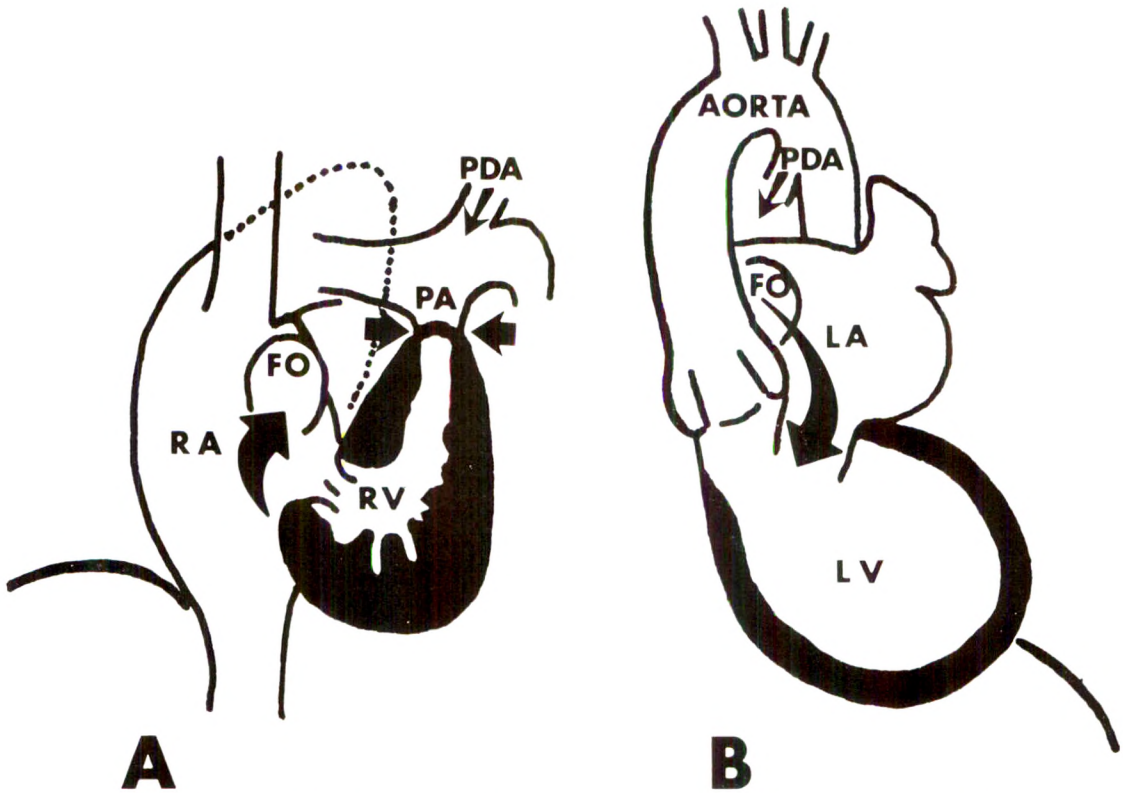


Fig. 1. Diagram of (*A*) the right and (*B*) the left heart in pulmonary atresia with intact ventricular septum (PA-IVS). Note the fused valve leaflets (arrows) occluding the right ventricular (RV) outflow tract, and forcing forward blood flow (curved arrow) to pass via a patent foramen ovale (FO) into the left heart. The right atrium (RA), left atrium (LA), left ventricle (LV) and aorta are enlarged. Blood flow to the pulmonary artery (PA) is via a patent ductus arteriosus (PDA) both before and after birth.

ventricular outflow tract by an atretic valve forces the entire systemic venous return (from fetus and placenta) to pass from the right atrium through the foramen ovale into the left atrium. Because virtually all forward flow traverses this route, the right atrium, left heart and aorta are enlarged. Flow in the ductus arteriosus in PA-IVS during fetal life is reversed (L to R) and consists only of the small fetal blood supply to lungs.

Despite these radical changes, fetal distress before birth is not evident, and deliveries are typically uncomplicated and at term.

After birth survival becomes dependent upon blood flow to the lungs via a patent ductus arteriosus (PDA). Inadequate flow through the spontaneously closing PDA characteristically causes cyanosis in the first few hours. Death from hypoxia typically follows in the first weeks after birth. (Rarely the PDA remains widely open as occurred in 1 of our cases.)

CLINICAL FEATURES

Cyanosis is usually prominent within a few hours after birth. By the second or third day of life many of these infants are in extremis with severe hypoxia. Signs of right heart failure may also be evident.

Sometimes no murmur is audible, but frequently a soft systolic murmur is noted and attributed to tricuspid regurgitation or blood flow through a patent ductus arteriosus. The second sound is single. An occasional baby surviving several weeks will develop a continuous murmur due to flow via the patent ductus arteriosus.

The normal or rightward direction of the

electrical axis on the electrocardiograms of these infants is extremely valuable in ruling out tricuspid atresia (in which the axis is almost invariably directed leftward and superiorly).

The size of the right ventricle can also be roughly estimated from the electrocardiogram. Infants with PA-IVS and small or tiny right ventricular lumens display electrocardiographic evidence of relative left ventricular predominance on the precordial leads. If, however, the right ventricle is normal or enlarged, evidence of right ventricular dominance (or hypertrophy) is seen. In addition, the electrical axis is further deviated toward the right in cases with large right ventricles.

ROENTGEN FINDINGS

CHEST ROENTGENOGRAMS

The chest roentgenograms show clear lung fields with diminished (or normal) vascular markings—features which in the presence of severe clinical hypoxia strongly suggest diminished pulmonary perfusion. The heart size is variable (Fig. 2, *A* and *B*), but cardiomegaly is usually present and often striking. In frontal projection the right atrial and left ventricular borders are prominent and the main pulmonary artery segment is usually flat and concave.[12] Marked cardiomegaly soon after birth or developing during the first weeks favors a large right ventricle.

The differential diagnosis involves mainly anomalies causing decreased pulmonary blood flow in early infancy such as: tricuspid atresia, Ebstein's anomaly and other tricuspid valve malformations; severe pulmonary stenosis with intact ventricular septum; and pulmonary stenosis or atresia with associated anomalies such as ventricular septal defect (*e.g.*, severe tetralogy of Fallot) or transposition of the great vessels.

ANGIOCARDIOGRAPHIC DIAGNOSIS

Emergency angiocardiography is crucial to definitive diagnosis before irreversible clinical deterioration occurs. Rarely, however, when a baby presents in extremis and the clinical need for an aorta to pulmonary shunt is obvious, it may be necessary to perform a shunting operation (with or without pulmonary valvulotomy) prior to special diagnostic studies.

Venous or right atrial angiocardiograms have been widely used and show the large obligatory right to left interatrial shunt and the left heart reasonably well. These examinations, however, do not reliably show either the right ventricle or the precise source of pulmonary blood supply (Fig. 3, *A* and *B*).

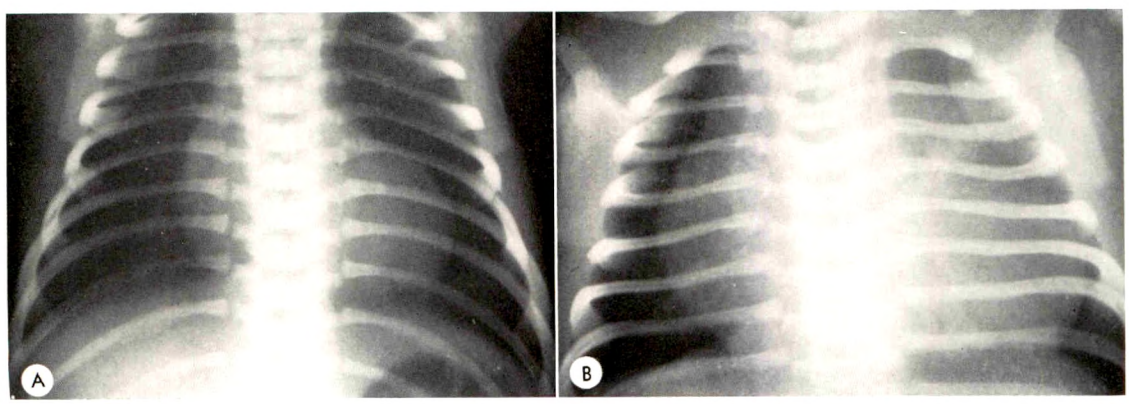


Fig. 2. (*A* and *B*) Chest roentgenograms in 2 babies with PA-IVS made on day of birth show clear lungs with decreased prominence of the pulmonary vasculature. The heart size is variable as suggested by these examples. Often, but not always, the largest hearts on plain roentgenograms are associated with large right ventricles.

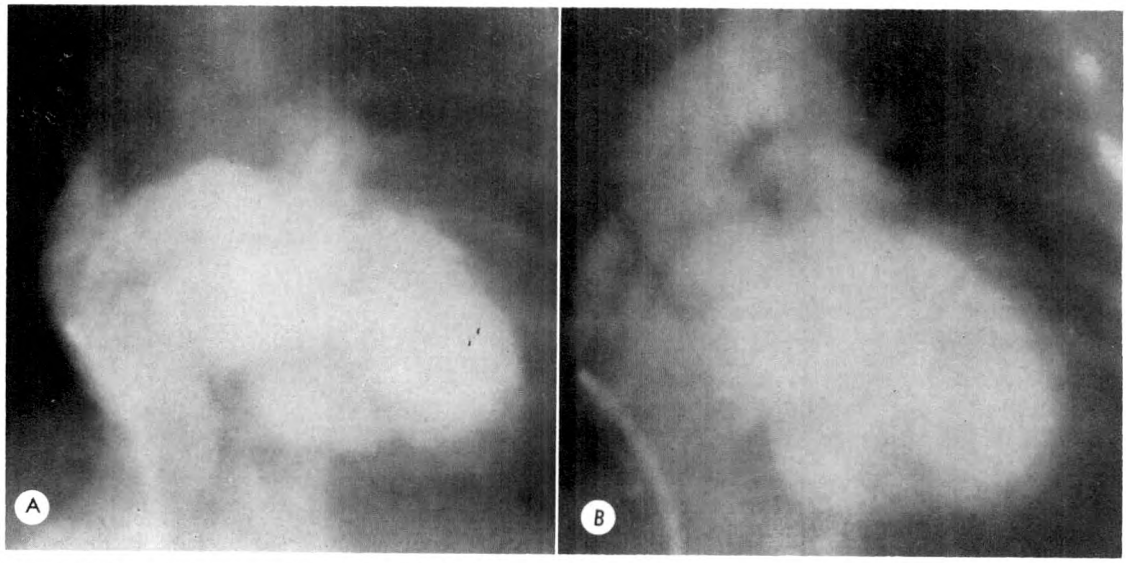


FIG. 3. Right atrial angiocardiograms in PA-IVS. (*A*) From the right atrium, the left atrium and left ventricle have opacified via the large obligatory right to left interatrial shunt. A right ventricle is not demonstrated. (*B*) One second later the large aorta and some pulmonary arteries have become opacified. The persistent atrial opacification obscures the route of pulmonary artery filling. Tricuspid atresia cannot be excluded.

Selective right ventricular angiocardiography best demonstrates the size of the right ventricular lumen, the total occlusion at the level of the pulmonary valve and the intactness of the ventricular septum, but poorly demonstrates the left heart and pulmonary vasculature (Fig. 4 through 8). Tricuspid atresia is ruled out. Right ventricular

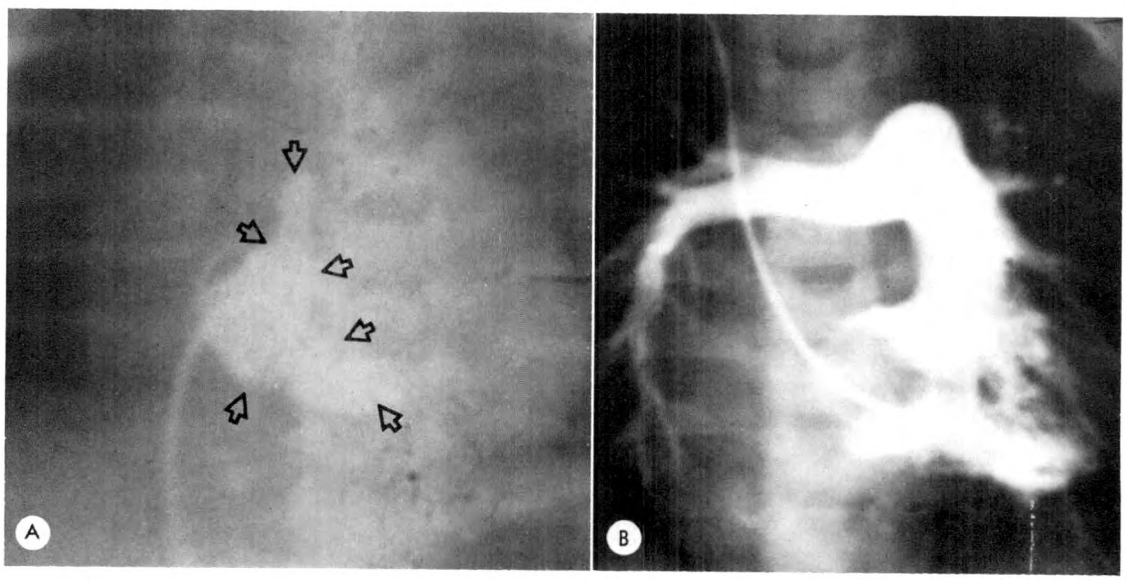


FIG. 4. Right ventricular angiocardiograms in PA-IVS and small right ventricle. (*A*) Cine frame from selective right ventricular angiocardiography at 3 days of age shows a small right ventricle with occluded outflow (top arrow). A Brock valvulotomy was performed. (*B*) Four years later a selective right ventricular angiocardiogram shows a larger right ventricle with a patent pulmonary outflow tract. The tricuspid valve is dome shaped. Repeat operation with pulmonary and tricuspid valvuloplasties has given a good result at 10 years.

pressures can be measured and the degree of tricuspid regurgitation estimated. When the right ventricle is strongly suspected of being small, the dose of contrast material is reduced to about 0.5 cc./kg. Occasionally the right ventricle cannot be entered, in which case a right atrial selective angiocardiogram may be helpful.

Selective left ventricular angiocardiograms, although not directly visualizing the right ventricle, *can in conjunction with the catheter findings exclude virtually all other diagnoses* (except the very rare combination of tricuspid and pulmonary atresia with intact ventricular septum). The left ventricle often is readily entered via catheter passage across the foramen ovale. The findings are sequential opacification of a large *left ventricle with intact ventricular septum*, a large aorta, and then the *pulmonary arteries via a patent ductus arteriosus. The main pulmonary artery is opacified retrograde to the atretic valve* without dilution by flow from the right ventricle (Fig. 9 through 11). In addition, visualization of the right coronary artery in the right atrioventricular groove, when compared to the opacified left ventricle, permits an estimate of the size of the right ventricle (Fig. 11, *A–C*; and 12, *A* and *B*). Late roentgenograms of the study will often show the pulmonary venous return.

A combination of selective right and left ventricular angiocardiograms is optimal for precise diagnosis. If for clinical or technical reasons, however, only one injection is feasible, the first ventricle entered should be studied, since either examination may be sufficient for preoperative diagnosis.

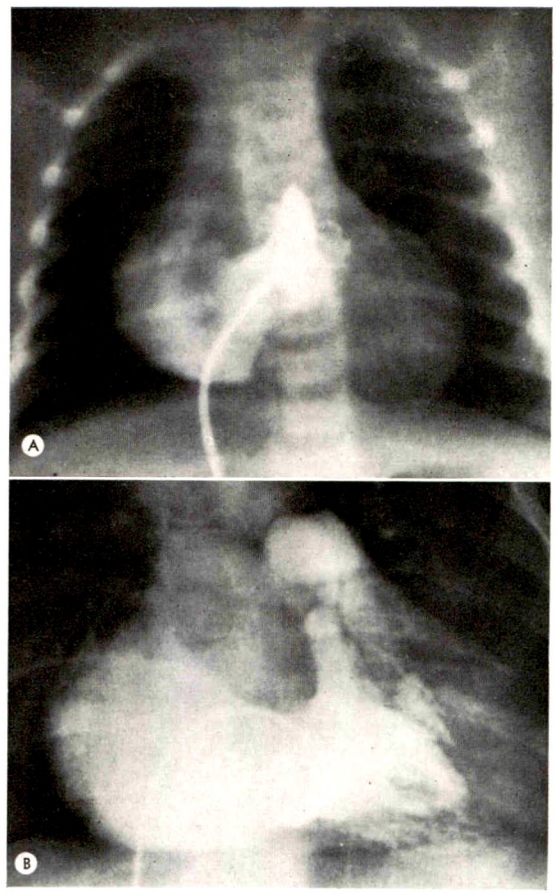


Fig. 5. Right ventricular angiocardiograms in PA-IVS and small right ventricle. (*A*) Selective anteroposterior right ventricular angiocardiogram at 3 days shows a small right ventricle, pulmonary atresia and tricuspid regurgitation. (*B*) Two years later after valvulotomy and shunt operation, repeat study shows some flow across the pulmonary outflow tract and a larger right ventricle. The right ventricular outflow tract was successfully reconstructed using a unicusp aortic homograft.[1]

SURGICAL TREATMENT

A. PULMONARY VALVULOTOMY ALONE

In 1956, Greenwold *et al.*[11] divided infants with PA-IVS into 2 anatomic types depending upon the size of the right ventricle: Type I—small or miniscule right ventricle; and Type II—normal or large right ventricle. They suggested that babies with larger right ventricles (Type II) would be favorable candidates for pulmonary valvulotomy. Subsequently, babies with angiocardiographic and electrocardiographic evidence of a large right ventricular cavity were particularly sought out for valvulotomy. The right ventricular cavity, however, was seldom well visualized by venous or right atrial angiocardiography and since, particularly in the newborn, electrocardiography could not rule out an adequately sized right ventricle, valvulotomy evolved as the prime surgical treatment for PA-IVS, regardless of right ven-

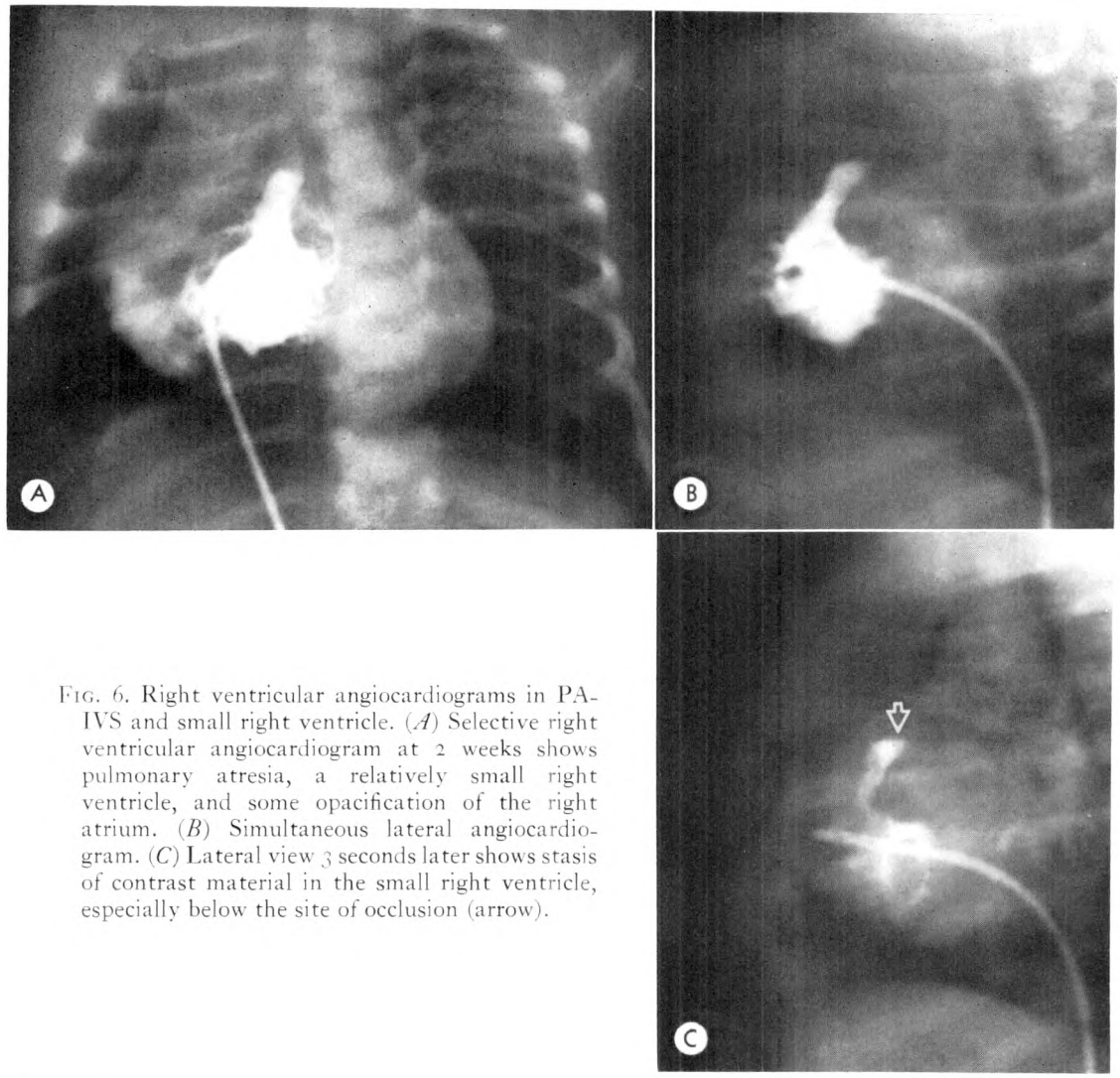
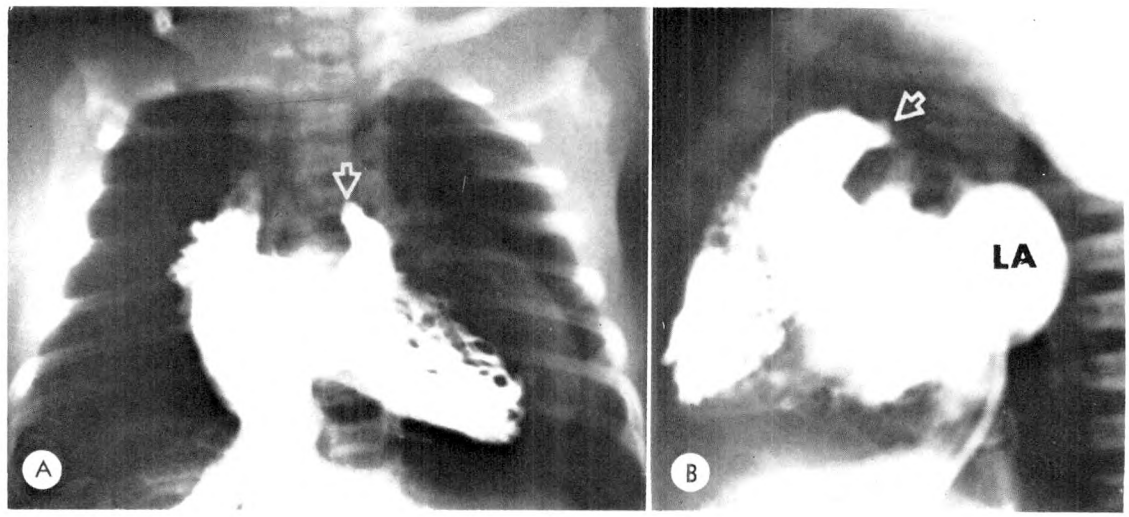


FIG. 6. Right ventricular angiocardiograms in PA-IVS and small right ventricle. (*A*) Selective right ventricular angiocardiogram at 2 weeks shows pulmonary atresia, a relatively small right ventricle, and some opacification of the right atrium. (*B*) Simultaneous lateral angiocardiogram. (*C*) Lateral view 3 seconds later shows stasis of contrast material in the small right ventricle, especially below the site of occlusion (arrow).



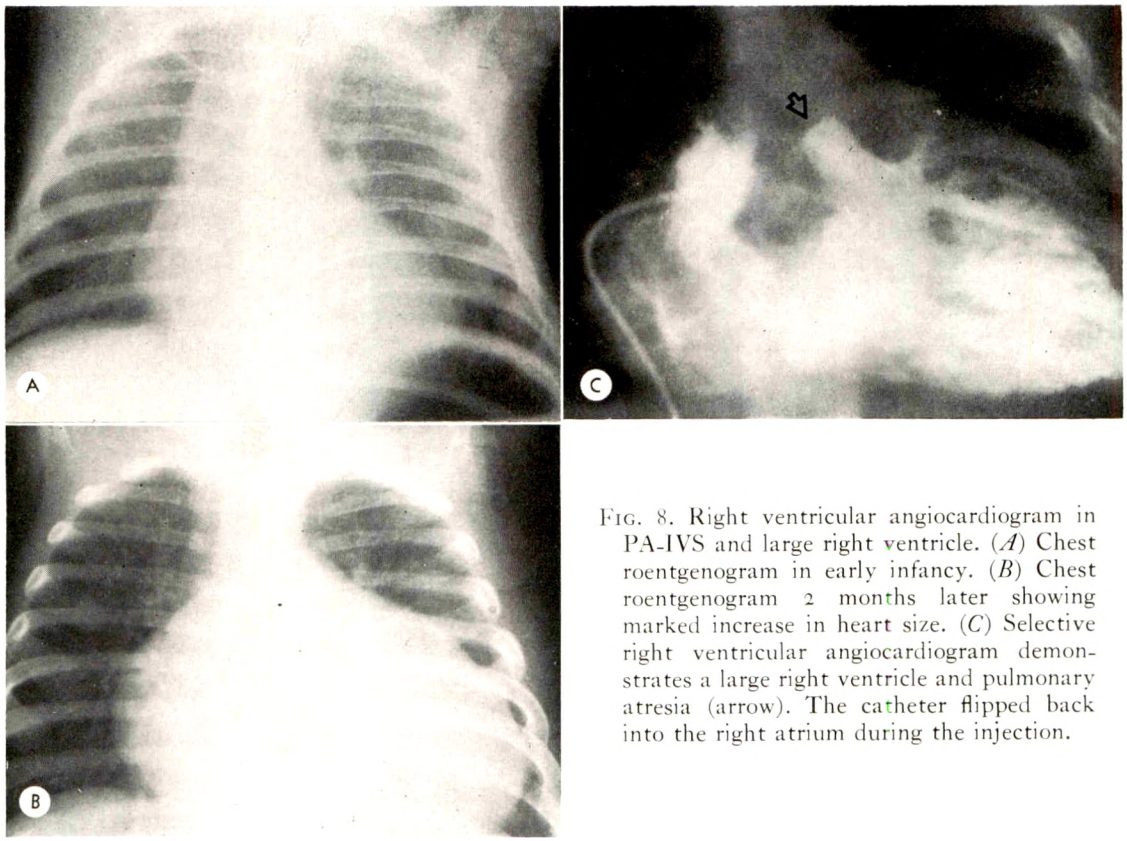


FIG. 8. Right ventricular angiocardiogram in PA-IVS and large right ventricle. (*A*) Chest roentgenogram in early infancy. (*B*) Chest roentgenogram 2 months later showing marked increase in heart size. (*C*) Selective right ventricular angiocardiogram demonstrates a large right ventricle and pulmonary atresia (arrow). The catheter flipped back into the right atrium during the injection.

tricular cavity size. The results have been generally poor.[1,3,5,13,15,19] Two recent reports,[2,16] however, have shown improved survival rates with valvulotomy, although still below the 50 per cent level—perhaps the improvement is due to earlier diagnosis, better pre- and postoperative care and increased surgical experience. Open operative correction using circulatory arrest and hypothermia has not proved promising in young infants with PA-IVS.[6]

B. AORTA TO PULMONARY SHUNTS

Since the obvious primary problem in sick babies with PA-IVS is *hypoxia* due to inadequate pulmonary blood flow, and since valvulotomy usually failed to reverse this hypoxic course, it is not surprising that attention has been turned to palliative shunting operations (aorta to pulmonary artery shunts) as a means of immediately improving pulmonary blood flow. Several recent reports have shown improved survival rates after such operations.[1,5,7,19] For example, although still advocating valvulotomy for infants with large right ventricles, Shams *et al.*[19] have reported survival of 7 of 8 babies with small right ventricles after a Potts anastomosis (left pulmonary artery to descending aorta). The ductus

FIG. 7. Right ventricular angiocardiograms in PA-IVS and normal sized right ventricle. (*A*) Selective anteroposterior right ventricular angiocardiogram showing pulmonary atresia (arrow), normal sized right ventricle and tricuspid regurgitation opacifying the right atrium. (*B*) Lateral right ventricular angiocardiogram reveals atretic pulmonary outflow tract (arrow) and opacification of the left atrium (LA) via the right to left interatrial shunt.

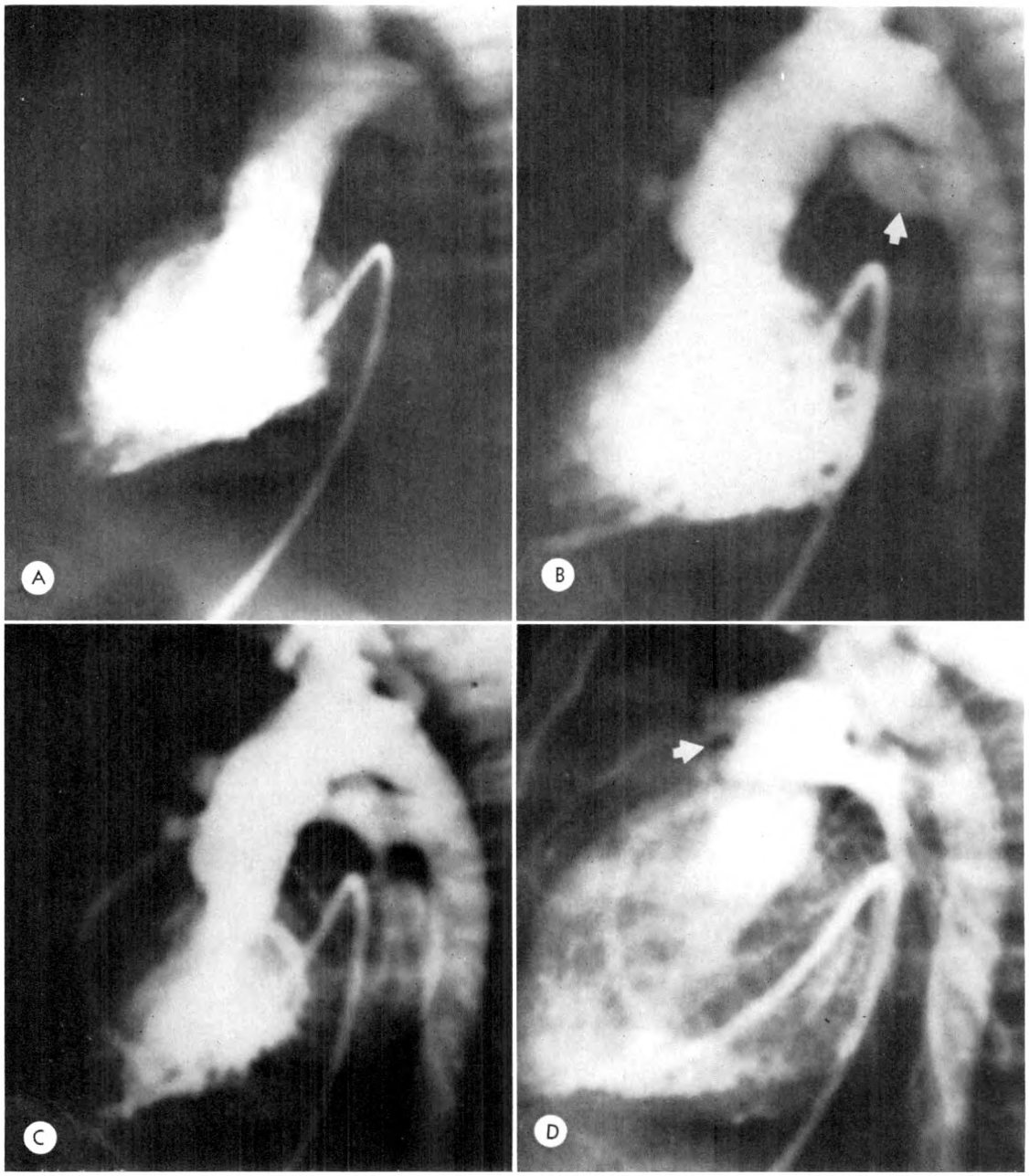


FIG. 9. Left ventricular angiocardiograms in PA-IVS at age 2 days. (*A*) Lateral projection during selective left ventricular injection shows proximal aortic opacification. (*B*) One-third second later the patent ductus arteriosus is opacified (arrow). (*C*) One-sixth second later in ventricular systole some pulmonary artery opacification is seen. (*D*) One-third second later in ventricular diastole dense opacification of the main pulmonary artery is seen to level of atretic pulmonary valve (arrow).

arteriosus is also ligated, so that there is no ambiguity about the source of pulmonary blood flow. Since the right chest is not entered, a later Glenn operation (superior vena cava to right pulmonary artery anastomosis) is more easily performed.

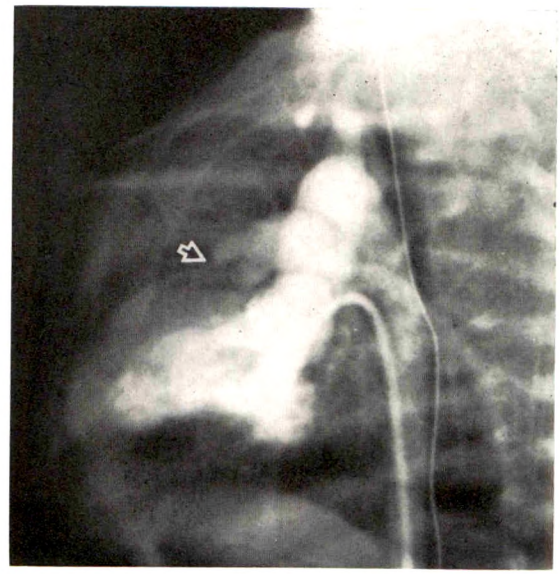


Fig. 10. Left ventricular angiocardiogram in PA-IVS at age 3 days. Lateral view shows opacification of the left ventricle and aorta and retrograde filling of the main pulmonary artery to level of atretic valve (arrow) via a patent ductus arteriosus.

Others have utilized the Waterston shunt (ascending aorta to right pulmonary artery) with success.[1,5,7]

Some groups[5,7,19] also advocate atrial balloon septostomy at the time of initial catheterization to obviate possible venous obstruction at the foramen ovale.[9,14] This procedure would not seem essential in most babies, since large flows must have been

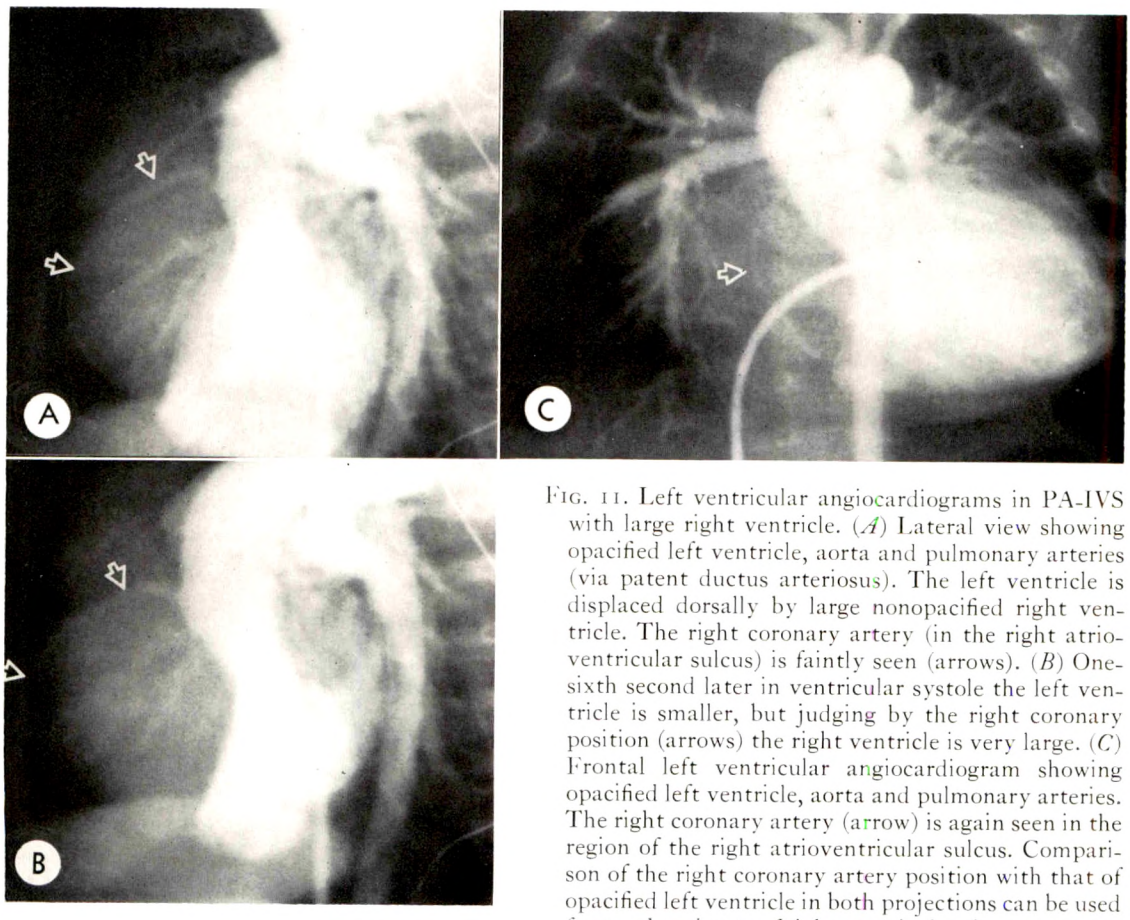


Fig. 11. Left ventricular angiocardiograms in PA-IVS with large right ventricle. (*A*) Lateral view showing opacified left ventricle, aorta and pulmonary arteries (via patent ductus arteriosus). The left ventricle is displaced dorsally by large nonopacified right ventricle. The right coronary artery (in the right atrioventricular sulcus) is faintly seen (arrows). (*B*) One-sixth second later in ventricular systole the left ventricle is smaller, but judging by the right coronary position (arrows) the right ventricle is very large. (*C*) Frontal left ventricular angiocardiogram showing opacified left ventricle, aorta and pulmonary arteries. The right coronary artery (arrow) is again seen in the region of the right atrioventricular sulcus. Comparison of the right coronary artery position with that of opacified left ventricle in both projections can be used for rough estimate of right ventricular size.

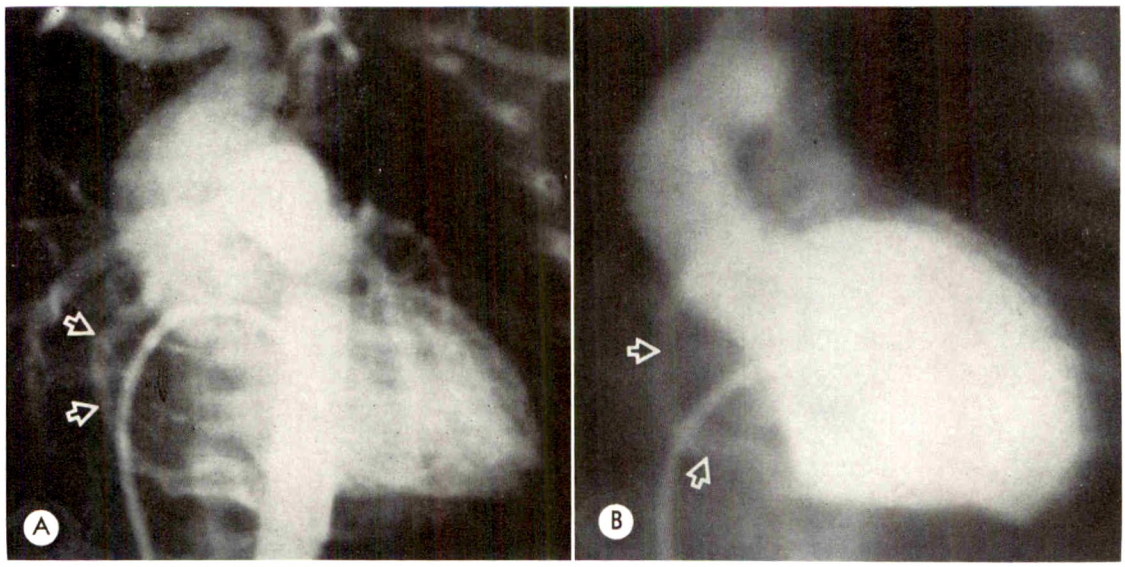


FIG. 12. Left ventricular angiocardiograms in PA-IVS. (*A*) Frontal projection showing opacification of left ventricle, aorta and pulmonary arteries. The right coronary artery (arrows) is well shown and suggests a substantial right ventricle is present (the right ventricle was about normal in size). (*B*) Frontal projection in another baby showing opacified left ventricle and proximal aorta. The right coronary artery (arrows) contains a small aneurysm (lower arrow) at the site of the sinusoidal connection to the small right ventricle.

successfully accommodated by this structure before birth, and spontaneous closure in the neonate with a large R to L shunt almost never occurs.

The value of the emergency shunting procedures is particularly evident upon review of the 27 babies with PA-IVS seen at the Columbia-Presbyterian Medical Center between 1960 and 1971 (15 babies seen between 1964 and 1969, 12 of whom had surgery, have previously been reported).[1] Twenty-five of the total group had angio-

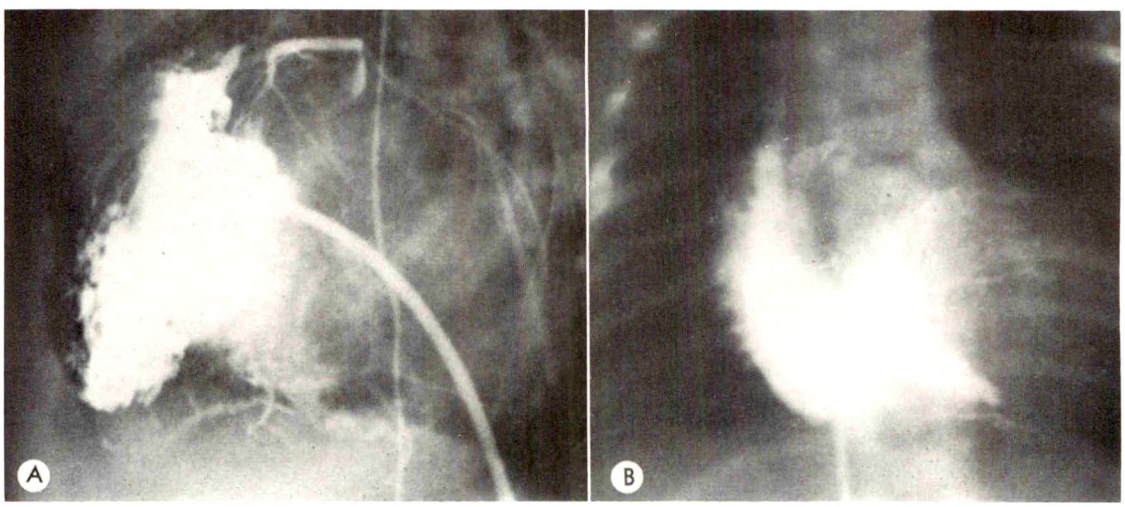


FIG. 13. (*A* and *B*) Right ventricular angiocardiograms in 2 cases of PA-IVS demonstrate opacification of some coronary arteries via persistent myocardial sinusoids. The systolic pressures measured in the right ventricles exceeded the aortic pressures in both cases. The right ventricle is particularly small in *B*.

cardiography and 22 had surgical treatment. The results of the initial operation are shown in Table 1.

Note that 12/13 babies initially treated with either a shunting operation alone, or this combined with a valvulotomy, were improved and (except for 1) were able to be sent home from the hospital. Unfortunately, 5 of these babies did not survive the first year, 3 dying following later valvulotomy operations, 1 of shunt thrombosis and 1 of an arrhythmia.

Table I

RESULTS OF INITIAL EMERGENCY SURGICAL TREATMENT IN 22 CASES OF PULMONARY ATRESIA WITH INTACT VENTRICULAR SEPTUM (1960–1970)

Operation	No. of Cases	Good Palliation		Living over One Year
		Small RV	Large RV	
Valvulotomy only	9	2/5	0/4	2/9
Shunt only	6	4/4	2/2	2/6
Combined	7	5/6	1/1	5/7

C. COMBINED VALVULOTOMY AND SHUNT OPERATIONS

The much improved results following the shunting procedures apply particularly to those babies with Type 1 (small) right ventricles. There are several reasons, however, why we advocate *combined valvulotomy and shunt operations*, even in babies with Type 1 (small) right ventricles.

First, in our experience, the combined operation has given promising long term results (Table 1).[1]

Secondly, the high pressures typically developed in these otherwise "blind" right ventricles probably influence mortality, since pulmonary atresia with ventricular septal defect, a lesion with a similar dependence upon a patent ductus arteriosus for pulmonary blood flow, has a much lower mortality in early infancy. The extremely high pressures typically developed in these ventricles are in part responsible for the myocardial necrosis described at necropsy in some cases.[14] Thus, decompression by valvulotomy seems reasonable, although the gain in lung perfusion may be small.

Thirdly, the eventual capacity for growth and function of the small right ventricle (as judged by emergency angiocardiography and electrocardiography in the newborn) is not easily predicted. It is known that many survivors (probably over 50 per cent) with PA-IVS show increasing evidence of right ventricular enlargement by electrocardiography after a few weeks. This may imply a relative increase in the size of the right ventricle.[5] At least 5 of our cases with initially small right ventricles eventually developed larger functional right ventricles (in 4 cases after valvulotomy). Others have reported similar findings after valvulotomy operations.[15,16]

Most large series of babies with PA-IVS have shown a strong preponderance of Type 1 (small right ventricle) cases.[3,5,10,15,19] Although the decision as to what constitutes a small (Type 1) right ventricle can be somewhat arbitrary, it is now clear that a number of these small right ventricles have more capacity for growth than expected. The factors necessary for realization of this potential are not understood. The valvulotomy operation may help by providing an opportunity for more volume change at less systolic pressure loading. The development of tricuspid regurgitation as well as growth of the heart in general may be important factors in stimulating progressive enlargement of the right ventricle.

On the other hand, from observations made on necropsy specimens, it seems probable that because of hypoplasia, endocardial and muscular fibrosis and tricuspid valve deformity some of the small right ventricles can never become functional chambers (Fig. 13*B*). Whether this type of ventricle represents 10 or 50 per cent of all cases of PA-IVS is not known. A pulmonary valvulotomy may not be feasible technically in some of these cases. It remains to be shown how often the

right ventricle can be salvaged and made a functional chamber by surgical techniques.

Thus, because of better survival rates and the proven possibility of eventual salvage of a functional right ventricle, we believe that the combination of a pulmonary valvulotomy, where feasible, and an aorta to pulmonary shunt[1] is the best treatment for babies with PA-IVS having small right ventricles (Type I).

In babies with PA-IVS and normal or large right ventricles (Type II), the mortality following valvulotomy alone is still very high. We believe that the combination of valvulotomy with an aorta to pulmonary shunt is likely to offer the best chance for long term survival to these patients also.

SUMMARY

1. Babies with pulmonary atresia and intact ventricular septum rarely survive for more than a few weeks after birth. Prompt diagnosis and recent advances in surgical treatment now offer greatly improved possibilities for long term survival.

2. Emergency angiocardiography is necessary for precise preoperative diagnosis.

   a. Venous or right atrial angiocardiograms are generally inadequate for accurate diagnosis.
   b. Selective right ventricular angiocardiography best shows the size and position of the right ventricle, the pulmonary valvular atresia and the intact ventricular septum, but poorly demonstrates the left heart and the lung vessels. Tricuspid atresia is ruled out.
   c. Selective left ventricular angiocardiography shows the left heart, aorta and pulmonary blood supply as well as the distal surface of the atretic pulmonary valve. The right coronary artery position permits an estimate of right ventricular size.
   d. A combination of both selective right and left ventricular angiocardiograms is ideal. Either examination alone, however, may be sufficient for preoperative diagnosis in a critically ill infant.

3. The small right ventricle of the neonatal period (by angiocardiographic and electrocardiographic criteria) often has a surprising potential for growth and may become a functioning cardiac chamber.

4. In our experience results are improved by combining a pulmonary valvulotomy with a shunting procedure as the initial treatment for nearly all cases of pulmonary atresia with intact ventricular septum.

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REFERENCES

1. Bowman, F. O., Jr., Malm, J. R., Hayes, C. J., Gersony, W. M., and Ellis, K. Pulmonary atresia with intact ventricular septum. *J. Thoracic & Cardiovasc. Surg.*, 1971, *61*, 85–94.
2. Celemajor, J. M., Bowdler, J. D., Gengos, D. C., Cohen, D. H., and Stuckey, D. S. Pulmonary valve function with intact ventricular septum. *Am. Heart J.*, 1968, *76*, 452–465.
3. Cole, R. B., Muster, A. J., Lev, M., and Paul, M. H. Pulmonary atresia with intact ventricular septum. *Am. J. Cardiol.*, 1968, *21*, 23–31.
4. Davignon, A. L., Greenwold, W. E., DuShane, J. W., and Edwards, J. E. Congenital pulmonary atresia with intact ventricular septum: clinicopathological correlation of two anatomic types. *Am. Heart J.*, 1961, *62*, 591–602.
5. Dhanavaravibul, S., Nora, J. J., and McNamara, D. G. Pulmonary atresia with intact ventricular septum: problems in diagnosis and results of treatment. *J. Pediat.*, 1970, *77*, 1010–1016.
6. Dillard, D. H., Mohri, H., and Merendino, K. A. Correction of heart disease in infancy utilizing deep hypothermia and total circulatory arrest. *J. Thoracic & Cardiovasc. Surg.*, 1971, *61*, 64–70.
7. Edmunds, L. H., Jr., Fishman, N. H., Heymann, M. A., and Rudolph, A. M. Anastomosis between aorta and right pulmonary artery (Waterston) in neonates. *New England J. Med.*, 1971, *284*, 463–471.
8. Edwards, J. E., Carey, L. S., Neufeld, H. N., and Lester, R. G. Congenital Heart Disease.

W. B. Saunders Company, Philadelphia, 1965, pp. 575–598.

9. Elliott, L. P., Adams, P., and Edwards, J. E. Pulmonary atresia with intact ventricular septum. *Brit. Heart J.*, 1963, *35*, 489–501.
10. Gersony, W. M., Bernhard, W. F., Nadas, A. S., and Gross, R. E. Diagnosis and surgical treatment of infants with critical pulmonary outlet obstruction. *Circulation*, 1967, *35*, 765–776.
11. Greenwold, W. E., Dushane, J. W., Burehell, H. B., Bruwer, A., and Edwards, J. E. Congenital pulmonary atresia with intact interventricular septum: two anatomic types. *Circulation*, 1956, *14*, 945–946.
12. Kieffer, S. A., and Carey, L. S. Radiological aspects of pulmonary atresia with intact ventricular septum. *Brit. Heart J.*, 1963, *25*, 655–662.
13. Kiely, B., Morales, F., and Rosenblum, D. Pulmonary atresia with intact ventricular septum. *Pediatrics*, 1963, *32*, 841–854.
14. Miller, W. W., Beligere, N., Waldhausen, J. A., Chatten, J., and Rashkind, W. J. Congenital pulmonary atresia with intact ventricular septum. *Am. J. Cardiol.*, 1969, *23*, 128–129.
15. Moller, J. H., Girod, D., Amplatz, K., and Varco, R. L. Pulmonary valvotomy in pulmonary atresia with hypoplastic right ventricle. *Surgery*, 1970, *68*, 630–634.
16. Murphy, D. A., Murphy, D. R., Gibbons, J. E., and Dobell, A. R. Surgical treatment of pulmonary atresia with intact interventricular septum. *J. Thoracic & Cardiovasc. Surg.*, 1971, *62*, 213–219.
17. Ogden, J. A. Secondary coronary arterial fistulas. *J. Pediat.*, 1971, *78*, 78–85.
18. Rook, G. D., and Gootman, N. Pulmonary atresia with intact interventricular septum: operative treatment with survival. *Am. Heart J.*, 1971, *81*, 476–481.
19. Shams, A., Fowler, R. S., Trusler, G. A., Keith, J. D., and Mustard, W. T. Pulmonary atresia with intact ventricular septum: report of 50 cases. *Pediatrics*, 1971, *47*, 370–377.

PNEUMOPERICARDIUM IN THE NEWBORN*

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PNEUMOPERICARDIUM in infants is rare and is usually of serious clinical significance. Cardiac tamponade and death may result if the clinician is not alerted to its significance.

A case is reported which shows the sequence of events in a newborn who developed a pneumopericardium which ultimately led to her death. In order to understand the roentgenographic findings, a detailed autopsy was carried out for the specific purpose of defining the pathogenesis of the pneumopericardium.

REPORT OF A CASE

B.L., a 2 pound, 4 ounce girl, was born to a 27 year old para 0-3-3-3 Negro female after 27 weeks gestation. The pregnancy had been unremarkable and labor was spontaneous. The infant was delivered in less than 4 hours after onset of labor.

At birth the Apgar was 1, and at 5 minutes was 5. Resuscitative measures were carried out, including endotracheal intubation and ventilation by hand. Thirty minutes after birth the infant was placed on a Bird respirator.

The respirator pressure was varied slightly according to the blood-gas values, but remained between 25 and 35 cm. of water. The respiratory rate was also varied between 52 and 60 per minute.

An initial roentgenogram of the chest during the first day of life revealed hyaline membrane disease. On the third day no change was noted in the pulmonary process.

The infant was maintained on a respirator, and early on the fourth day of life she became cyanotic. A chest roentgenogram at that time revealed a pneumopericardium (Fig. 1). Aortic pressure monitoring was instituted through an umbilical catheter: the initial pressure reading was 16–17/10–11 mm. Hg, indicating cardiac tamponade. A percutaneous pericardial tap

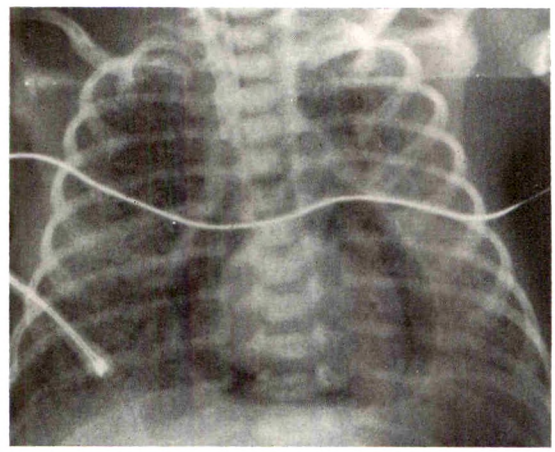


FIG. 1. Chest roentgenogram on the fourth day of life reveals hyaline membrane disease and a large pneumopericardium.

yielded 19 ml. of air with a dramatic increase in the arterial pressures to 45/25 mm. Hg, showing relief of the cardiac tamponade (Fig. 2). Even though a repeat chest roentgenogram revealed a persistent pneumopericardium, an adequate pulse pressure was maintained.

Later during this fourth day, the patient's clinical condition again deteriorated, with a recurrence of cyanosis and loss of palpable femoral pulses. A second pericardial tap yielded 10 ml. of air, following which the femoral pulses again became palpable. The patient's condition continued to deteriorate and death occurred about 3 hours later.

Postmortem Examination. A careful postmortem examination was performed with a special effort to determine the etiology of the pneumopericardium. Postmortem roentgenograms revealed the pneumopericardium and also a right pneumothorax (Fig. 3). The chest was opened under water and the pneumothorax was confirmed.

After removal of the anterior ribs and sternum, the pericardial sac was clearly seen to be distended with air and the heart to be

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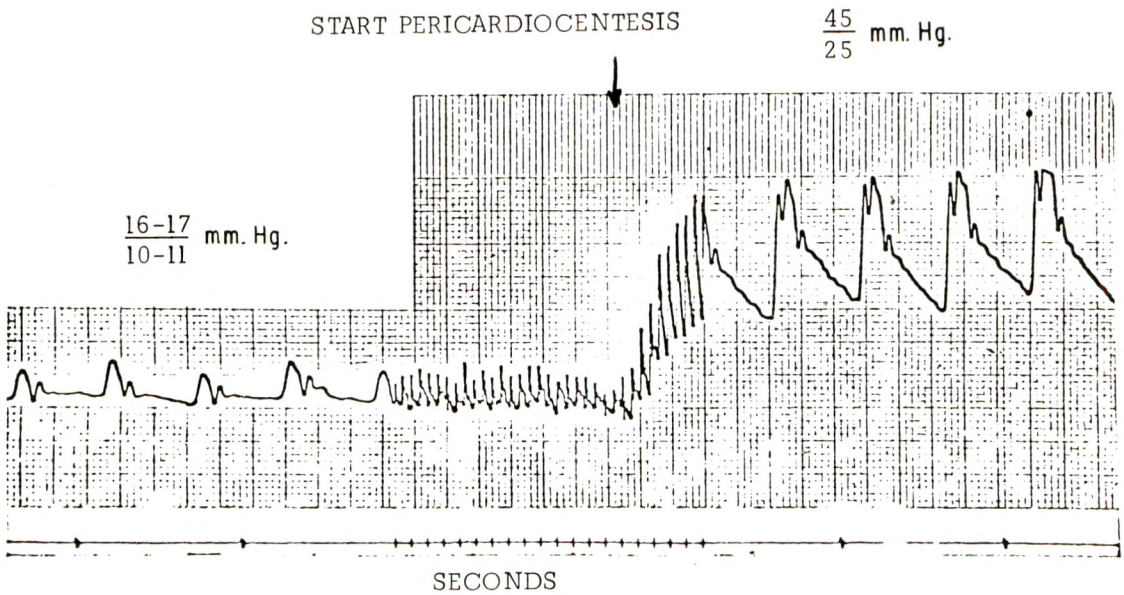


FIG. 2. Aortic pressure tracing. A dramatic increase in blood pressure is noted when pericardiocentesis is performed.

small. After 10 ml. of air was aspirated from the pericardial sac, 10 ml. of contrast material was injected (Fig. 4, *A* and *B*). Despite distention of the pericardial sac, no perforation or dissection of the contrast material out of the sac could be found. After both mainstem bronchi were tied off close to the lungs, contrast material was then injected into the trachea and no fistulous communications could be demonstrated between the trachea and pericardial cavity.

The organs were then removed from the chest and the lungs expanded under 10 cm. of water pressure and fixed with 10 per cent formalin. After fixation, a considerable amount of air was noted in the interstitium of the lungs, demarcating the secondary pulmonary lobules (Fig. 5). Microscopically, air was noted in the interstitium and the interstitial vascular sheaths of the lungs (Fig. 6, *A* and *B*).

DISCUSSION

Pneumopericardium in infants is rare. A review of the literature reveals only 7 previously reported cases.[4–7,9] Five of the 7 infants were newborns and 3 of these were premature. All 5 of the newborns were in respiratory distress, 3 with hyaline membrane disease. The 2 infants who were not newborn (ages $3\frac{1}{2}$ and 5 months) had endotracheal anesthesia at surgery for hydrocephalus. Both had cardiac arrest at the time of the surgery and when their chests were opened, pneumopericardium and pneumomediastinum were found. Six of the 7, including 4 of the premature infants, were

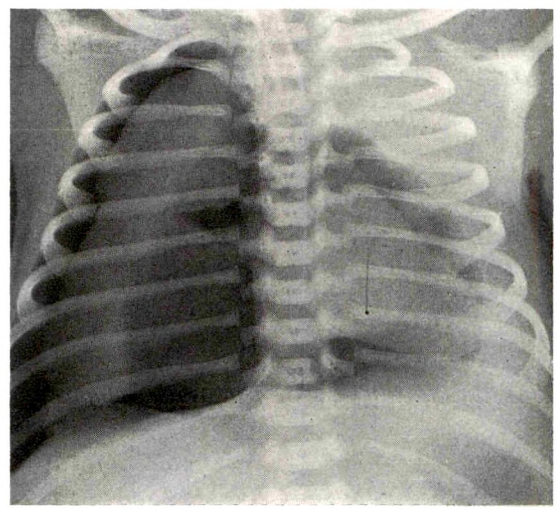


FIG. 3. A postmortem roentgenogram reveals the pneumopericardium and a right pneumothorax.

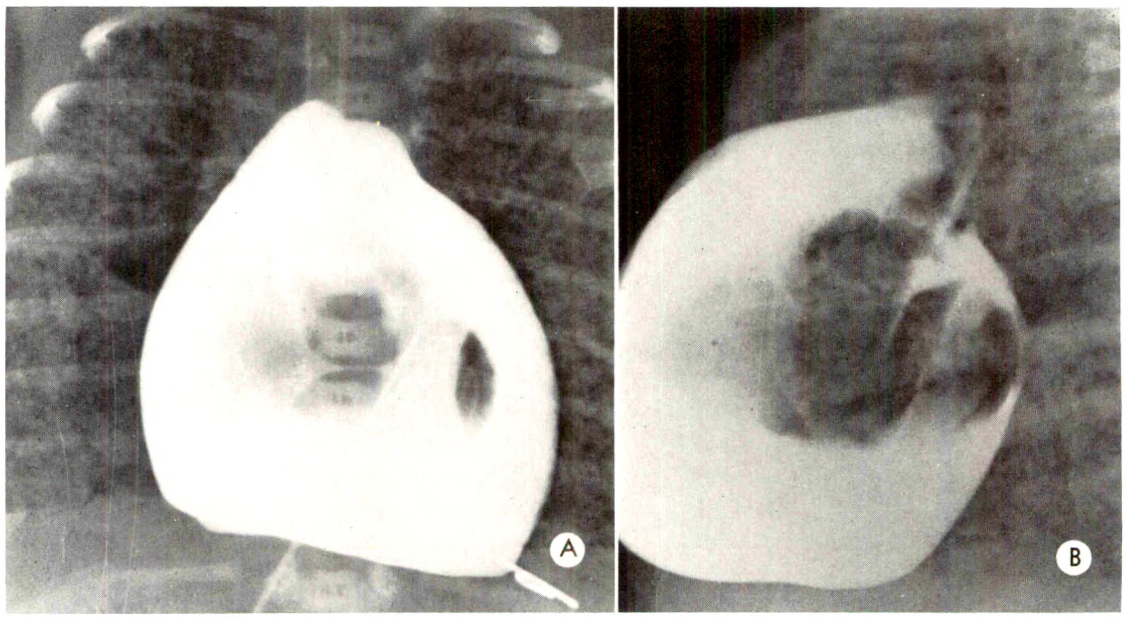


FIG. 4. (*A* and *B*) Postmortem roentgenograms with the pericardial sac distended with contrast material reveal no perforation or dissection.

being treated with positive pressure respirators.

Macklin[8] has shown experimentally that once alveolar rupture occurs, air can dissect through the interstitium and interstitial vascular sheaths of the lung to enter either the mediastinum or pleural cavity. The alveolar walls may be ruptured by positive pressure respirators, especially if the lungs are abnormal. Diseased lungs with both emphysema and atelectasis are prone to have alveolar rupture because there is a large pressure differential across the alveolar walls.[2] Hyaline membrane disease has areas of focal emphysema and atelectasis and alveolar rupture may occur with the use of positive pressure respirators.

In our case, as in the previously reported 7 cases, no pericardial tear was demonstrated. As a result of the use of the positive pressure respirator in this infant, the alveolar walls were apparently ruptured. At autopsy, air was demonstrated in the interstitium and the interstitial vascular sheaths of the lungs. This tends to support Macklin's experimental findings. The air apparently dissected from the interstitium of the lung through the pericardial reflection around the great vessels.

Cimmino[3] speculated that the serosal layers of the pericardium in young infants are unusually susceptible to dissection and rupture. In adults the pericardium is more resistant to air dissection, and pneumopericardium is usually not seen with the use of respirators.

By placing air under pressure in the pericardial cavity of a young female, Adcock and co-workers[1] demonstrated that cardiac tamponade occurred when the pericardial air pressure reached 145 mm. of water. Although pericardial pressures were not recorded in our case, the striking rise in arterial pressure when air was removed from the pericardium is evidence of relief of the cardiac tamponade (Fig. 2). Six of the 7 cases previously reported had a tension pneumopericardium and cardiac tamponade. Three of the 6 died from the tamponading effect of the air on the heart.

Clinically, cardiac tamponade is manifested by respiratory distress, cyanosis, decreased arterial saturation and diminished intensity of the heart sounds. When a ten-



Fig. 5. A photograph of the autopsy specimen of the left lung reveals interstitial air demarcating the pulmonary lobules, best visualized along the periphery.

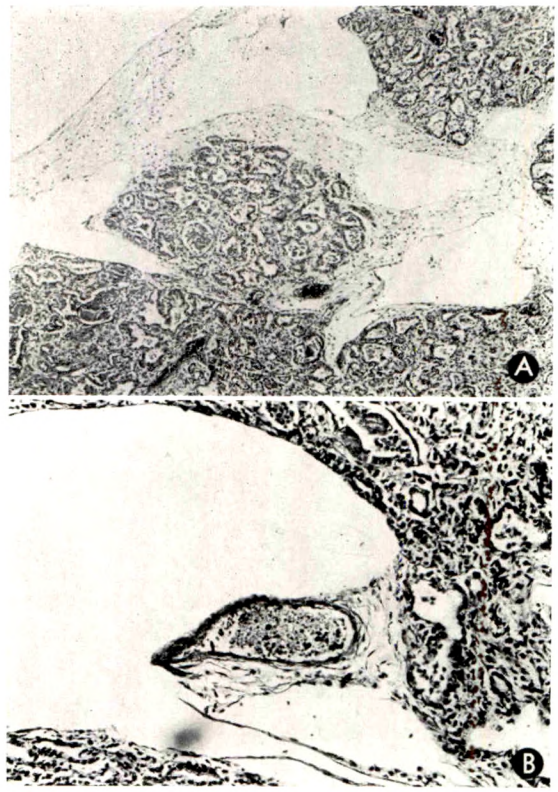


Fig. 6. (*A*) Photomicrograph reveals subpleural and interstitial air. (*B*) Photomicrograph reveals air in the perivascular space.

sion pneumopericardium is suspected, immediate needle aspiration of the pericardial sac is life-saving.[4,6] If the pericardial air reaccumulates and the child cannot be removed from the respirator, a catheter may be left in the pericardial sac to relieve the pressure.[9]

SUMMARY

A case report of a newborn premature infant with hyaline membrane disease, pneumopericardium and cardiac tamponade is presented.

Pneumopericardium with cardiac tamponade in a newborn is a potentially lethal condition, and prompt recognition is necessary so that immediate treatment can be instituted.

A roentgenogram of the chest should be obtained on any newborn, especially one with hyaline membrane disease, who, while on a respirator, shows increased respiratory distress. If pneumopericardium is present, cardiac tamponade should be suspected.

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REFERENCES

1. Adcock, J. D., Lyons, R. H., and Barnwell, J. B. Circulatory effects produced in patient with pneumopericardium by artificially varying intra-pericardial pressure. *Am. Heart J.*, 1940, *19*, 283–291.
2. Clernick, V., and Avery, M. E. Spontaneous alveolar rupture at birth. *Pediatrics*, 1963, *32*, 816–824.
3. Cimmino, C. V. Some radio-diagnostic notes on pneumomediastinum, pneumothorax and pneumopericardium. *Virginia M. Month.*, 1967, *94*, 205–212.

4. COHEN, S., and LOCKHART, C. Pneumopericardium: complication of prolonged ventilation. *Anesthesiology*, 1970, *32*, 465–467.
5. DURWAR, P. C. Pneumopericardium in neonate. *Australian Radiol.*, 1966, *10*, 229–230.
6. GROSFELD, J. L., KILMAN, J. W., and FREY, T. R. Spontaneous pneumopericardium in newborn infant. *J. Pediat.*, 1970, *76*, 614–616.
7. LOFTIS, J. W., SUSEN, A. F., MARCH, J. H., and SHERMAN, F. E. Pneumopericardium in infancy. *A.M.A. Am. J. Dis. Child.*, 1962, *103*, 61–65.
8. MACKLIN, C. C. Transport of air along sheaths of pulmonic blood vessels from alveoli to mediastinum. *Arch. Int. Med.*, 1939, *64*, 913–926.
9. MATTHIEW, J. M., NUSSLE, D., TORRADO, A., and SADEGHI, H. Pneumopericardium in newborn. *Pediatrics*, 1970, *46*, 117–119.

THE CERVICAL AORTIC ARCH*

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CONGENITAL malformations of the aortic arch are commonly encountered.[1,8] Occasional cases are worthy of reporting because of their special interest. We have recently studied a young child in whom the aortic arch was located high in the neck. To date, only 11 such cases have been reported.[2,3,5,7–15] None of these have appeared in the radiologic literature.

It is the purpose of this paper to review the clinical features, the roentgenologic findings and the embryologic considerations of this rare aortic arch malformation.

REPORT OF A CASE

M.J., GMH No. 223825. This 7 year old girl was admitted to Grady Memorial Hospital for evaluation of a $2\frac{1}{2}\times 3$ cm. pulsatile mass in the right neck which was present since birth. Otherwise, the child was asymptomatic.

A chest roentgenogram (Fig. 1*A*) showed the heart to be of normal size. The aortic knob shadow was absent. An oblique cut-off of the tracheal air shadow at the level of the thoracic inlet was visible. The lateral view of the chest (Fig. 1*B*) showed the trachea to be displaced forward at the level of the aortic arch.

Barium swallow studies of the esophagus (Fig. 2, *A* and *B*) revealed an obliquely directed defect running downward from right to left just below the level of the clavicles. On the lateral view, there was forward displacement of the esophagus at the same level. Aortography (Fig. 3, *A–C*) showed the arch of the aorta to lie in the right neck. The arch descended on the right to the level of T-4 at which point it crossed to the left of the spine. Below the level of T-6, the aorta descended in a normal fashion on the left side. The first branch of the ascending aorta was the left common carotid artery. The right external and internal carotid arteries arose separately from the apex of the arch. The right subclavian artery was the next vessel to arise from the arch. The left subclavian artery originated from a small conical diverticulum just distal to the retroesophageal segment of the aorta. Figure 4 illustrates the anatomy of the cervical aorta, its relation to the trachea and esophagus, and the branching of the arch vessels.

DISCUSSION

Definition. In this anomaly, the ascending aorta arises normally from the left ventricle and extends in such a fashion that the aortic arch is situated high in the neck on either side.

Incidence and Clinical Significance. Eleven cases have been reported in the literature, 2 left-sided,[5,11,12] and 9 cases with the aortic arch in the right side of the neck.[2,3,7–10,13–15]

In all cases, a pulsatile mass was present in the neck above the clavicle. The pulsating mass may be mistaken for an aneurysm of the subclavian, carotid or innominate artery.[2,10] Symptoms of a vascular ring may be present or may develop.[2,3,15] There have been no reported examples of cervical aortic arch malformation associated with intracardiac congenital heart disease.

Development. In the development of the cervical aortic arch, there are 2 anatomic features that require an explanation—namely: (1) the location of the aortic arch in the neck; and (2) the branching of the arch vessels.

We have retained Edwards' concept of a functioning double aortic arch as the basic pattern to explain the development of the cervical aortic arch.[6,18] In this concept, there is an embryonic aortic arch on each side, and interruption of either the right or left arch at one or more points can explain the embryologic basis for the malforma-

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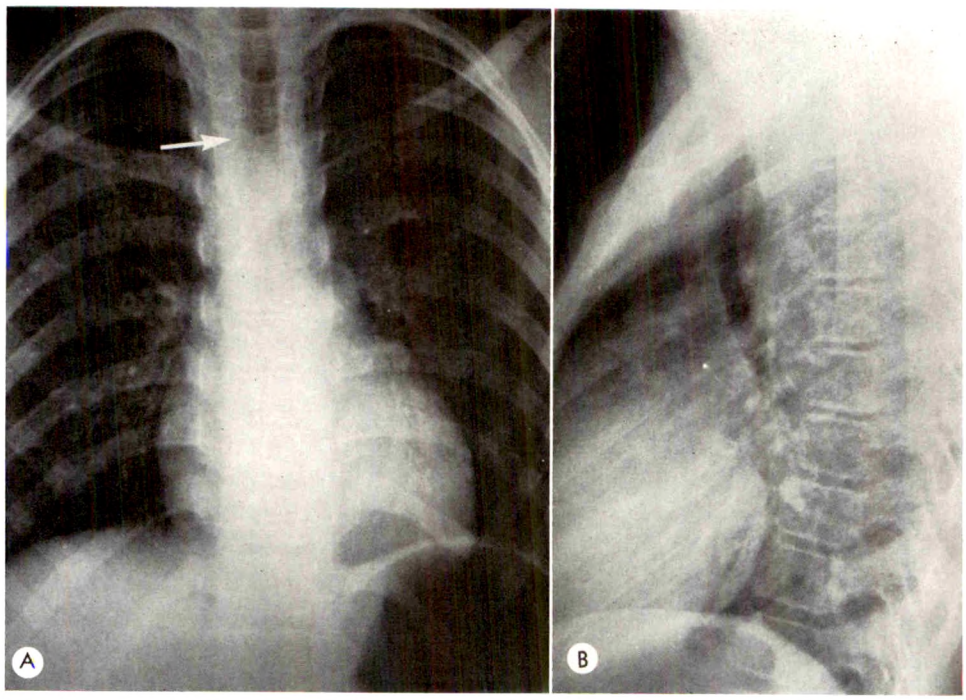


FIG. 1. Right cervical aortic arch. (*A*) The aortic shadow is absent. There is no widening of the mediastinum. The heart is normal in size. An oblique cut-off of the tracheal air shadow above the level of the clavicles is present (arrow). (*B*) In the lateral view, the trachea is displaced forward by the anomalously-placed aorta.

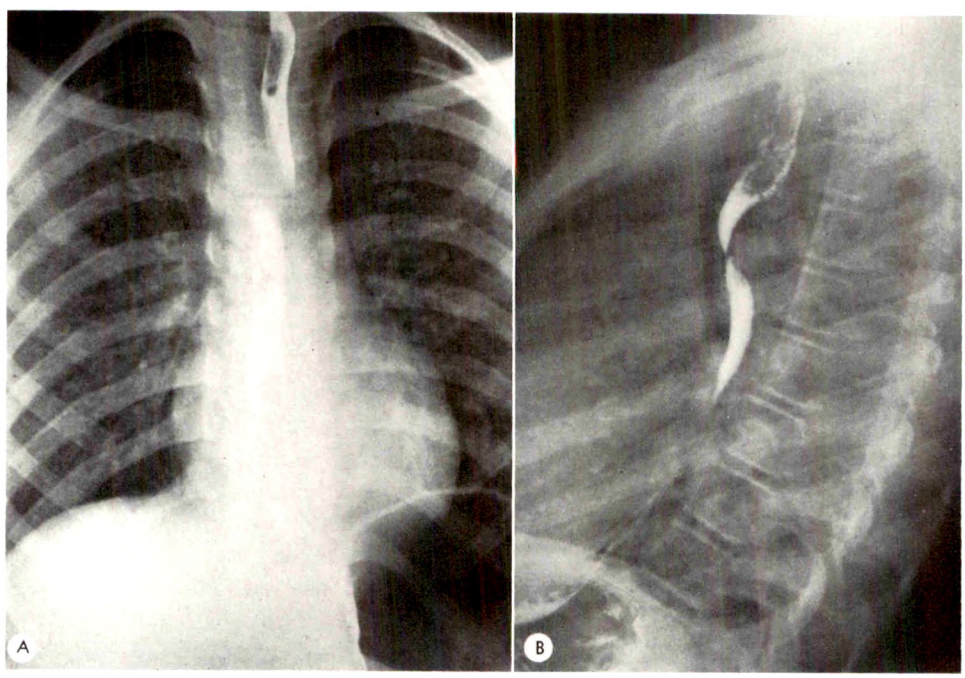


FIG. 2. Right cervical aortic arch. Barium swallow examination. (*A*) The retroesophageal aorta produces an oblique posterior compression defect on the esophagus. (*B*) The esophagus is displaced anteriorly by the aorta.

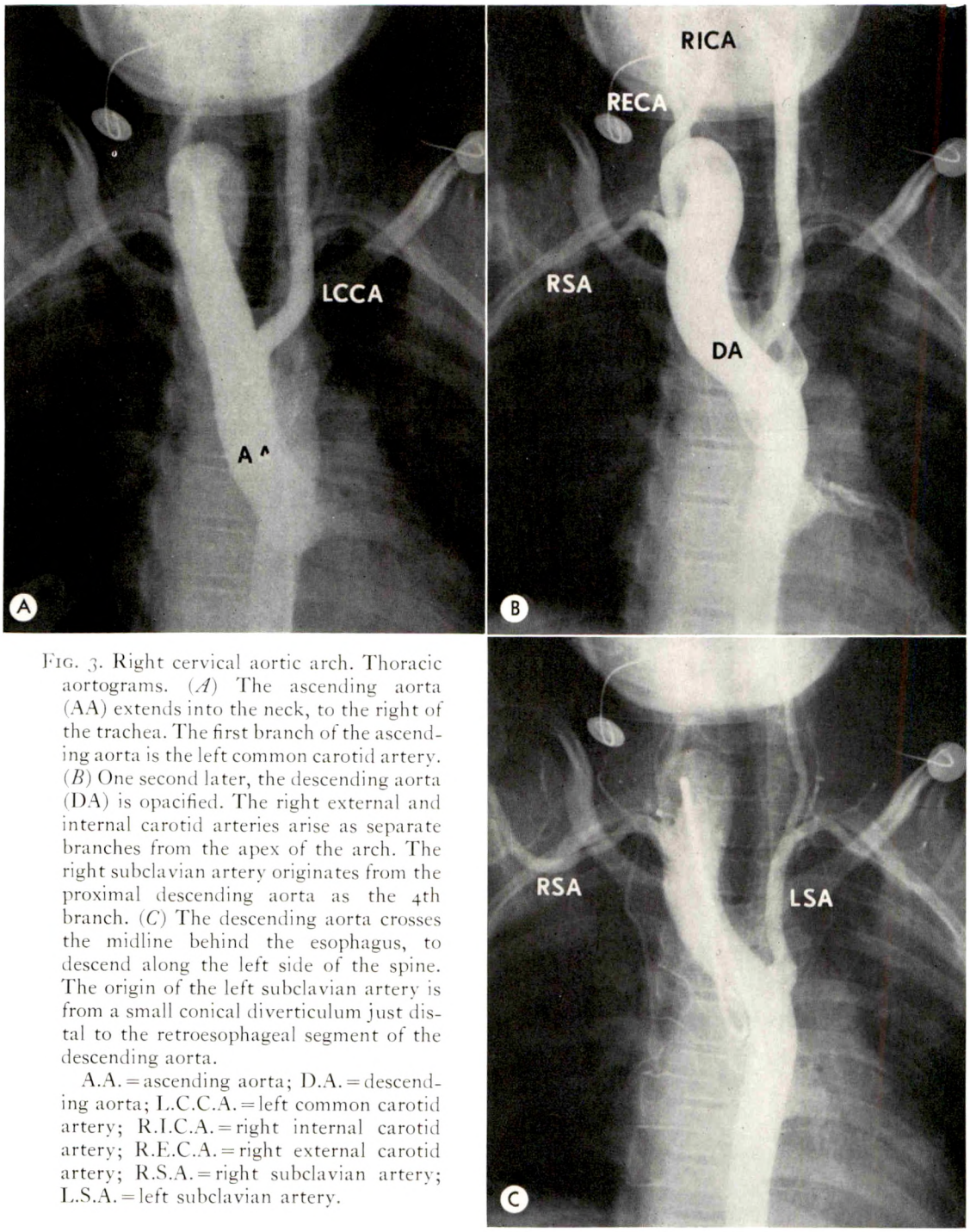


FIG. 3. Right cervical aortic arch. Thoracic aortograms. (*A*) The ascending aorta (AA) extends into the neck, to the right of the trachea. The first branch of the ascending aorta is the left common carotid artery. (*B*) One second later, the descending aorta (DA) is opacified. The right external and internal carotid arteries arise as separate branches from the apex of the arch. The right subclavian artery originates from the proximal descending aorta as the 4th branch. (*C*) The descending aorta crosses the midline behind the esophagus, to descend along the left side of the spine. The origin of the left subclavian artery is from a small conical diverticulum just distal to the retroesophageal segment of the descending aorta.

A.A. = ascending aorta; D.A. = descending aorta; L.C.C.A. = left common carotid artery; R.I.C.A. = right internal carotid artery; R.E.C.A. = right external carotid artery; R.S.A. = right subclavian artery; L.S.A. = left subclavian artery.

tions of the aortic arch. In previous articles, the development of some of the right aortic arch anomalies[16] and the double aortic arch malformations[17] have been discussed.

In the reported cases of cervical aortic arch, the branching of the arch vessels has been of several types. In the 2 cases of left cervical aortic arch, there was normal branching of the arch vessels in 1 pa-

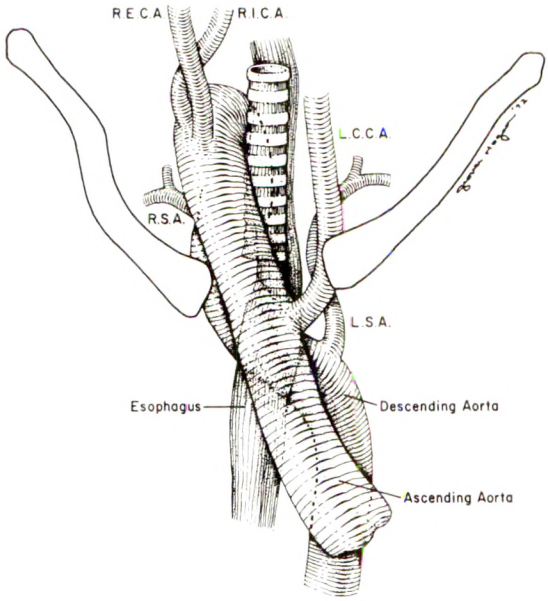


FIG. 4. Right cervical arch. The anatomy of the cervical aorta, its relation to the trachea and esophagus, and the branching of the arch vessels are shown.

L.C.C.A. = left common carotid artery; R.E.C.A. = right external carotid artery; R.I.C.A. = right internal carotid artery; R.S.A. = right subclavian artery; L.S.A. = left subclavian artery.

tient.[11,12] In the other, there were separate origins of the left external and internal carotid arteries, and an aberrant right subclavian artery.[5]

In most of the reported cases of right cervical aortic arch, the right carotid arteries arose as independent vessels.[2,3,8–10] In a few cases, the branching of the arch vessels is not well documented, but apparently in some of these cases the carotid arteries arose from a common trunk.[14,15] In all cases of right cervical aortic arch, the left subclavian artery originated as the last branch of the aortic arch from the retroesophageal segment of the aorta.

For an explanation of these cervical aortic arch anomalies using the concept of a functioning double aortic arch system, it is necessary to postulate the existence of 2 types of double aortic arches (Fig. 5, *A* and *B*). In 1, each embryonic aortic arch gives rise to a common carotid artery (Fig. 5*A*), and in the other, there is separate origin of the external and internal carotid arteries from each embryonic aortic arch (Fig. 5*B*).

Figure 6 illustrates the basic Rathke diagram consisting of paired dorsal and ventral aortae with their 6 pairs of interconnecting branchial arches. Figure 7, *A–C*, shows the alterations in the Rathke diagram for the development of a hypothetical double aortic arch with the carotid arteries arising as a common trunk.[1,4]

In Figure 8, *A–C*, the alterations in the Rathke diagram for the development of a hypothetical double aortic arch with the carotid arteries arising as independent vessels are illustrated.[1,4]

Normally, the aortic arch represents persistence of the 4th branchial arch.[1,4,6] Some authors postulate that the cervical aortic arch may represent an exception to this rule, the aortic arch being derived instead from the 2nd[12] or 3rd branchial arch.[2,8,10] Thus, it is for this reason that we have shown in Figures 7 and 8 the theoretical possibilities for the development of the aortic arch from the 2nd and 3rd branchial arches as well as from the 4th branchial arch.

By interrupting either the right or left embryonic arch in one of these hypothetical double aortic arch systems, the various types of cervical aortic arch may be explained.

Figure 9 shows the result of interruption of the left embryonic arch between the left subclavian artery and the carotid arteries in the hypothetical double arch pattern with separate origin of the carotid arteries. A right aortic arch is formed, and the first branch is the left common carotid artery, followed by separate origins of the right external and internal carotid arteries. The next branch is the right subclavian artery, followed by an aberrant left subclavian artery. Such is the branching of the arch vessels in most of the reported cases of right cervical aortic arch.

Likewise, interruption of the right em-

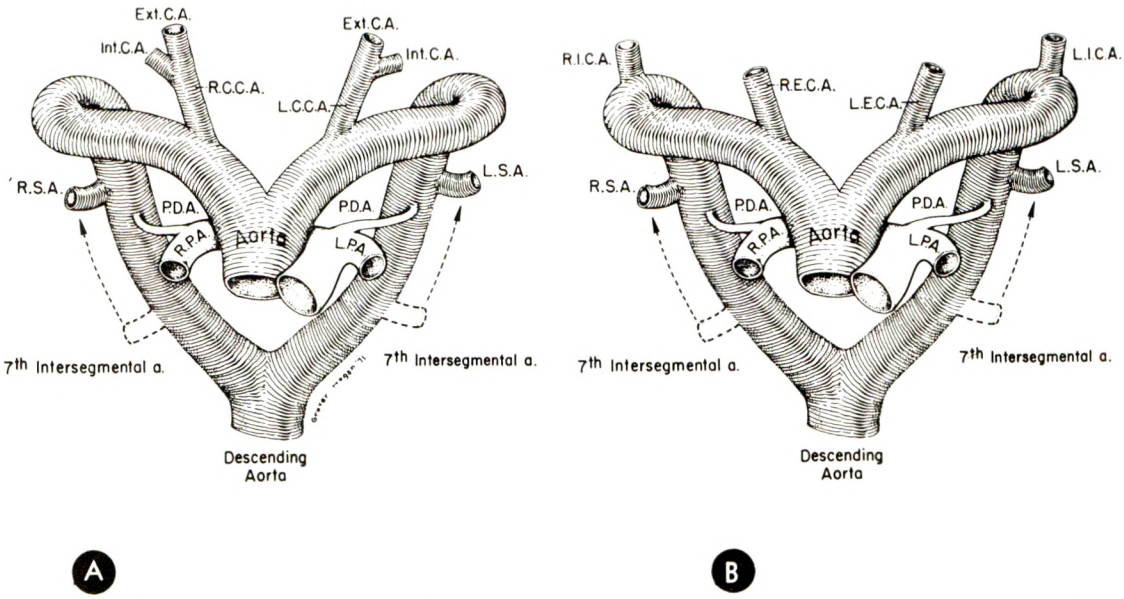


Fig. 5. (*A*) Hypothetical functioning double aortic arch with the carotid arteries arising from each arch as a common trunk.

L.C.C.A. = left common carotid artery; R.C.C.A. = right common carotid artery; Int.C.A. = internal carotid artery; Ext.C.A. = external carotid artery; L.S.A. = left subclavian artery; R.S.A. = right subclavian artery; P.D.A. = patent ductus arteriosus; L.P.A. = left pulmonary artery; R.P.A. = right pulmonary artery.

(*B*) Hypothetical functioning double aortic arch with the external and internal carotid arteries arising separately from each arch.

R.E.C.A. = right external carotid artery; R.I.C.A. = right internal carotid artery; L.E.C.A. = left external carotid artery; L.I.C.A. = left internal carotid artery.

bryonic arch between the right subclavian artery and the carotid arteries in this hypothetical pattern (Fig. 10) explains the development of the one case of left cervical aortic arch with independent origin of the carotid arteries.[5]

Interruption of the right embryonic arch between the right subclavian artery and descending aorta in the hypothetical double arch pattern with the carotid arteries arising as a common trunk would result in a left aortic arch with normal branching of the arch vessels (Fig. 11). In Mahoney and Manning's case of left cervical aortic arch, the branching of the arch vessels was of this type.[12]

Theoretically, the cervical aortic arch may represent persistence of either the 2nd, 3rd, or 4th branchial arch. Those who postulate persistence of the 3rd branchial arch emphasize the high position of the aortic arch and the separate origin of the carotid arteries as evidence for this theory.[2,8,10] This argument is not necessarily valid, because, as shown in Figure 8,

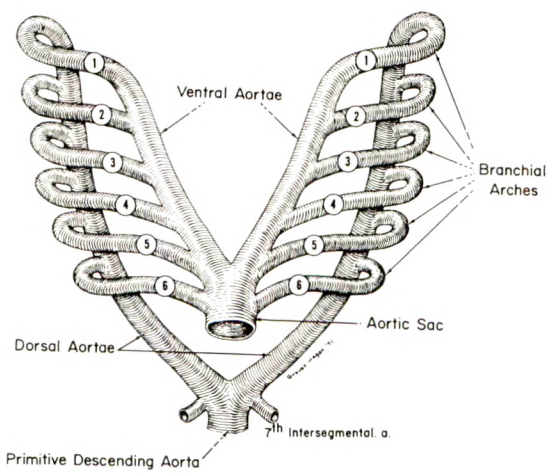


Fig. 6. Basic Rathke diagram consisting of paired dorsal and ventral aortae with their 6 pairs of interconnecting branchial arches.

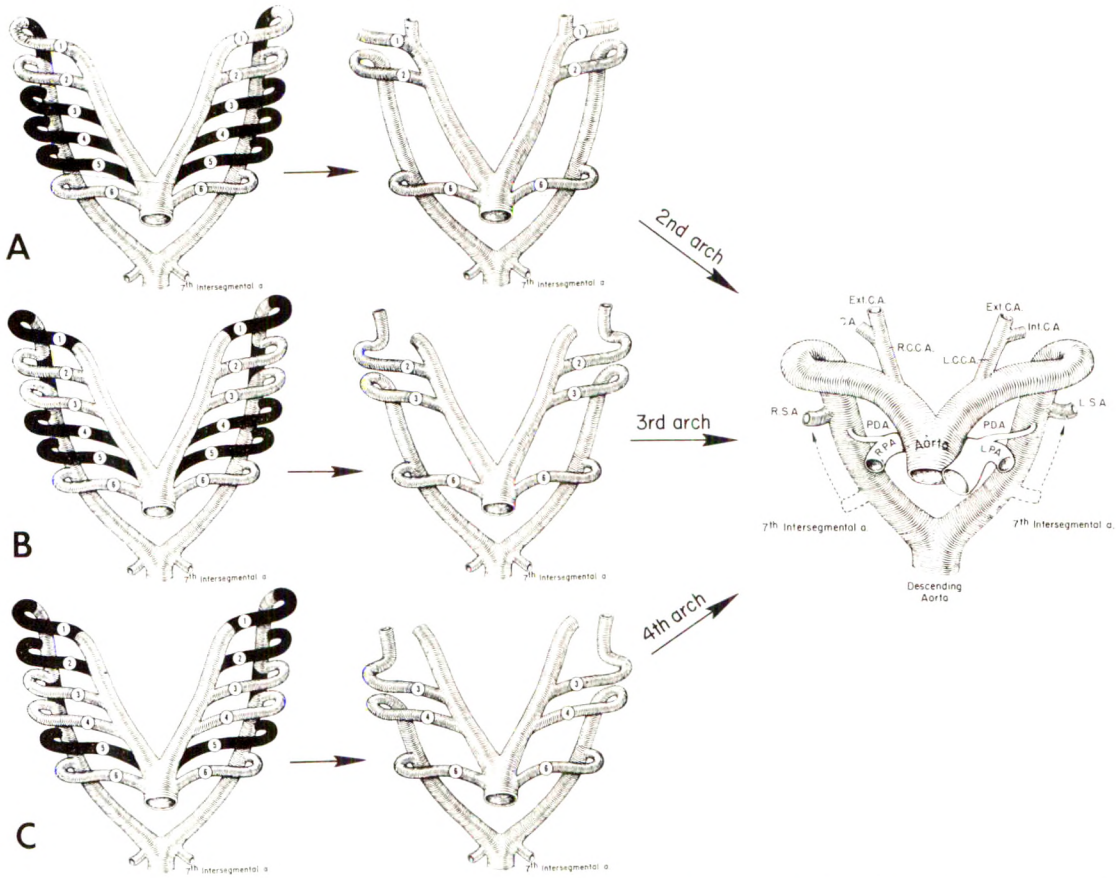


FIG. 7. Alterations in the Rathke diagram for the development of a hypothetical double aortic arch with the carotid arteries arising from each arch as a common trunk. The theoretic development of the aortic arch in this hypothetical pattern from: (*A*) the 2nd branchial arch; (*B*) the 3rd branchial arch; and (*C*) the 4th branchial arch, is shown. The 6 paired branchial arches are numbered. Areas in black indicate segments of regression.

L.C.C.A. = left common carotid artery; R.C.C.A. = right common carotid artery; Ext.C.A. = external carotid artery; Int.C.A. = internal carotid artery; R.S.A. = right subclavian artery; L.S.A. = left subclavian artery.

the carotid arteries may arise independently from the aortic arch—regardless of whether the arch is formed from the 2nd, 3rd, or 4th branchial arch.

The location of the aortic arch in the neck would seem to favor formation of the aortic arch from either the 2nd or 3rd branchial arch. If the cervical arch is derived normally, *i.e.*, from the 4th arch,[7,13] then, for some unknown reason, it would have to be retained abnormally in the neck. There appears to be no way of telling which of these possibilities is the correct one.

Chest Roentgenography. Nonspecific widening of the mediastinum may be the only abnormality. The trachea is usually displaced forward by the retroesophageal position of the distal arch. We have observed an oblique cut-off of the tracheal air shadow at the level of the thoracic inlet in the frontal projection due to the superimposition of the ascending aorta on its upward course to the neck. The aortic knob shadow is absent.

Barium swallow studies of the esophagus show a posterior compression defect from the retroesophageal position of the distal arch. The esophagus may be displaced

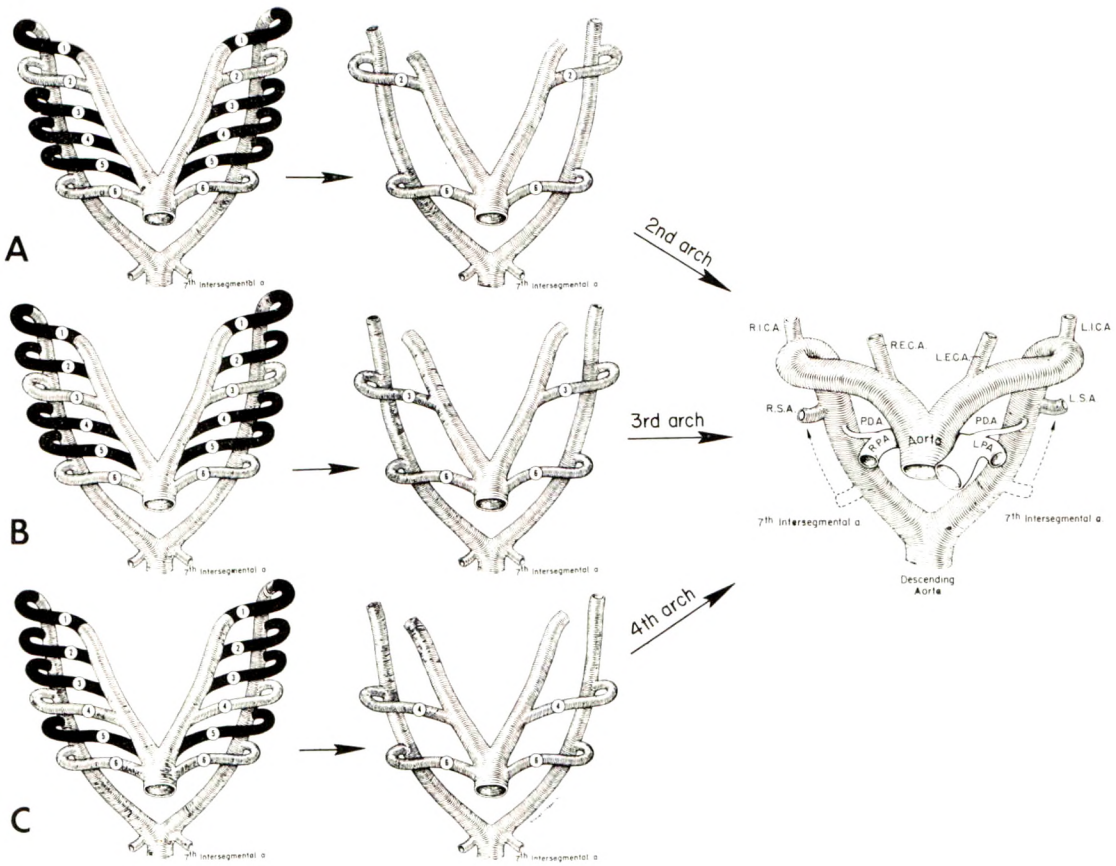


Fig. 8. Alterations in the Rathke diagram for the development of a hypothetical double aortic arch with the internal and external carotid arteries arising separately from each arch. The theoretic development of the aortic arch in this hypothetical pattern from: (*A*) the 2nd branchial arch; (*B*) the 3rd branchial arch; and (*C*) the 4th branchial arch, is shown. The 6 paired branchial arches are numbered. Areas in black indicate segments of regression.

L.E.C.A. = left external carotid artery; L.I.C.A. = left internal carotid artery; R.E.C.A. = right external carotid artery; R.I.C.A. = right internal carotid artery.

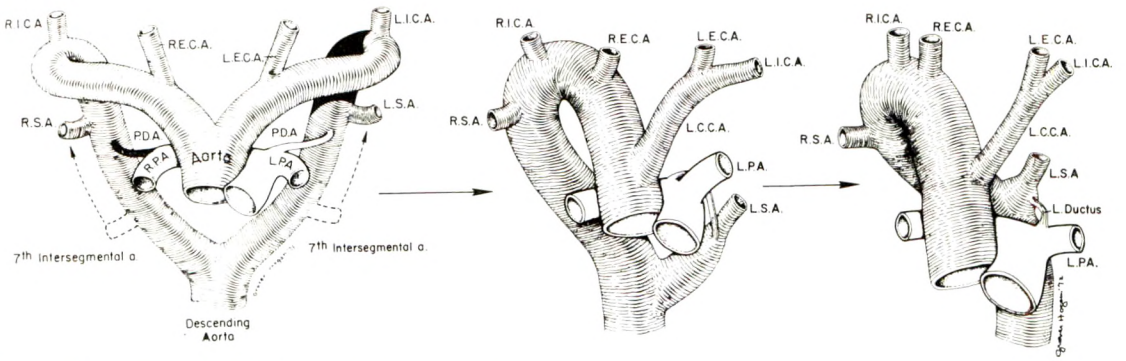


Fig. 9. Development of right cervical aortic arch with the right internal and external carotid arteries arising as separate branches. There is interruption of the left embryonic arch between the left subclavian artery and the carotid arteries in the hypothetical double aortic arch with independent origin of the carotid arteries. The left common carotid artery is the first branch, followed by the right external carotid artery, the right internal carotid artery, the right subclavian artery and the left subclavian artery, in that order. Areas in black indicate segments of complete regression.

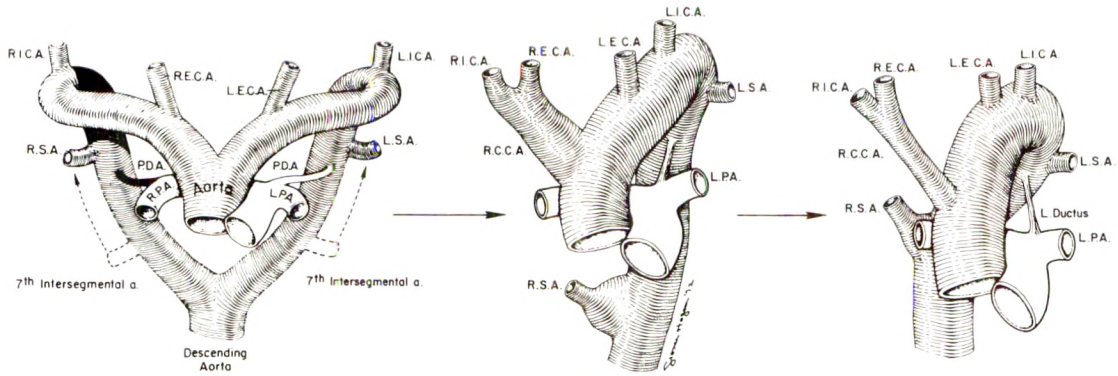


FIG. 10. Development of left cervical aortic arch with the left internal and external carotid arteries arising as separate branches. There is interruption of the right embryonic arch between the right subclavian artery and the carotid arteries in the hypothetical double aortic arch with separate origin of the carotid arteries. Areas in black indicate segments of complete regression.

laterally in the neck by the cervical aorta.

Angiographic Features. The aortographic findings are striking. The aorta ascends into the neck, either to the right or left side, the apex of the arch makes a hairpin loop, and returns to the thorax, crossing the midline behind the esophagus, to descend along the spine opposite the side of the arch.

When the arch is right-sided, the left common carotid artery is the 1st branch and originates from the ascending aorta. The right external and internal carotid arteries usually originate as independent branches, although they may arise from a common trunk. The right subclavian artery is the next branch off the arch. The origin of the left subclavian artery is just distal to the retroesophageal segment of the aorta and may be from a conical diverticulum. In 1 case, the left subclavian artery was either isolated from the descending aorta, or was connected to the descending aorta by an atretic segment.[13]

In the 2 reported cases of left-sided cervical aortic arch, the branching of the arch vessels was different. In the case of de Jong and Klinghamer,[5] the right common carotid artery was the first branch of the ascending aorta followed by separate origins of the left external and internal carotid arteries. The left subclavian artery arose from the apex of the left arch, and the right subclavian artery had an aberrant origin as the last branch of the distal aortic arch.

The origin of the arch vessels in the case reported by Mahoney and Manning[12]

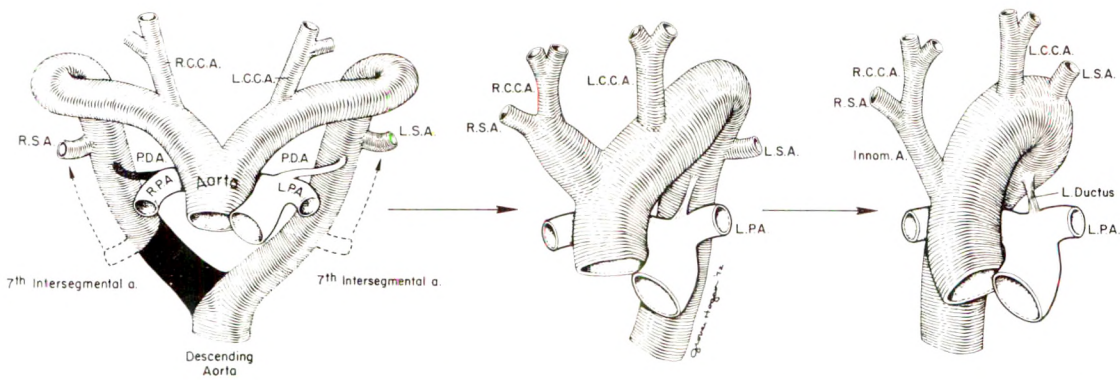


FIG. 11. Development of left cervical aortic arch with normal branching of the arch vessels. There is interruption of the right embryonic arch between the right subclavian artery and descending aorta in the hypothetical double aortic arch with the carotid arteries arising as a common trunk. Areas in black indicate segments of complete regression.

(same case reported by Lipchik and Young[11]) apparently was identical to the branching of the normal left arch. The innominate artery was the first branch, followed by the left common carotid and left subclavian arteries.

Diagnosis. If one is aware of this rare arch anomaly, there are characteristic features which should permit its recognition.[3,13]

Clinically, this anomaly should be suspected when one encounters a child with a pulsatile mass in the neck, with or without symptoms of a vascular ring. Compression of the neck mass may cause a decrease in heart rate, with diminution of the blood pressure in the opposite arm and legs.[13]

Barium swallow studies of the esophagus may be most helpful, and previous reports have not emphasized the value of this examination in the diagnosis of cervical aortic arch. The large pulsating oblique defect just below the clavicles on the frontal view, with a prominent rounded indentation on the posterior esophageal wall in the lateral projection, identifies the retroesophageal course of the descending aorta. However, contrast studies of the aorta are necessary for a definitive diagnosis.

SUMMARY

The 12th reported case of cervical aortic arch, the first to appear in the radiologic literature, is presented.

This anomaly should be suspected in any child exhibiting a pulsatile swelling in the neck.

The roentgenologic features of this malformation are described.

It is postulated that the cervical aortic arch may be formed from either the 2nd, 3rd, or 4th branchial arch.

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REFERENCES

1. Barry, A. Aortic arch derivatives in human adult. *Anat. Rec.*, 1951, *111*, 221–238.
2. Beaven, T. E., and Fatti, L. Ligature of aortic arch in neck. *Brit. J. Surg.*, 1947, *34*, 414–416.
3. Chang, L. W., Kaplan, E. L., Baum, D., and Figley, M. M. *J. Pediat.*, 1971, *79*, 788–793.
4. Congdon, E. D. Transformation of aortic arch system during development of human embryo. *Contrib. Embryol.*, 1922, *14*, 47–110.
5. de Jong, I. H., and Klinghamer, A. C. Left sided cervical aortic arch. *Am. J. Cardiol.*, 1969, *23*, 285–287.
6. Edwards, J. E. Anomalies of the derivatives of aortic arch system. *Med. Clin. North America*, 1948, *32*, 925–949.
7. Gravier, J., Vialtel, M., and Pinet, F. À propos d'une tumeur pulsatile du cou: un cas d'aorte cervicale. *Pédiatrie*, 1959, 437–438.
8. Harley, H. R. Development and anomalies of aortic arch and its branches. *Brit. J. Surg.*, 1959, *46*, 561–573.
9. Hastreiter, A. R., D'Cruz, I. A. *et al.* Right-sided aorta. II. Right aortic arch, right descending aorta, and associated anomalies. *Brit. Heart J.*, 1966, *28*, 772–739.
10. Lewis, C., and Rogers, L. Cervical aortic knuckle which resembles aneurysm. *Lancet*, 1953, *1*, 825–826.
11. Lipchik, E. O., and Young, L. W. Unusual symptomatic aortic arch anomalies. *Radiology*, 1967, *89*, 85–90.
12. Mahoney, E. B., and Manning, J. A. Congenital abnormalities of aortic arch. *Surgery*, 1964, *55*, 1–14.
13. Massumi, R., Wiener, L., and Charif, P. Syndrome of cervical aorta: report of case and review of previous cases. *Am. J. Cardiol.*, 1963, *11*, 678–685.
14. Reid, D. G. Three examples of right aortic arch. *J. Anat. Physiol.*, 1913, *48*, 174–181.
15. Shepherd, R. M., Kerth, W. J., and Rosenthal, J. R. Right cervical aortic arch with left descending aorta: case report and review of literature. *A.M.A. Am. J. Dis. Child.*, 1969, *118*, 642–648.
16. Shuford, W. H., Sybers, R. G., and Edwards, F. K. Three types of right aortic arch. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *109*, 67–74.
17. Shuford, W. H., Sybers, R. G., and Weens, H. S. Angiographic features of double aortic arch. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1972, *116*, 125–140.
18. Stewart, J. R., Kincaid, O. W., and Edwards, J. E. An Atlas of Vascular Rings and Related Malformations of the Aortic Arch System. Charles C Thomas, Publisher, Springfield, Ill., 1964, pp. 8–13, 124–129.

REBOUND ENLARGEMENT OF THE THYMUS AFTER SUCCESSFUL CORRECTIVE SURGERY FOR TRANSPOSITION OF THE GREAT VESSELS*

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THE size of the thymus is variable in the presence of illness. The poor general health of infants with transposition of the great vessels results in thymic atrophy. After successful palliative or corrective surgery, the thymus at times enlarges.[2] This enlargement in isolated cases can be so dramatic as to simulate a mediastinal mass.

In this communication the chest roentgenograms of 40 patients with complete transposition of the great vessels who have had good results after surgery are reviewed. Regeneration of the thymic mass after surgery was evaluated by chest roentgenography.

MATERIAL

The chest roentgenograms of 40 patients who had a Mustard operation were reviewed. Each patient had had a palliative procedure, either a balloon or surgical septostomy prior to definitive surgery. The size of the thymus in each patient before and after surgery, as judged from the anteroposterior and lateral roentgenograms, were compared.

RESULTS

Seventeen patients showed a rebound thymic enlargement to a moderate or marked degree. In 7 the enlargement was noted after surgical septostomy (Blalock-Hanlon) (Fig. 1, *A* and *B*); in 6 after a surgical correction (Mustard procedure) (Fig. 2, *A* and *B*); and in 3 after a balloon septostomy (Rashkind procedure) (Fig. 3, *A* and *B*). The remaining 24 patients did not show a change in thymic size that could be detected on the chest roentgenograms. In 1 patient the thymic enlargement was massive and simulated a mediastinal mass (Fig. 4, *A* and *B*).

All patients that showed thymic enlargement are doing well clinically. However, 80

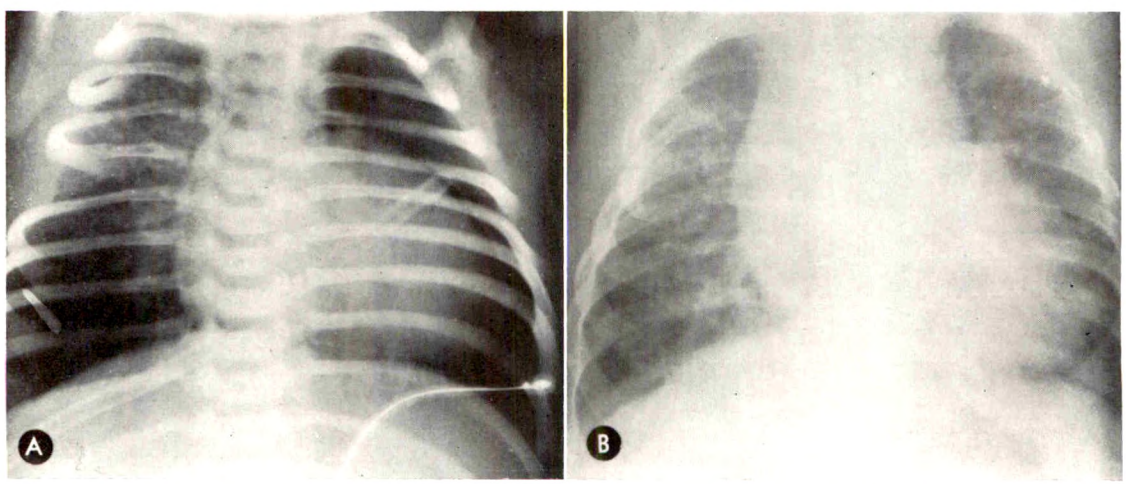


FIG. 1. Anterior chest roentgenograms of a 1 month old infant the day of a Blalock-Hanlon operation (*A*) and (*B*) 3 months later.

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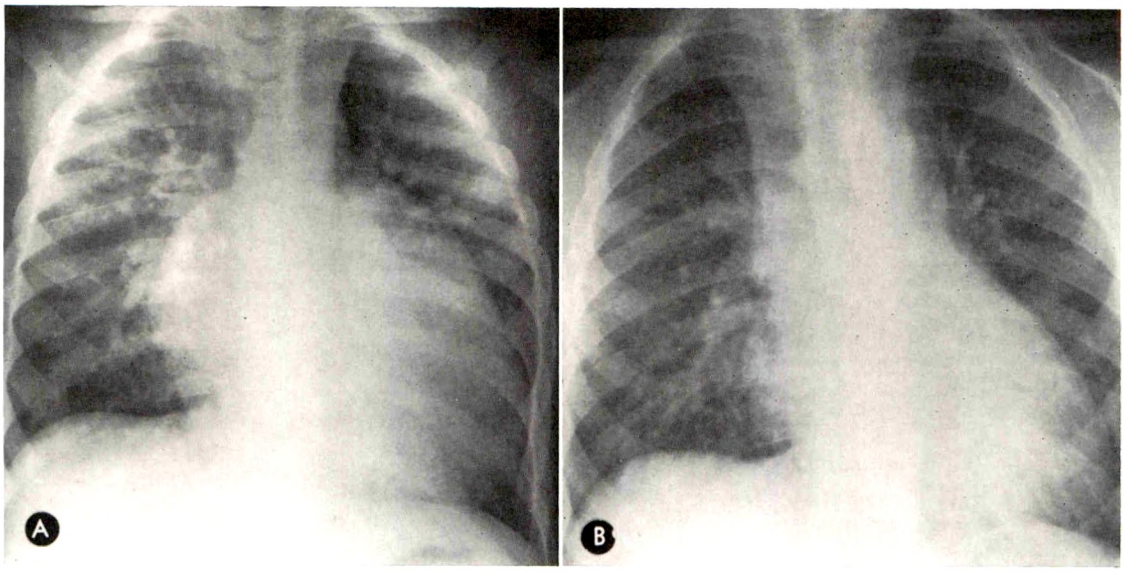


FIG. 2. A 2 year old child 1 month prior to a Mustard procedure (*A*) and (*B*) 6 weeks postoperative.

procedures were performed on these 40 patients, and clinical improvement resulted after 56 procedures. In other words, 28 per cent of patients who benefited clinically from the operation showed thymic enlargement.

DISCUSSION

It is a well-known fact that the thymus quickly atrophies with such stresses as fever, malnutrition, or following the administration of corticoids or lysozyme. After recovery, or when the administration of adrenocorticoids or lysozymes is stopped, the thymus has been noted in some patients to regrow and occasionally to surpass the original size.[1,2,4]

The stressful existence and poor feeding of the infant with transposition of the great vessels are probably the cause of thymic atrophy. The absent thymic shadow is responsible for the "narrow waist" appearance of the heart in severe congenital cyanotic heart conditions.[3,5]

SUMMARY

The regrowth of the thymus after corrective surgery has received little attention. When it occurs, it can safely be taken as a

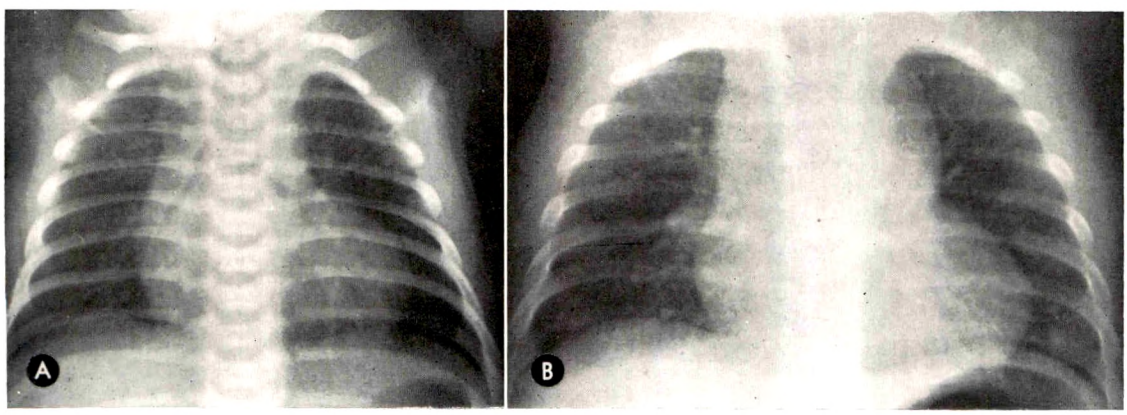


FIG. 3. A 2 month old infant before (*A*) and (*B*) 1 month after a balloon septostomy.

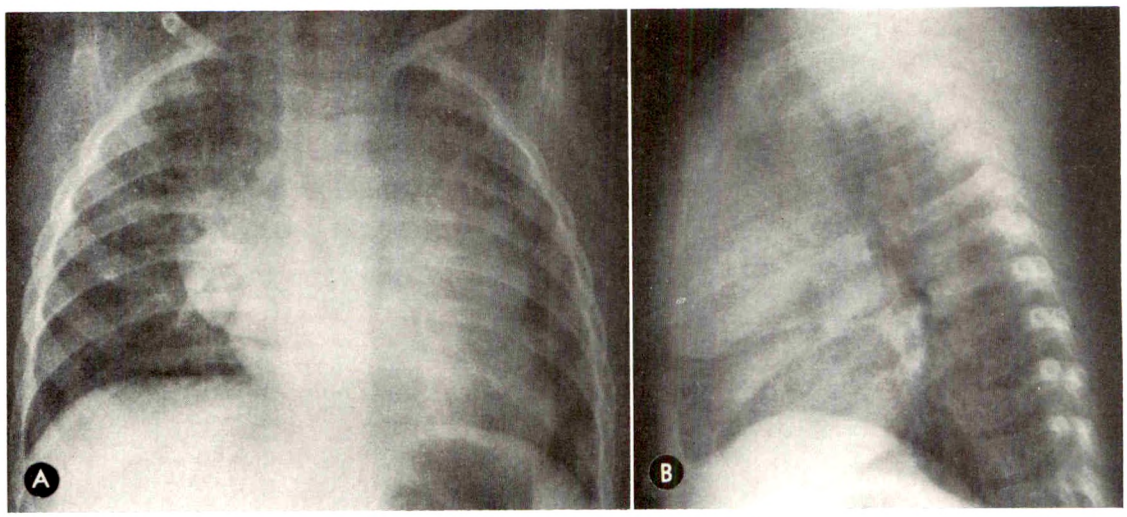


FIG. 4. A 10 month old infant. (*A*) Posteroanterior and (*B*) lateral chest roentgenograms show massive enlargement of the thymus 4 months after corrective surgery for transposition of the great vessels.

sign of improvement in the patient's condition.

Occasionally, the regrowth of thymus is massive. This rebound enlargement should not be mistaken for a mediastinal hematoma, abscess or other complications of cardiac surgery.

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REFERENCES

1. CAFFEY, J., and SILBEY, R. Regrowth and overgrowth of thymus after atrophy induced by oral administration of adrenocorticoids to human infants. *Pediatrics*, 1960, *26*, 762–770.
2. CAFFEY, J. Pediatric X-Ray Diagnosis. Year Book Publishers, Inc., Chicago, 1967, pp. 361–366.
3. EDWARDS, J. E., CAREY, L. S., NEUFELD, H. N., and LESTER, L. G. Congenital Heart Disease. W. B. Saunders Company, Philadelphia, 1965, pp. 382.
4. LEVY, J. M., STOLL, C., and FRANCFORT, J. J. L'hypertrophie thymique du nourisson: effect du lysozyme. *Ann. Paediat.*, 1971, *18*, 138–145.
5. MOSS, A. J., and ADAMS, F. H. Heart Disease in Infants, Children, and Adolescents. Williams & Wilkins Company, Baltimore, 1968, pp. 539.

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SKELETAL CHANGES ASSOCIATED WITH ADOLESCENT CONGENITAL HEART DISEASE*

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IN CLINICAL medicine and radiology we often think in terms of the effects that disease in one organ system may have upon another organ system. For example, cyanotic congenital heart disease (CHD) is associated with lung abscess, brain abscess, polycythemia, and clotting disorders. The first 2 result directly from right-to-left shunting, while the latter 2 result from overproduction of hematopoietic tissue.

Exclusive of the association of bone dysplasias and trisomies with CHD, relatively little has been written about the general effects of CHD upon the skeleton. Table 1 summarizes some of the skeletal abnormalities previously described in association with CHD.

While soft tissue clubbing commonly accompanies cyanotic CHD, pulmonary osteoarthropathy remains a most unusual complication. Periosteal proliferative changes have been produced in the experimental animal following pulmonary artery-left atrial anastomosis and have been described rarely in patients with cyanotic CHD.[17,22,24] The second metacarpal, radius and ulna of a 16 year old patient with pseudotruncus arteriosus and pulmonary osteoarthropathy are demonstrated in Figure 1, *A* and *B*. This is the only case diagnosed roentgenographically from the Johns Hopkins Pediatric Cardiac Clinic which includes in excess of 3,500 patients, 850 of whom are children and adolescents with cyanotic CHD.

Somerville in 1961 described 9 cases of gout complicating cyanotic CHD.[21] Tophi were present in 7 patients and the joints involved; the roentgenographic appearances and the sex incidence were similar to hereditary gout. All patients were over 18 years of age, and there was a rough correlation between the hemoglobin level and the level of serum uric acid. We have studied roentgenographically 3 patients with clini-

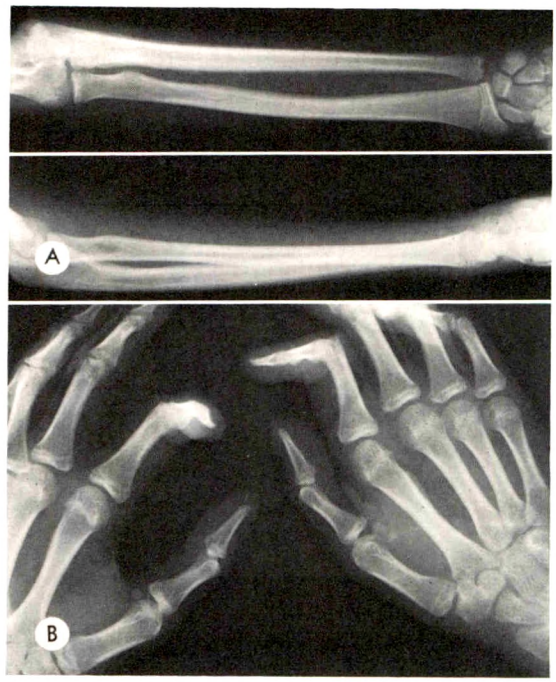


FIG. 1. (*A*) Forearm and (*B*) hands from a 16 year old patient with pseudotruncus arteriosus and pulmonary osteoarthropathy. Periosteal proliferation is present along the medial and lateral aspects of the ulna and along the lateral aspect of both second metacarpal bones.

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TABLE I

SKELETAL CHANGES ASSOCIATED WITH CONGENITAL HEART DISEASE (CHD)

1. Hypertrophic Pulmonary Osteoarthropathy (22, 24)*
2. Gout (21)
3. Delayed Skeletal Maturation (4, 20)
4. Medullary Cavity Expansion (15, 18)
5. Premature Fusion of the Sternum (8, 10)
6. Scoliosis (14, 25)

* References included in parentheses.

cal symptoms suggesting gout and elevated serum uric acid. Only soft tissue swelling was present on the roentgenograms, but it is of interest that all 3 patients responded symptomatically to colchicine therapy and uricosuric agents.

In order to assess the other 4 skeletal abnormalities associated with CHD (Table 1), we undertook a prospective study of 129 randomly selected adolescent and young adult patients with CHD from the outpatient cardiac clinic of the Johns Hopkins Hospital.

METHOD

All patients were ambulatory and, while restricted from vigorous activity, most were employable or able to attend school regularly. Skeletal growth was assessed by measurement of the total (T) and medullary (M) widths of the second metacarpal bone by methods previously described.[10,23] Cortical width (C) was the difference between T and M. Values for C, T, and M were compared with age and sex matched controls from a previous study.[23] Skeletal maturation (bone age) was determined by comparison of the patient's hand roentgenogram with a standard atlas.[11]

The presence of scoliosis was assessed from roentgenograms of the thoracic and lumbar spine and the magnitude of the curvature was determined by the Cobb method.[5] In most instances (78 per cent) prior chest roentgenograms were available for evaluation of the age of onset, rapid progression and stabilization of the curves.

Lateral chest roentgenograms were evaluated for manubrial-sternal fusion and fusion of the body of the sternum (mesosternum). Earlier roentgenograms when available were evaluated for the time and sequence of fusion of the sternal segments.[7]

Four patients were excluded from the study because of associated skeletal dysplasias (Ellis van Creveld and Holt-Oram).

RESULTS

SKELETAL GROWTH AND MATURATION

Values for metacarpal T were below the 16th percentile in 40 per cent of cyanotic patients while M was mildly increased above the 84th percentile in 28 per cent of cyanotic patients. Metacarpal cortical width (C) was below the 16th percentile in 66 per cent of the cyanotic population (Fig. 2, *A–C*). As a group the acyanotic patients were much less affected, the corresponding values for T and C were 28 and 34 per cent below the 16th percentile, respectively. There were no important differences in the values for T, M, and C among the male and female patients in the cyanotic and in the acyanotic groups. Similarly, patients in the third decade had values for C and T just as low as those for patients in the second decade.

Bone age was retarded below the 16th percentile in 50 per cent of the cyanotic patients. There was no significant retardation in bone age among the acyanotic patients. Five of 12 cyanotic patients with normal bone age had metacarpal C values below the 16th percentile, indicating that reduced cortical bone formation was possible in the presence of normal skeletal maturation.

SCOLIOSIS

Four of the 125 patients (3 per cent) had congenital anomalies of the spine (butterfly and hemivertebra) leading to thoracic scoliosis (Fig. 3, *A* and *B*). Of the remaining 121 patients, 53 or 44 per cent of the total had thoracic scoliosis without detectable bone anomaly. In all but 2 cases the primary curvature was thoracic or thoracolumbar. It was mild ($<10°$) in 30 (25 per cent), moderate ($10–30°$) in 19 (16 per cent) and severe ($>30°$) in 4 (3 per

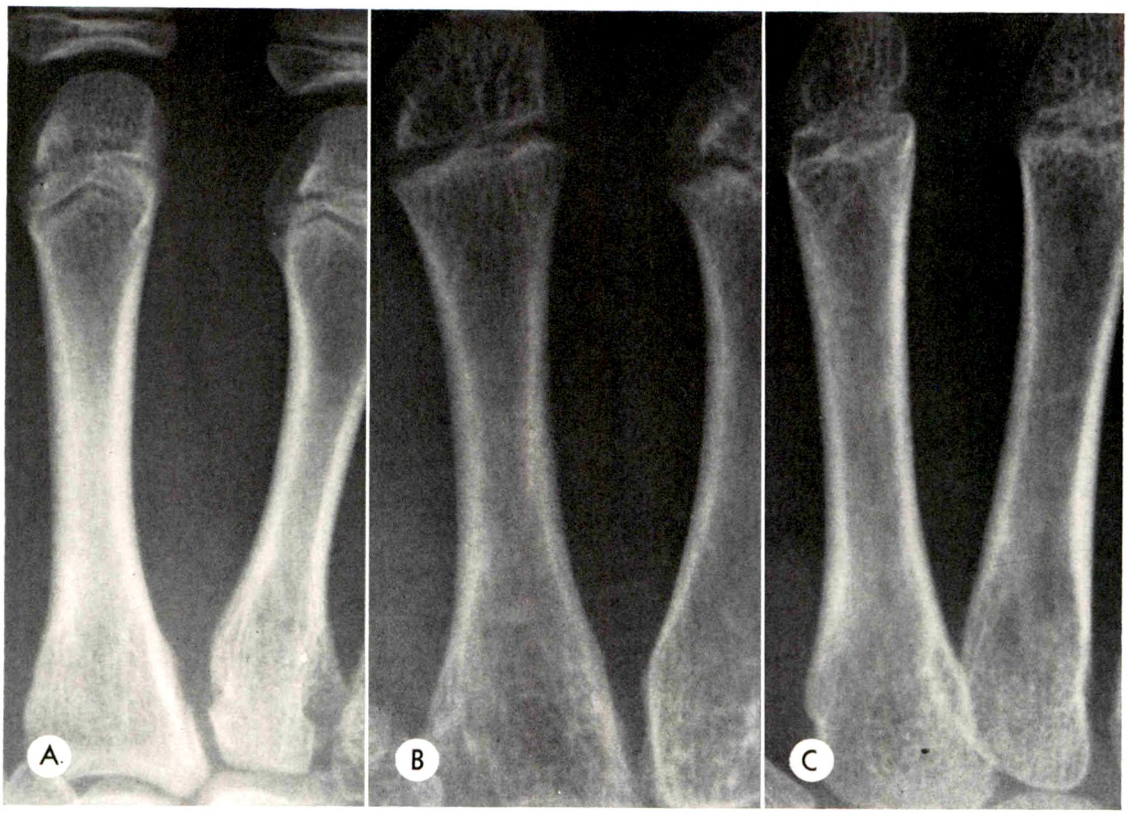


FIG. 2. Second metacarpal bones from two 14 year old patients with (*A*) isolated dextrocardia and (*B*) tetralogy of Fallot. (*C*) Second metacarpal bone from a 12 year old patient with truncus arteriosus. Cortical thickness is normal in *A* and reduced below the second percentile in *B* and *C*. Bone age was normal in *A* and delayed 2 years in *B* and *C*.

cent). There were striking differences in the incidence of scoliosis among the cyanotic and acyanotic groups. Thirty-seven (66 per cent) of the patients with cyanotic CHD had scoliosis, while only 16 (24 per cent) of the acyanotic patients had scoliosis. All 4 patients with severe scoliosis (38°–76°) were cyanotic, and cyanotic patients with moderate scoliosis outnumbered acyanotic patients with moderate scoliosis 2:1 (Fig. 4, *A–F*). Scoliosis was more common in female than in male patients in the ratio of 1.7:1.

The average age of onset of the scoliosis was 11 years in those with curvatures less than 30° and 6 years in those with curvatures greater than 30°. The average age at the time of rapid progression of the curvature was 12 years in both groups, and the curves tended to become stable in the late teens.

STERNUM

The data on sternal fusion are more conveniently discussed by examining separately manubrial-sternal (M-S) joint fusion and fusion of the body of the sternum (mesosternum). Lateral chest roentgenograms were adequate for evaluation in 119 patients.

Eighteen patients (16 per cent) had fusion of the M-S joint as compared with an incidence of 2 per cent among normal adolescents.[3] M-S joint fusion was more common in cyanotic than in acyanotic patients (13/18) and 8 of those 13 cyanotic patients were females.

Fusion of the mesosternum occurred in 70 of the 119 patients (59 per cent). As with M-S joint fusion, mesosternal fusion occurred most frequently in the cyanotic female group, where 19/20 (95 per cent) were fused (Fig. 5, *A–C*). Comparison of

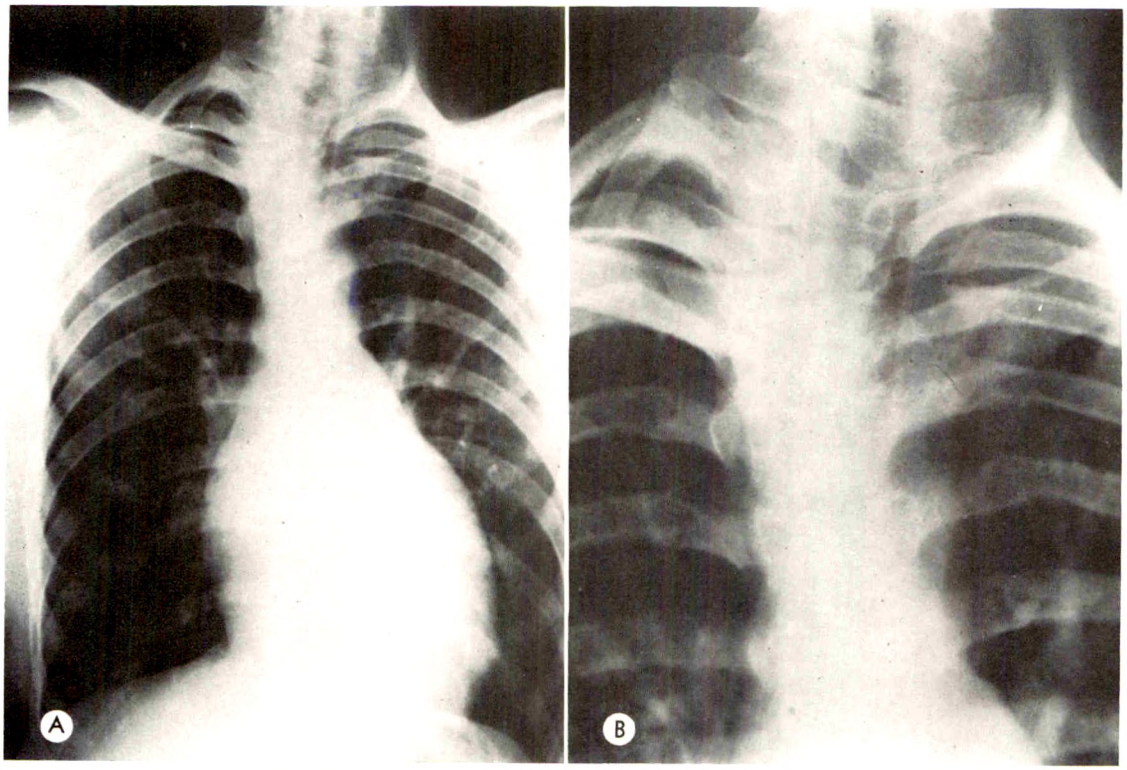


FIG. 3. (*A* and *B*) Chest roentgenograms from a 14 year old patient with ventricular septal defect and thoracic scoliosis associated with a spinal anomaly. Hemivertebrae and butterfly vertebrae are demonstrated in the upper thoracic spine.

our data with the incidence of mesosternal fusion in normals revealed an increased incidence of fusion in the patients with CHD at all ages evaluated.[19]

DISCUSSION

For several reasons, it was anticipated that the adolescent and young adult population would provide an interesting group for study of the effects of CHD upon the skeleton. First, these patients, many of whom have severe heart disease, were able to survive into adolescence, thus allowing time for maximal changes in the skeleton to occur. Second, the work of Garn and his associates[9,10] has established that the rate of cortical bone formation in normal populations is maximal during adolescence. Finally, on the basis of our clinical observations, it was apparent that the scoliosis associated with CHD was most prevalent among adolescent and young adult patients.

Cortical thinning and medullary cavity expansion have been documented previously both by pathologic and roentgenographic examination of patients with cyanotic heart disease.[15,18] In these studies, however, cortical thinning was attributed primarily to marrow hyperplasia. It is clear from our studies of cyanotic patients that the reduction in cortical bone (C) is not simply a function of marrow expansion but is due also to failure of subperiosteal bone growth as manifested by reduced total width of the bone (T). Patients in the third decade are just as affected as those in the second decade, and our limited observations of 4 cyanotic patients in the fourth decade indicate that they too have a striking reduction in cortical bone.

Roentgenographically the metacarpals and the rest of the skeleton appear osteoporotic and if one considers osteoporosis an inability to form osteoid matrix, then this is a form of "osteoporosis." This "osteo-

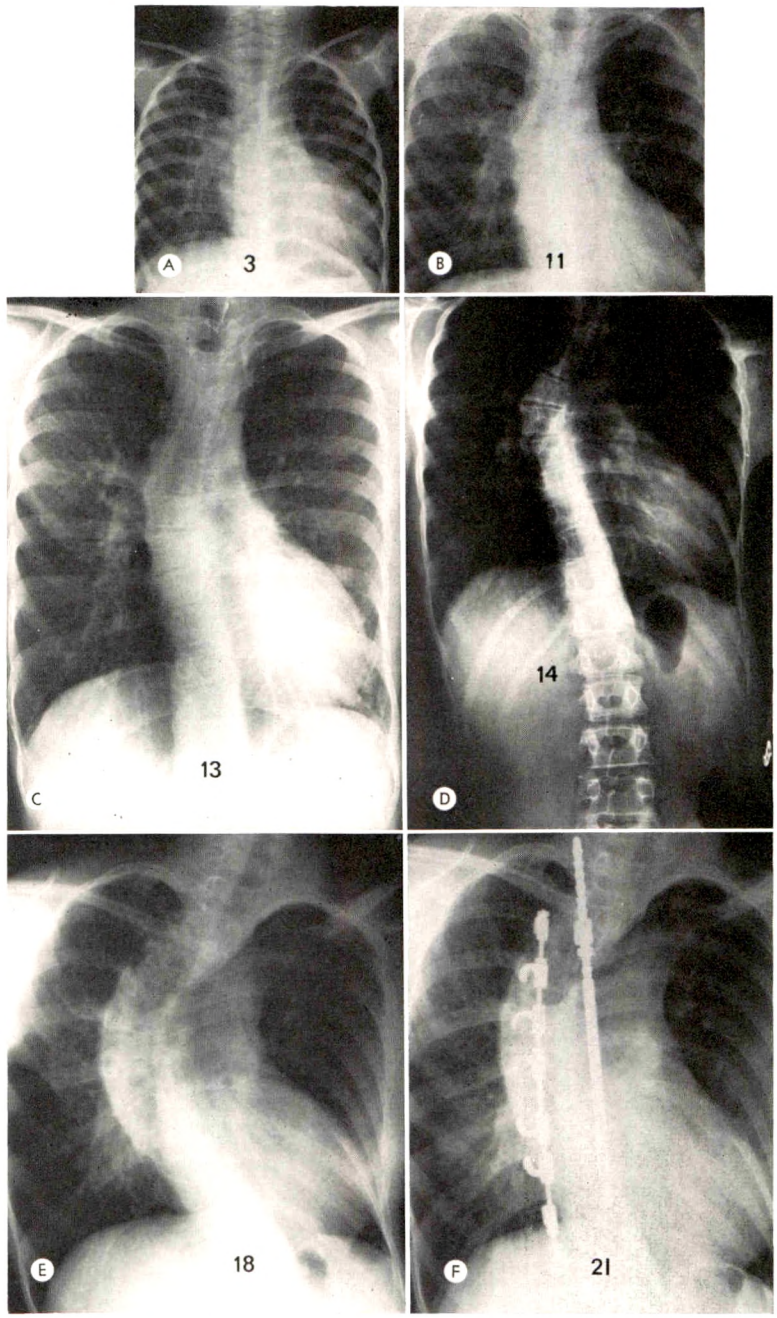


Fig. 4. (*A–F*) Serial chest and spinal roentgenograms from a patient with transposition of the great vessels and thoracic scoliosis without spinal anomaly. A beginning curvature, convex right, is noted at age 11 and the period of rapid progression occurs between ages 13 and 14. At age 18, the curvature was maximal measuring 76° and, following Harrington rod placement, the chest roentgenogram at age 21 shows some regression of the curvature.

penia," associated with CHD, appears to be a primary failure to develop cortical bone rather than a failure to replace once normal cortical bone as occurs in adult onset osteoporosis. The pathologic studies of Mariani and Bosman[15] have shown marrow hyperplasia and enlarged capillary vessels in the marrow spaces. It is of interest that they also described coarsened and thickened trabeculae and loss of finer

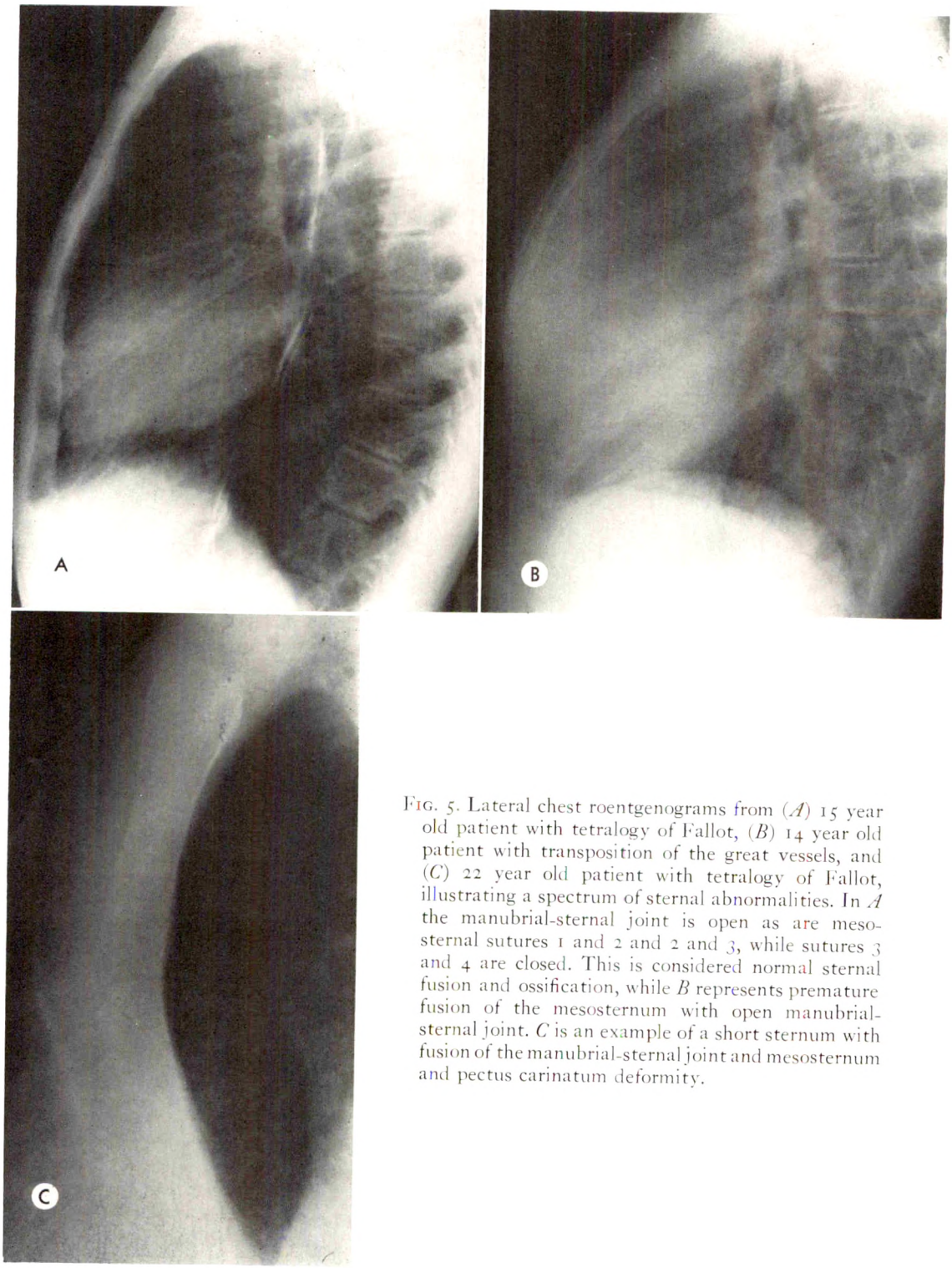


FIG. 5. Lateral chest roentgenograms from (*A*) 15 year old patient with tetralogy of Fallot, (*B*) 14 year old patient with transposition of the great vessels, and (*C*) 22 year old patient with tetralogy of Fallot, illustrating a spectrum of sternal abnormalities. In *A* the manubrial-sternal joint is open as are mesosternal sutures 1 and 2 and 2 and 3, while sutures 3 and 4 are closed. This is considered normal sternal fusion and ossification, while *B* represents premature fusion of the mesosternum with open manubrial-sternal joint. *C* is an example of a short sternum with fusion of the manubrial-sternal joint and mesosternum and pectus carinatum deformity.

trabeculae in the vertebra of patients with cyanotic CHD.

Classical parameters for evaluation of the severity of CHD include measurements of height, body weight and hemoglobin concentration.[1,4,13,16] Data from this study

Table II

COMPARISON OF THE SCOLIOSIS ASSOCIATED WITH CHD WITH IDIOPATHIC SCOLIOSIS

	Scoliosis CHD	Idiopathic Scoliosis
Incidence of Scoliosis Exceeding 10°	19%	1%
Male:Female Ratio	<1:2	>1:6
Onset	Early adolescence	Early adolescence
Rapid Progression	Early adolescence	Early adolescence
Stabilization	Late adolescence	Late adolescence

and our earlier work indicate that measurements of cortical bone can be substituted or at least can supplement measurements of height, weight and hemoglobin as indicators of the severity of CHD.[23] Retarded bone age also appeared to reflect severity of the heart disease as has been reported previously.[4,20]

As with the reduction in cortical bone formation, thoracic scoliosis was much more common and severe in patients with cyanotic CHD. In a previous study from this hospital, Luke and McDonnell[14] documented Doctor Helen Taussig's original observation of the increased frequency of scoliosis in CHD. A 2 per cent over-all incidence of scoliosis was found among all patients, infants through adults, attending the Johns Hopkins Pediatric Cardiac Clinic. Six per cent of the cyanotic and 0.8 per cent of acyanotic patients were affected. Our purpose in the current study was to define the natural history of the scoliosis associated with CHD including the time of its development, progression and stabilization. Deleting those patients with a bone anomaly of the thoracic spine, the over-all incidence of scoliosis was 44 per cent of the total group examined with 19 per cent having measurable curvatures (>10°). The reasons for the differences in the incidence of scoliosis between this study and the earlier one are twofold: (1) The average age of onset of the scoliosis was 11 years, so that any study examining all ages would obviously report a lower incidence. The earlier study was conducted among the entire clinic population, and therefore it is comprised of a proportionately greater number of younger patients. (2) The current study was prospective and included scoliosis less than 10°, provided it was present consistently on at least 3 previous chest roentgenograms.

Table II compares the features of the scoliosis associated with CHD with those of idiopathic scoliosis. Except for the greater over-all incidence and the more equal male: female ratios, the scoliosis of CHD resembles idiopathic scoliosis with regard to time of onset, rapid progression and stabilization of the curves. However, 4 of the patients had severe scoliosis, beginning on the average at 6 years of age. For this reason, any patient with CHD who develops scoliosis early in life should have a thorough evaluation of the spine. Awareness of the pulmonary disability caused by severe scoliosis and the surgery necessary to correct these severe curvatures should provide the impetus for the radiologist and pediatrician to evaluate the spine in addition to the heart on serial chest roentgenograms.

Our study documents the previously described association of CHD with premature fusion of the sternum.[6,8] As with reduced cortical bone formation and scoliosis, premature fusion of the sternum occurs more commonly in patients with cyanotic forms of CHD. Awareness of this association may prove helpful occasionally in differentiating cardiac from pulmonary causes of respiratory distress in infancy.[7]

Thus, it appears that there are significant effects of CHD upon the skeletal system. While the pathogenesis of these skeletal abnormalities is not known, knowledge of their existence and importance may lead to

better understanding of the patient with CHD.

SUMMARY

The association of reduced bone growth, retarded skeletal maturation, scoliosis, and premature fusion of the sternum with congenital heart disease was evaluated prospectively in 129 adolescent and young adult patients. Reduced cortical bone growth, retarded bone age and premature fusion of the sternum occurred frequently and were more common in cyanotic than in acyanotic patients. Thoracic scoliosis, beginning in adolescence, was also quite common and was most severe in the cyanotic patients.

It is hoped that the awareness of these skeletal abnormalities and their clinical importance will lead to improved management and understanding of the patient with congenital heart disease.

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REFERENCES

1. Adams, F. H., Lund, G. W., and Disenhause, R. B. Observations on physique and growth of children with congenital heart disease. *J. Pediat.*, 1954, *44*, 674–680.
2. Andrean, L., and Hall, P. Diminished segmentation or premature ossification of sternum in congenital heart disease. *Brit. Heart J.*, 1961, *23*, 140–142.
3. Ashley, G. T. Morphological and pathological significance of synostosis at manubrial-sternal joint. *Thorax*, 1954, *9*, 159–166.
4. Boyer, L. M., and Robinson, S. J. Growth history of children with congenital heart disease. *Arch. Dis. Childhood*, 1969, *117*, 564–572.
5. Cobb, J. R. Outline for study of scoliosis. *Am. Acad. Orthop. Surgeons*, 1948, *5*, 261–275.
6. Currarino, G., and Silverman, F. Premature obliteration of sternal sutures and pigeon-breast deformity. *Radiology*, 1958, *70*, 532–540.
7. Fischer, K., White, R. I., Jr., Jordan, C. E., Neill, C. A., and Dorst, J. P. Association of sternal abnormalities with congenital heart disease. In preparation.
8. Gabielsen, T. O., and Ladyman, G. H. Early closure of sternal sutures and congenital heart disease. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1963, *89*, 975–983.
9. Garn, S. M., and Wanger, B. The Adolescent Growth of the Skeletal Mass and Its Implications to Mineral Requirements from Adolescent Nutrition and Growth. Edited by F. P. Heald. Appleton-Century-Crofts, Inc., New York, 1969.
10. Garn, S. M. The Earlier Gain and Later Loss of Cortical Bone. Charles C Thomas, Publisher, Springfield, Ill., 1970.
11. Gruelich, W. W., and Pyle, S. T. Radiographic Atlas of Skeletal Development of the Hand and Wrist. Second edition. Stanford University Press, Stanford University, California, 1964.
12. Jordan, C. E., White, R. I., Jr., Fischer, K. C., Neill, C. A., and Dorst, J. P. Association of scoliosis with congenital heart disease. To be published.
13. Linde, L. M., Dunn, O. J., Schireson, R., and Rasof, B. Growth in children with congenital heart disease. *J. Pediat.*, 1967, *70*, 413–419.
14. Luke, M. J., and McDonnell, E. J. Congenital heart disease and scoliosis. *J. Pediat.*, 1968, *73*, 725–733.
15. Mariani, M., and Bosman, C. Über Skelettveränderungen bei kongenitalen Herzmissbildungen mit Zyanose. *Beitr. path. Anat.*, 1962, *126*, 145–167.
16. Mehrizi, A., and Drash, A. Growth disturbance in congenital heart disease. *J. Pediat.*, 1962, *61*, 418–429.
17. Mendlowitz, M., and Leslie, A. Experimental simulation in dog of cyanosis and hypertrophic osteoarthropathy which are associated with congenital heart disease. *Am. Heart J.*, 1942, *24*, 141–152.
18. Nice, C. M., Daves, M. D., and Wood, G. H. Changes in bone associated with congenital cardiac disease. *Am. Heart J.*, 1964, *68*, 25–31.
19. Paterson, A. M. The Sternum. Williams & Norgate, Ltd., London, 1904.
20. Ryba, W., Hedrall, G., and Carlgren, L. E. Skeletal age of children with congenital heart disease. *Acta pediat. scandinav.*, 1967, Suppl. 175, pp. 83.
21. Somerville, J. Gout in cyanotic congenital heart disease. *Brit. Heart J.*, 1961, *23*, 31–34.
22. Wahi, P. L., and Bawa, Y. S. Hypertrophic osteoarthropathy in cyanotic heart disease. *Indian J. Med. Sc.*, 1961, *15*, 453–455.
23. White, R. I., Jr., Jordan, C. E., Fischer, K. C., Dorst, J. P., Nagy, J. M., Garn, S. M., and Neill, C. A. Delayed skeletal growth and maturation in adolescent congenital heart disease. *Invest. Radiol.*, 1971, *6*, 21–27.
24. Williams, B., Ling, J. T., Leight, L., and McGaff, C. J. Patent ductus and osteoarthropathy. *A.M.A. Arch. Int. Med.*, 1963, *111*, 346–350.
25. Wright, W. D., and Niebauer, J. J. Congenital heart disease and scoliosis. *J. Bone & Joint Surg.*, 1956, *38-A*, 1131–1136.

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DERMATOGLYPHICS IN CONGENITAL HEART DISEASE*

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DERMATOGLYPHS are configurations of epidermal ridges in the volar aspects of the palms and soles. Scientific dermatoglyphic studies date to 1823 when Purkinje classified finger ridge patterns into 9 separate groups. The first practical application of this observation was fingerprinting for personal identification. During the first 2 decades of this century, the use of dermatoglyphics was extended from its place in criminology to anthropology, comparative zoology, and human genetics, principally through the efforts of Galton and his students.

Medical applications of dermatoglyphics probably began with the recognition of abnormal palmar creases in Down's syndrome. Penrose[9] initially reported the distal placement of the palmar axial triradius in Down's syndrome and later noted similar changes in a variety of chromosomal aberrations. More recently there have been reports of dermatoglyphic changes in other disorders; the literature has been thoroughly reviewed by Alter.[1]

Dermatoglyphic changes in congenital heart disease were reported by Hale *et al.*[5] and Sanchez-Cascos,[13] who noted distal placement of the axial triradius, reminiscent of patterns in patients with chromosomal disorders. In addition, Sanchez-Cascos[12] reported an excess of arch patterns in patients with pulmonic stenosis, increased frequency of ulnar loops with ventricular septal defects, and increased occurrence of whorls with tetralogy of Fallot and aortic coarctation, suggesting that dermatoglyphic analysis might play a role in the differential diagnosis of congenital heart disease.

This paper presents a pilot study of the hypothesis that particular dermatoglyphs are associated with specific cardiac anomalies.

MATERIAL AND METHOD

The control group consisted of 100 white males of western European ancestry who had no evidence of congenital or rheumatic heart disease; 60 per cent of the group was drawn from the medical and paramedical staffs of the Johns Hopkins Hospital and 40 per cent from patients on the Osler or Private Medical Services. Patients with coronary heart disease were not excluded.

The study group consisted of 52 white males of western European ancestry with congenital heart disease who were admitted to the Johns Hopkins Hospital or who attended either the adult or adolescent cardiac out-patient clinics during the months of December, 1965 and January, 1966. All diagnoses were confirmed at surgery or by cardiac catheterization.

Fingerprints and hypothenar patterns were recorded by the author after gross observation of the hands. In many instances permanent records were obtained by making ink blot impressions of the palms and fingers. Fingerprints were classified according to Galton's categories[2] (Fig. 1), namely, arch, loop, and whorl (simple, lateral pocket, and central pocket). Fingertip loops were subdivided into ulnar (single triradius on the ulnar side) and radial types. Hypothenar patterns were classified as loop, whorl, or "Van Gogh" configurations (Fig. 2), the last so named because of its resemblance to the central cloud formation in Van Gogh's *Starry Night* (Fig. 3). The central axis of the hypothenar loop was graded by assigning

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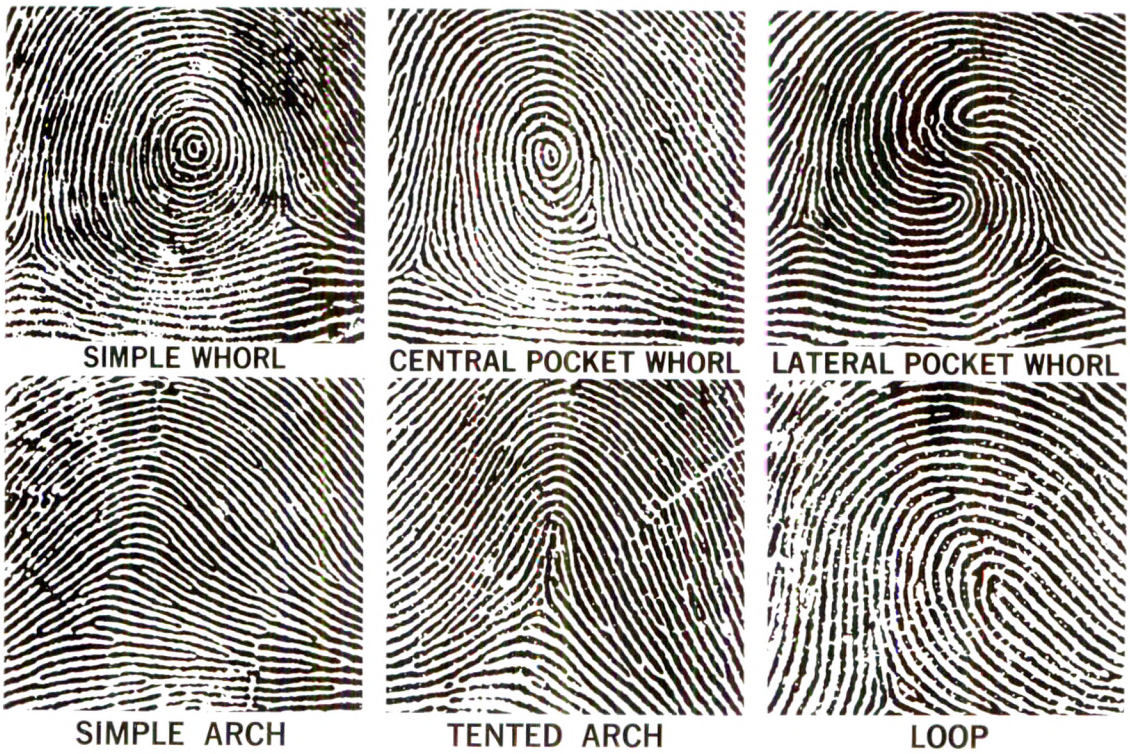


FIG. 1. Representative fingerprint patterns.

numbers from 10 to 50 to the fingers and their interspaces from the thumb to the small finger and extending the central axis until it intersected one of these numbered regions. A loop with an axis of 40 to 50 was scored separately as a "central"

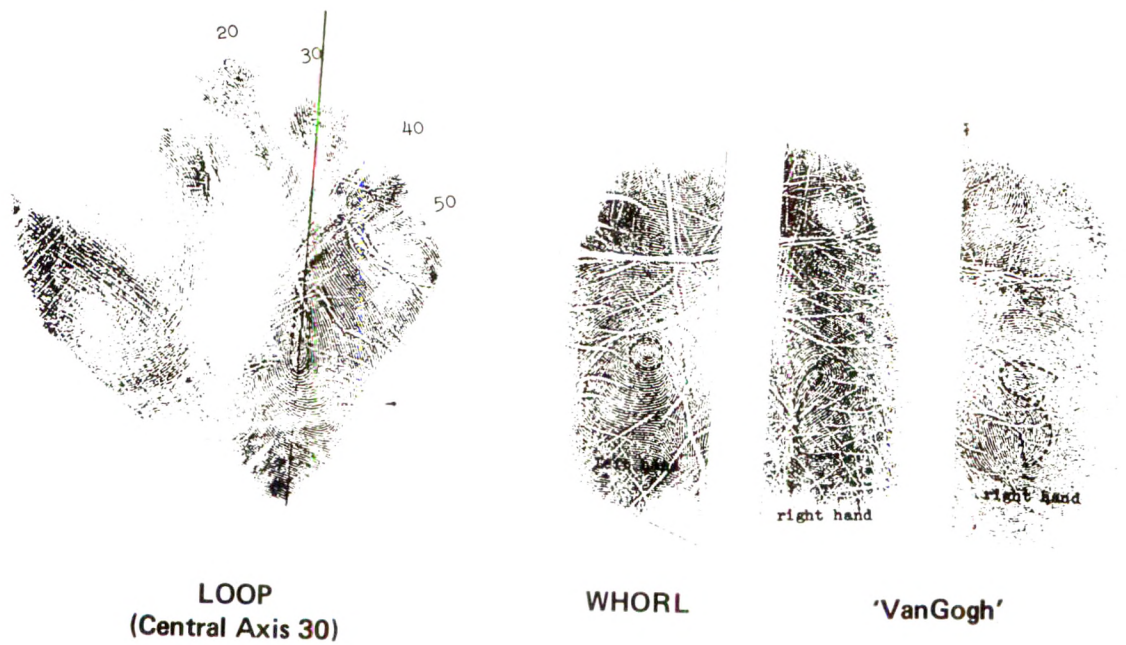


FIG. 2. Hypothenar patterns.



Fig. 3. The *Starry Night* of Vincent Van Gogh (courtesy of the Museum of Modern Art, New York City).

pattern. Examples of these patterns are shown in the illustrations.

A complete dermatoglyphic analysis was not performed. In particular, ridge counts, whorl type separation, palmar triradius location, and thenar region patterns were not considered, although this is essential in future studies.

RESULTS

The fingerprint and hypothenar patterns of the control group are presented in Table I, those of the congenital heart disease group subdivided by diagnosis, in Table II. The per cent frequencies of each fingerprint type per hand in both groups are given in Table III. For comparison with the control, the series of 2,000 adult British males "of the lower type artisan and labouring classes" collected by Waite[15] and Cummins and Midlo's[2*] series of 5,000 men and women processed by Scotland Yard in 1905 are included. Differences between control and comparison groups reflect the effects of ethnic and sexual factors on dermatoglyphic heterogeneity.

From Table III we see that the tetralogy of Fallot group has more whorls, the remainder of the congenital heart disease group fewer whorls per hand than the control population. Applying the chi-square contingency method to compare whorl and ulnar loop frequencies between either the control group or Waite's series and either tetralogy or other congenital heart disease groups, it is found that the apparent differences are significant in all instances at $p<.05$. This limited material corroborates

* Page 67.

TABLE I

FINGERTIP (12345) AND HYPOTHENAR (H) PATTERNS IN THE CONTROL GROUP OF 100 WHITE MALES. SYMBOLS W, A, U, R STAND FOR FINGERTIP WHORL, ARCH, ULNAR LOOP, AND RADIAL LOOP. W, C, VG STAND FOR HYPOTHENAR WHORL, "CENTRAL" AND "VAN GOGH" PATTERNS. NUMBERS 10, 15, 20, . . . ARE HYPOTHENAR LOOP AXES

Left		Right		Left		Right		Left		Right	
12345,	H	12345,	H	12345,	H	12345,	H	12345,	H	12345,	H
URUUU		URUUU		UWUWW		WWUWW		WAUUU	15	WUUUU	
UAUUU		UUUUU		UWUUU		UWUUU		UUUUU		UAUUU	
WUUUU		WWWWU		UAUUW		WUUWW		WWWWW		WWWWW	
UWUWU		UWUWU	10	WWWWU		WWUWU		WWWWU		WWWWW	
UUAUU		UAUWU		WWUUU		WRUUU		UUUUU		UUUUU	
UWUWU		WWUWU		WUUUU		WRUUU		AAUUU		UAUUU	
URAAU	15	WRUUU		WUUUU		WWWUU		UWUUU		WUUUU	
UUUWU		UWUWU		UAUUU		UAUUU	10	WRUUU		WRUUU	
UWWWW		UWWWW		UUUWU		UUUWU		UWUWU		WWUWW	10
UAUUU		UAAAU		UUUUU		WUUUU		WUUUU		WRUWU	
UUUUU		WUUWU		WWWWW		WWWWW		WWUUU	15	WRUUU	15
WWWWU		WWUWU		URUUU		URUUU		WRUUU		WRWUU	
UAAUU	15	UAAUU	VG	UUUUU		UUUUU		UAAUU		UAAUU	
WRUUU		WRUUU		URUUU	C	UAUUU	15	UWUWU		UWWWU	
UAUUU		WAUUU		URAUU		URAUU		UWWWU		RWWWU	
UWWWU	20	WWWWU	15	UUUUU		UAUUU		WWWWW		WWWWU	
WRUUU		WWUUU		WWUWW	20	WWWWW	20	UUAUU	10	URWWU	10
WUWWU		WWUWU	15	UUUUU		URUUU	W	UUAUU		UUUUU	
UAUUU		UAUWU		URWWU	10	UWUUU	10	UUWWU		WWUWU	
UUUUU	15	URUUU		URAWU		UWUUU		WWWWU		WWUWU	10
URUUU		UAUUU		AWAWU		ARRWU		UUUUU		UUUWU	
WAUWU		WAUWU		UWWWU		UWWWU		UAAAA		UUUUU	
WWUWU	15	WWUWU	15	UUAWU		URUUU		UWWWW		WWWWW	
UAAUU		UAUUU		URAUU	C	UAAUU	C	UAAWU		UAUUU	
WRUUU		WWUWU		URUUU		URUUU		UAAWU		UAUUU	
UAUUU		URUUU		WUUUU		WRUWU		WUUUU		WWUUU	10
UAUWU	VG	UAUUU	VG	WWWWW		WWWWW		WWWWU		WWWWW	10
UWUWU		WRUWU		WWWWU		WWWWW		URWUU	10	UWRUU	C
UAUUU		UAUWU	15	WAUUU		UWUWU		WWUWU	20	WWWWW	20
UAAUU		UAUUU		AAUUU		AAUWU		WUUUU	10	WRURU	
WWWWU		WWWWW		WWUWW	10	WUUWU	10	UWUWU		WWUWU	
UUUUU		UUUUU		URUUU		UAUUU		WWUUU		URUUU	
UWUWU		UWWWW		UAUWU		UAUWU					

the whorl data of Sanchez-Cascos.[13]

Table IVA lists the occurrences of hypothenar patterns. It can be seen that there is a .22 (22 per cent) incidence of right hypothenar patterns amongst controls, but the incidence in the tetralogy of Fallot group is .50. The frequency of occurrence per hand is shown in Table IVB. While there is no difference between control and tetralogy of Fallot groups as regards combined right and left hands or left alone, a right hypothenar pattern alone is nearly 4 times more common with tetralogy of Fallot. Restricting attention to the right hand and accepting the control incidence of 22 per cent (which compares well with incidences reported in large populations of presumably healthy males by Cummins and Midlo[21†]), the chance of observing 10 hypothenar patterns in a random selection of 20 patients is only .004. This implies that the group of 20 patients with tetralogy

† Page 115.

of Fallot comprises a significantly different dermatoglyphic population with reference to the hypothenar region than the control group. In addition, it appears as if the mean axis of right hypothenar loops in the 2 groups may be different, but a larger series will be necessary to evaluate this feature. Hypothenar whorls are generally rare in "healthy" populations, but occurred twice in 20 patients with tetralogy of Fallot. This finding may also prove to be significant.

Examination of Table 1 reveals that 24 of 100 controls have mirror image symmetry. Three of 20 tetralogies, 2 of 10 ventricular septal defects, and 5 of 22

TABLE II

FINGERTIP AND HYPOTHENAR PATTERNS IN PATIENTS WITH CONGENITAL CARDIAC DEFECTS; SYMBOLS AS IN TABLE I

	Left		Right		Left		Right	
	12345,	H	12345,	H	12345,	H	12345,	H
Tetralogy of Fallot (20 males)								
	WRUWU		UAURU		UWWWW	VG	UWUWW	VG
	UUUUU		URUUU	30	UWWWU	15	WWUWU	15
	UUUUU		UAUUU		UAWAU		WUUUU	
	UUWWU	10	WWUWU	10	URUUU	20	WRUUU	
	WWUWU		WWUWU		WAUWU		WAUWU	20
	UAAAU		UAAAU		UWUUW		UWWWW	C
	UAAUU		UAUUU		UAUWU		WAUWU	20
	WUWWW		WWWWW		UUUUU		UAUUU	25
	WWWWW		UWWWU		WWWWW		WWWWU	W
	UUUWU		UWUWU	W	WWUWU		WWUWW	
Ventricular Septal Defect (10 males)								
	UUUUU		UUUUU		UUUUU		UAUUU	10
	UUUWU		UUUUU		UUUWU		UUUWW	
	WAUUU		UUUUU		UAAUU		UAAAU	
	URUUU	15	UAUWU		UAUUU		UAAUU	
	UUUUU		UUUUU		URAUU	15	URUUU	15
Other Anomalies (22 males)								
aortic stenosis	URUUU		UUUUU		URUWU		URUUU	
aortic stenosis	UAUUU		UAUUU	15				
pulmonic stenosis	UUUUU		UURUU		AAAUU		URUUU	
pulmonic stenosis+sinus venosus	UUUUU	15	URUUU	15	WWWWW		UWUUU	
sinus venosus defect	WUUWW		WUUWW					
transposition	UWWWW		WWWWU		UWUUU		UWUWU	
transposition	WUUUU	10	WWWWW	10				
transposition+pulmonic stenosis	WUUWU		WWUWU	15				
transposition+tricuspid atresia	UUUUU		URUUU					
pseudotruncus	UUUUU		URUUU	W				
atrial septal defect	WWUWW		WWWWW		UAAAU		UAAAA	
atrial septal defect+ pulmonic stenosis	UAAUU		UAAUU		AAAAU		AAAAU	
ostium primum defect	UWUUU		UAURU					
patent ductus arteriosus	UWWUU		UWWWU		WWWWW		WWWWW	
tricuspid atresia	AWWWU		ARWWU					

TABLE III

PER CENT FREQUENCY OF OCCURRENCE OF FINGERPRINT PATTERNS BY HAND

	Cummins and Midlo[2] (5,000 men and women)	Waite[15] (2,000 British males)	Controls (100 white males)	Tetralogy of Fallot (20 white males)	Congenital heart disease excluding tetralogy (32 white males)
Whorl					
right hand	28.7	30.9	35.0	40	22
left hand	22.1	23.9	29.8	38	22
right and left hands	25.4	27.9	32.4	39	22
Arch					
right hand	4.4	7.0	6.3	9	12
left hand	5.4	7.2	8.6	9	12
right and left hands	4.9	7.1	7.4	9	12
Radial Loop					
right hand	6.0	5.2	5.0	3	5
left hand	5.3	4.3	3.4	2	2
right and left hands	5.7	4.7	4.2	2.5	3.5
Ulnar Loop					
right hand	60.8	56.9	53.8	48	60
left hand	67.2	64.6	58.2	51	64
right and left hands	64.0	60.8	56.0	50	62

other congenital heart defects are also entirely symmetric. Again, a larger series will be necessary to evaluate this feature. Analysis of individual couplet symmetry may be a more delicate measure of the tendency towards mirror image symmetry. Waite[15] analyzed associations between various finger couplets by means of con-

TABLE IV

HYPOTHENAR REGION PATTERNS

A. HYPOTHENAR PATTERN FREQUENCIES: SYMBOLS AS IN TABLE I

		Patterns				Loop Axis					
	Total	VG	W	C	Lp	10	15	20	25	30	mean
Control group											
N = 100											
right	22(22%)	2	1	2	17	9	6	2	—	—	13
left	18(18%)	2	0	2	14	5	6	3	—	—	14
Tetralogy of Fallot group											
N = 20											
right	10(50%)	1	2	1	6	1	1	2	1	1	20
left	4(20%)	1	0	0	3	1	1	1	—	—	15

B. HYPOTHENAR REGION PATTERNS BY HAND

left	right	*Control group* N = 100	*Tetralogy of Fallot group* N = 20
yes	yes	.13	.15
yes	no	.05	.05
no	yes	.09	.35
no	no	.73	.45

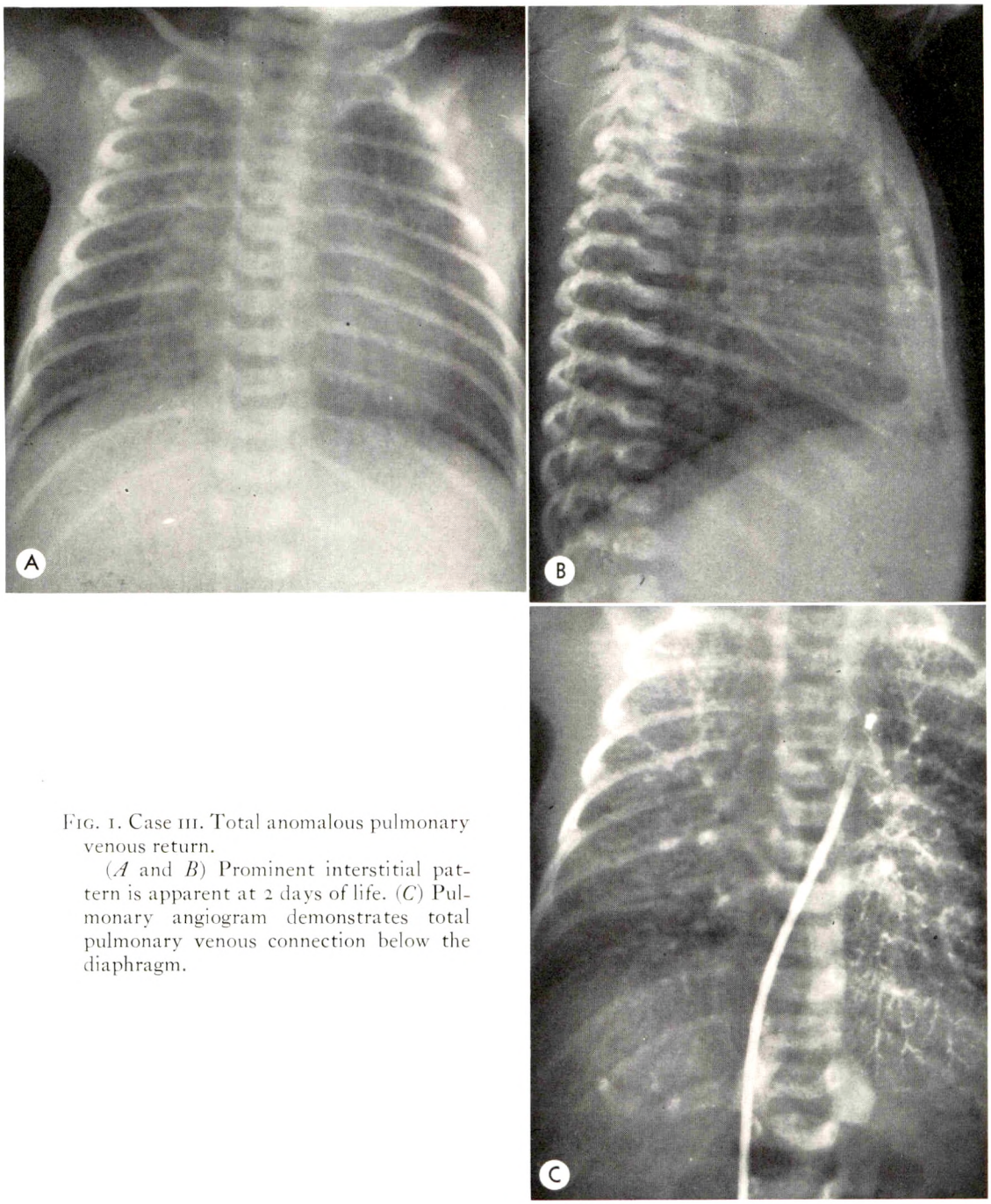


FIG. 1. Case III. Total anomalous pulmonary venous return.

(*A* and *B*) Prominent interstitial pattern is apparent at 2 days of life. (*C*) Pulmonary angiogram demonstrates total pulmonary venous connection below the diaphragm.

lymphangiectasia have been reported to date in the world literature[2–4,6–14,16,18–30] and one of the authors has seen another unreported case.[5] Approximately $\frac{1}{3}$ of the reported cases of pulmonary lymphangiectasia were found in patients with cardiac anomalies associated with pulmonary venous obstruction (PVO).[3,10,13,14,23–25,27] Seven of our 17 patients had congenital heart disease with varying degrees of PVO, whereas no cardiac abnormalities were found in the remaining 10.

TABLE IV

PROPOSED CLASSIFICATION OF PULMONARY LYMPHANGIECTASIA

	Cardiac (appr. $\frac{1}{3}$)	Noncardiac (appr. $\frac{2}{3}$)	
		Early	Late
Onset	Early (hours—days)	First 24 hours of life	Early or may be delayed for days, weeks, months
Gestation	Term > Premature	Term > premature	Insufficient data
Mortality	High	High	Variable
Duration of Life	Days—Months	Usually die in less than 24 hours	May live weeks, months, years
Roentgenographic Findings	Heart—variable size and configuration—depends on underlying cardiac malformation Lungs—pulmonary venous obstruction (PVO), atelectasis, pneumonia, normal in some	Lungs—(1) may show nonspecific changes; increased interstitial markings, nodular pattern, etc. (2) may be normal	Lungs—normal early; pneumonia, atelectasis later; prominent lymphatic pattern in some cases
Etiology	(1) Most are associated with PVO (2) Some may be primary abnormality of lymphatic development	Congenital abnormality of lymphatic development at 12–20 weeks of gestation	Uncertain; possibly a congenital malformation of lymphatics; possibly acquired in some cases

The following classification is based upon the reported cases as well as the 17 patients in our series.

CLASSIFICATION

I. CARDIAC-ASSOCIATED PULMONARY LYMPHANGIECTASIA

Pulmonary lymphangiectasia associated with obstructed pulmonary venous return was first reported by Shortland-Webb and colleagues.[25] In their patient, congenital pulmonary lymphangiectasia was associated with complex congenital heart disease in which a small left atrium received 4 normally draining pulmonary veins, but did not communicate with either ventricle.

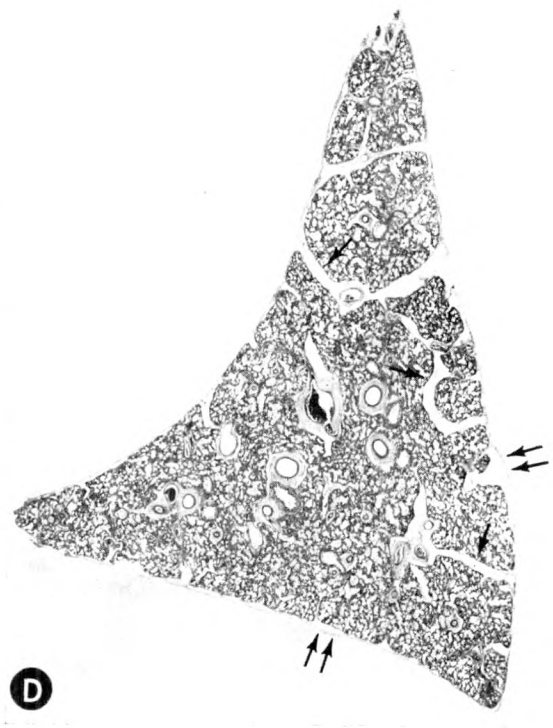


FIG. 1. (*D*) Moderate dilatation of the septal (single arrows) and subpleural (double arrows) lymphatics is apparent on histologic whole mount of the lung.

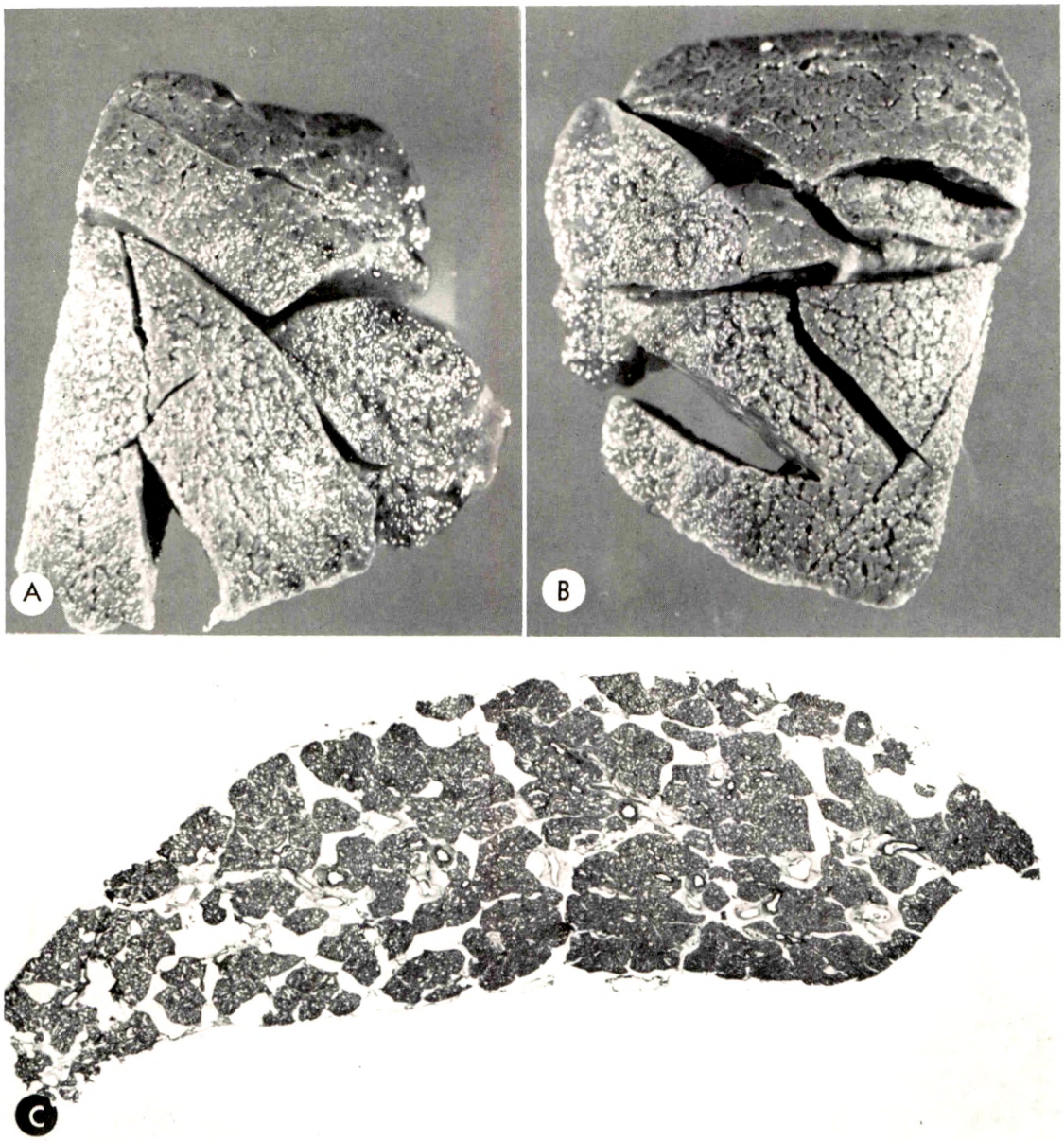


Fig. 2. Case VI. This child died at 1 day of life and autopsy revealed complex congenital heart disease including TAPVR (Table 1).

(*A* and *B*) Diffuse dilatation of subpleural lymphatics is apparent on the external surface of the gross pathologic specimen. (*C*) Histologic whole mount demonstrates severe, diffuse dilatation of the subpleural and septal lymphatics.

Simple, less prominent dilatation of pulmonary lymphatics, moreover, is commonly seen in acquired heart disease in which there is elevated pulmonary venous pressure, notably mitral stenosis.

A review of the autopsy findings at the University of Florida Hospital in 12 patients with cardiac lesions and PVO disclosed pulmonary lymphangiectasia to be severe in 2, moderate in 5, and minimal in

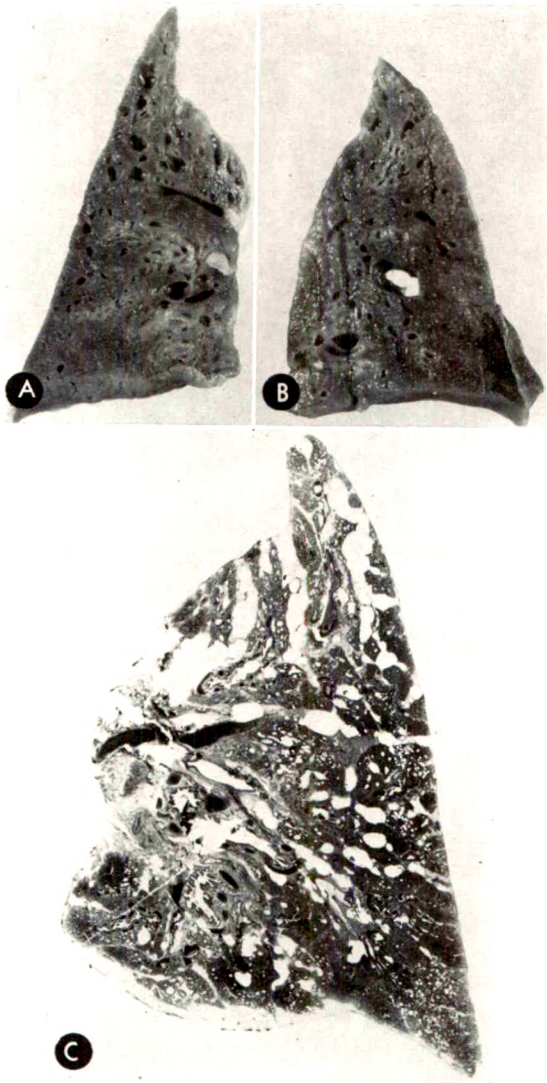


FIG. 3. Case X. This infant died at 1½ hours of life, never having established normal respirations. No heart disease was found.

(*A* and *B*) Severe, diffuse cystic lymphangiectasia is present. Some hemorrhage has occurred into the lymphatic spaces. (*C*) Histologic whole mount confirms the lymphatic dilatation throughout the parenchyma with areas of hemorrhage and atelectasis.

5 (Table III). The 7 patients (Cases I–VII) with moderate or severe involvement are included in our series. Fourteen additional patients with pulmonary lymphangiectasia associated with congenital heart lesions causing PVO have been reported.[3 10 13,14,20,23–25,27]

This total of 21 patients represents approximately ⅓ of the 62 patients with pulmonary lymphangiectasia reported in the world literature.

It seems reasonable to separate pulmonary lymphangiectasia associated with congenital heart disease and PVO from that variety which occurs without this form of cardiac disease. Hemodynamic factors, operating to keep pulmonary lymphatics dilated, may play a role in the pathogenesis of pulmonary lymphangiectasia in the former group rather than deranged embryogenesis of pulmonary lymphatics.

In 2 of our patients with congenital heart disease and PVO (Cases VI and VII), the pulmonary abnormality was severe and extensive and the cystic, dilated lymphatic spaces were quite large. The possibility exists in these 2 patients, that the lung changes resulted from primary developmental abnormality of lymphatics rather than from obstructed pulmonary lymphatics alone. Nevertheless, we have classified these 2 patients in the cardiac-associated group.

The roentgenographic findings in patients with cardiac-associated pulmonary lymphangiectasia are variable. Obvious pulmonary venous obstruction was evident on chest roentgenograms of 2 patients, while the lungs were clear in 2 others. Diffuse infiltrates representing areas of pneumonia and/or atelectasis were seen in the remaining 3.

II. NONCARDIAC-ASSOCIATED PULMONARY LYMPHANGIECTASIA

A. *Early onset.* Seven patients (Cases VIII–XIV) whom we have designated in a subgroup as "early," developed respiratory symptoms shortly after birth and all died within the first 24 hours. At autopsy, the lungs of these patients demonstrated severe, diffuse cystic lymphangiectasia. This clinical pattern of early onset of disease with rapid demise is characteristic of the majority of patients reported in the literature as congenital cystic lymphangiectasia and one may assume that their lung disease resulted from the abnormality

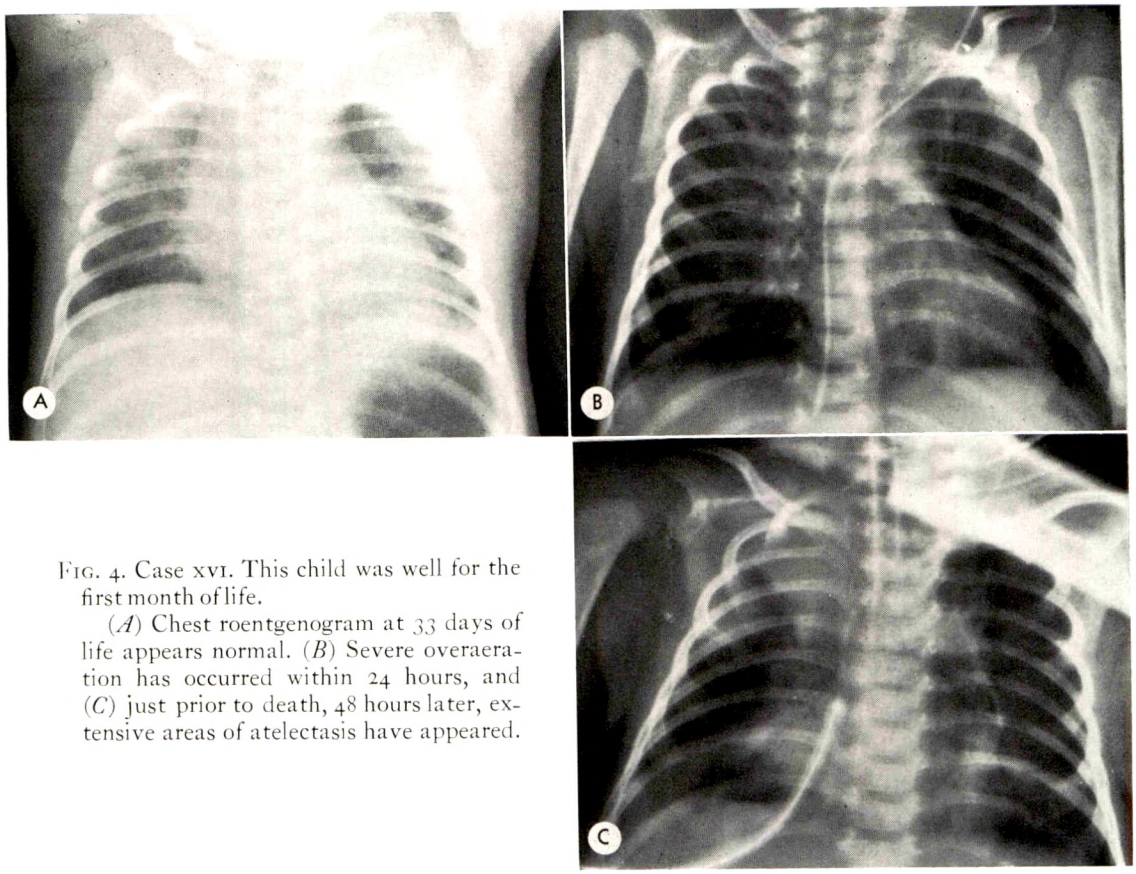


Fig. 4. Case xvi. This child was well for the first month of life.

(*A*) Chest roentgenogram at 33 days of life appears normal. (*B*) Severe overaeration has occurred within 24 hours, and (*C*) just prior to death, 48 hours later, extensive areas of atelectasis have appeared.

of embryogenesis described by Laurence.[13]

B. *Late onset.* Three of our patients (Cases xv–xvii) enjoyed periods of relatively normal life and had normal chest roentgenograms before the onset of pulmonary disease. Two lived for a month and 1 for 2 months. Cystic fibrosis and meconium ileus complicated the course of 1 (Case xvii), but there were no extrapulmonary abnormalities in the other 2 (Cases xv and xvi). When respiratory disease developed, the roentgenograms manifested those changes characteristic of diffuse infection and atelectasis and did not show dilated interlobular lymphatics.

Increasing numbers of patients with pulmonary lymphangiectasia are being discovered in whom respiratory symptoms begin after variable intervals of normal life. Some of these patients have lived for weeks or months and others into childhood.[5,12,14,16,18,20,27,29] It would, therefore, appear that a group of patients with pulmonary lymphangiectasia is now being recognized in whom the onset of disease may be delayed beyond the first few days of life. In some of these patients, the underlying abnormality may be suspected from the chest roentgenograms which demonstrate prominent interstitial lymphatics and Kerley lines. In others, the chest roentgenogram may show only those changes of acute or chronic pneumonia, overexpansion, or atelectasis. In these, the diagnosis can only be established histologically.

Whether pulmonary lymphangiectasia in these patients has the same pathogenesis as that seen in patients who die early cannot be stated with certainty. It is apparent, however, that the abnormality can no longer be considered only as invariably fatal. In all probability, a variety of forms exist which differ in mode of onset, clinical expression and roentgen features. In those

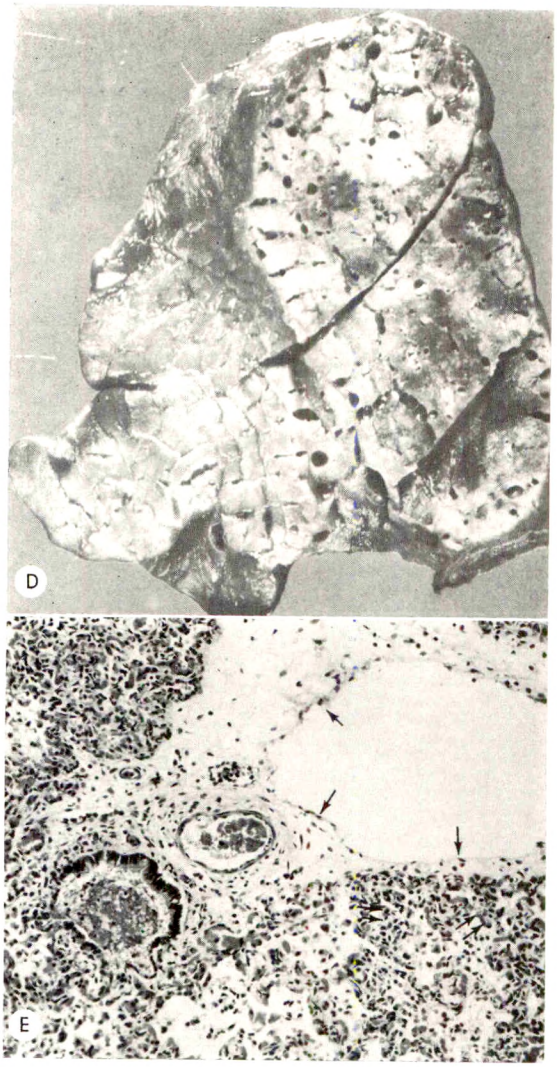


FIG. 4. (*D*) The gross specimen demonstrates severe, diffuse cystic lymphangiectasia and areas of lobular pneumonia. (*E*) Histologic preparation demonstrates dilated, endothelial lined lymphatics (single arrows) and early lobular pneumonia (double arrows) (×400).

patients in whom the onset of clinical symptoms is delayed or who live longer than the expected time, the possibility exists that the lymphangiectasia may be an acquired abnormality.

CONCLUSIONS

We have added 17 examples of pulmonary lymphangiectasia to 45 patients reported in the literature and 1 known to us by personal communication. We are in agreement with Shortland-Webb and colleagues[25] as well as others,[20,26] regarding the probable association between some forms of pulmonary lymphangiectasia and obstructed pulmonary venous return. In 5 of 12 patients in our hospital who died with cardiac lesions producing obstructed pulmonary venous return, moderate pulmonary lymphangiectasia was found.

In an additional 2 patients with obstructed pulmonary venous return, the degree of cystic lymphangiectasia was quite severe and more than might be expected only from PVO. A definitive etiology for the lymphangiectasia cannot be stated with certainty in these 2 patients, although the severity of involvement and similarity of the pathologic changes to those with noncardiac variety suggest that abnormal embryologic development of the pulmonary lymphatics might have accounted for the changes.

In the remaining 10 patients, no associated cardiac lesions were found, although other congenital abnormalities were frequent. Seven of these 10 patients fit the clinical and pathologic description of congenital cystic pulmonary lymphangiectasia by virtue of early onset of symptoms, rapid demise and characteristic histopathologic appearance of the lungs at autopsy. The remaining 3 patients, in whom respiratory symptoms began after periods of relative well-being and in whom severe and overwhelming infection was superimposed upon the abnormal lymphatics, may represent a variation or different expression of this disease. With the introduction of pulmonary infection, the abnormal lungs may not be able to respond in a normal way, resulting in overwhelming pneumonia and death, while in others chronic lung disease may ensue. We cannot exclude the possibility that, in some instances, the dilated, tortuous pulmonary lymphatics were acquired postnatally as a result of infection, obstruction, or some other unknown factor.

For the purposes of this report, we have not included other abnormalities of

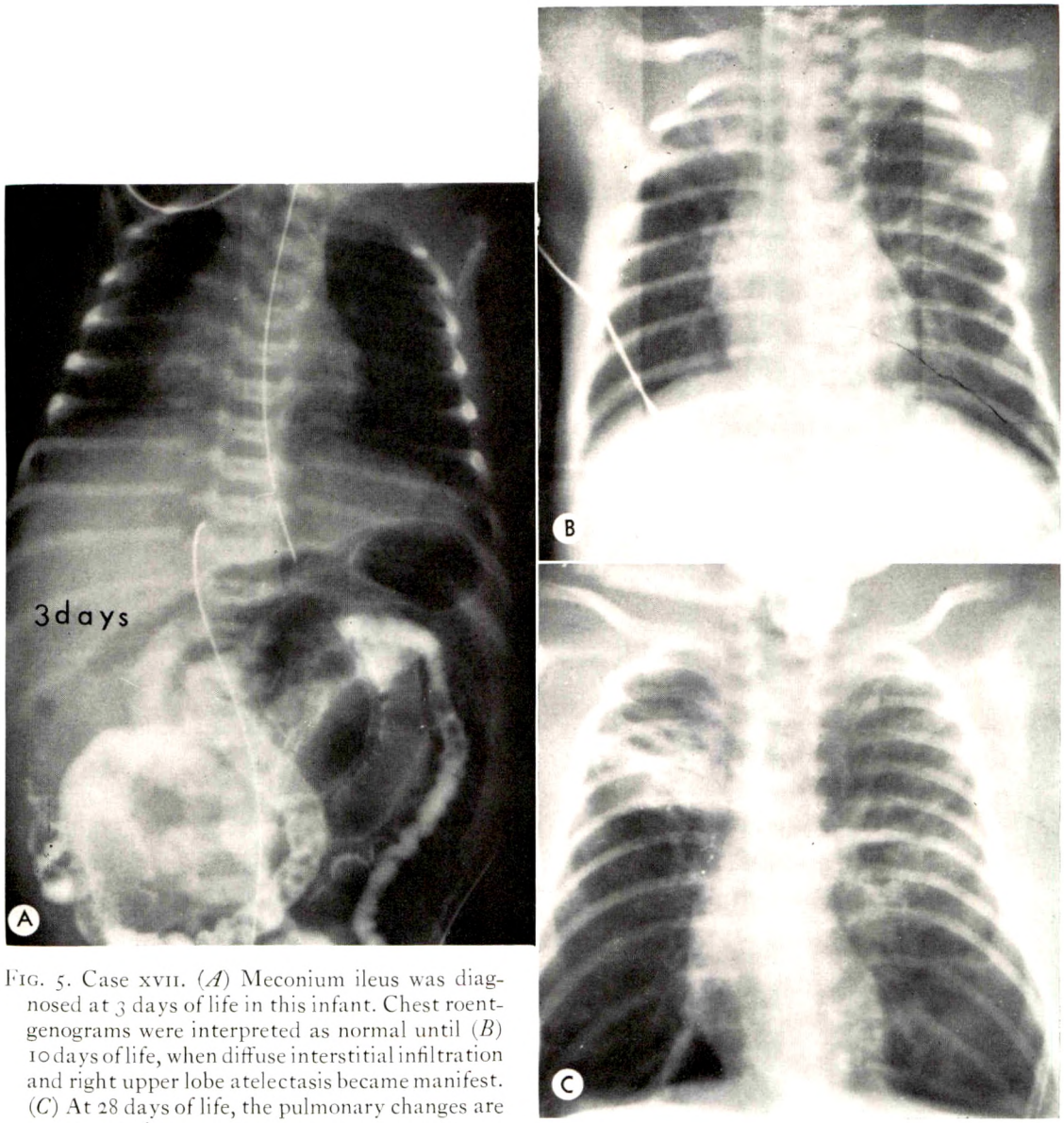


FIG. 5. Case XVII. (*A*) Meconium ileus was diagnosed at 3 days of life in this infant. Chest roentgenograms were interpreted as normal until (*B*) 10 days of life, when diffuse interstitial infiltration and right upper lobe atelectasis became manifest. (*C*) At 28 days of life, the pulmonary changes are more extensive.

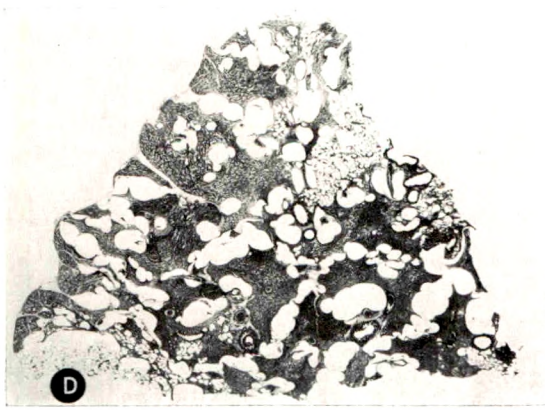


FIG. 5. (*D*) Histologic whole mount of the lung demonstrates severe, diffuse cystic lymphangiectasia as well as extensive pneumonia.

lymphatics which occur in the newborn. The reader is referred to other excellent articles which cover this subject.[1,15,17]

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REFERENCES

1. ALLEN, R. P., TAYLOR, R. L., and REIQUAM, C. W. Congenital lobar emphysema with dilated septal lymphatics. *Radiology*, 1966, *86*, 929–931.
2. ARKOFF, R. S. Congenital pulmonary lymphangiectasis. *California Med.*, 1968, *109*, 464–466.
3. BREDT, cited by Laurence, K. M.[13]
4. BROWN, M. D., and REIDBORD, H. E. Congenital pulmonary lymphangiectasis. *A.M.A. Am. J. Dis. Child.*, 1967, *114*, 654–657.
5. CAPITANIO, M. A., and KIRKPATRICK, J. A., JR. Personal communication.
6. CARTER, R. W., and VAUGHN, H. M. Congenital pulmonary lymphangiectasis; report of case with roentgen findings. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1961, *86*, 576–578.
7. EKELUND, H., PALMSTIERNA, S., and OSTBERG, G. Congenital pulmonary lymphangiectasis. *Acta paediat. scandinav.*, 1966, *55*, 121–125.
8. FRANK, J., and PIPER, P. G. Congenital pulmonary cystic lymphangiectasis. *J.A.M.A.*, 1959, *171*, 1094–1098.
9. FRONSTIN, M. H., HOOPER, G. S., BESSE, B. E., and FERRERI, S. Congenital pulmonary cystic lymphangiectasia; case report and review of 32 cases. *A.M.A. Am. J. Dis. Child.*, 1967, *114*, 330–335.
10. GIAMMALVO, J. T. Congenital lymphangiomatosis of lung: form of cystic disease; report of case with autopsy findings. *Lab. Invest.*, 1955, *4*, 450–456.
11. GIEDION, A., MÜLLER, W. A., and MOLZ, G. Angeborene Lymphangiectasie der Lungen; eine radiologisch erkennbare Ursache des Atemnotsyndroms beim Neugeborenen. *Helvet. paediat. acta*, 1967, *2*, 170–180.
12. JAVETT, S. N., WEBSTER, I., and BRAUDO, J. L. Congenital dilatation of lymphatics. *Pediatrics*, 1963, *31*, 416–425.
13. LAURENCE, K. M. Congenital pulmonary cystic lymphangiectasis. *J. Path. & Bact.*, 1955, *70*, 325–333.
14. LAURENCE, K. M. Congenital pulmonary lymphangiectasis. *J. Clin. Path.*, 1959, *12*, 62–69.
15. LAUWERYNS, J. M., and BOUSSAUW, L. Pulmonary lymphatics in neonatal hyaline membrane disease. *Pediatrics*, 1968, *41*, 917–930.
16. MAIDMAN, L., and BARNETT, R. N. Congenital dilatation of pulmonary lymphatics. *A.M.A. Arch. Path.*, 1957, *64*, 104–106.
17. MCKENDRY, J. B. J., LINDSAY, W. K., and GERSTEIN, M. C. Congenital defects of lymphatics in infancy. *Pediatrics*, 1957, *19*, 21–35.
18. MEADOWS, J. A. Two cases of rare causes of respiratory distress in newborn: 1. Congenital pulmonary lymphangiectasis. 2. Wilson-Mikity syndrome. *J. M. A. Alabama*, 1968, *37*, 1039–1044.
19. MOFFAT, A. D. Congenital cystic disease of lungs and its classification. *J. Path. & Bact.*, 1960, *79*, 361–372.
20. NOONAN, J. A., WALTERS, L. R., and REEVES, J. T. Congenital pulmonary lymphangiectasis. *A.M.A. Am. J. Dis. Child.*, 1970, *120*, 314–319.
21. O'HARA, A. E., and LIBSHITZ, H. I. Congenital pulmonary lymphangiectasis. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1968, *103*, 119–122.
22. RHATIGAN, R. M., and HOBIN, F. P. Congenital pulmonary lymphangiectasis and ichthyosis congenita; case report. *Am. J. Clin. Path.*, 1970, *53*, 95–99.
23. RYWLIN, A. M., and FOJACO, R. M. Congenital pulmonary lymphangiectasis associated with blind common pulmonary vein. *Pediatrics*, 1968, *41*, 931–934.
24. SACREZ, R. *et al.* Lymphangiectasies pulmonaires congénitales avec fibroelastose de l'endocarde. *Arch. franç. pédiat.*, 1960, *17*, 1012–1023.
25. SHORTLAND-WEBB, W. R., TOZER, R. A., and CAMERON, A. H. Intra-uterine closure of atrial septum. *J. Clin. Path.*, 1966, *19*, 549–554.
26. SINGLETON, E. B., and WAGNER, M. L. Radiographic Atlas of Pulmonary Abnormalities in Children. W. B. Saunders Company, Philadelphia, 1971, pp. 52 and 228.
27. TAN-VINH, L., COCHARD, A., and DONG, V. T. Lymphangiectasies pulmonaires congénitales et lymphangite pleuro-pulmonaire, cancereuse metastatique de l'enfant. *Arch. franç. pédiat.*, 1964, *21*, 165–182.
28. THEROS, E. G. Exercise in radiologic-pathologic correlation. *Radiology*, 1967, *89*, 524–531.
29. TUCKER, A. S. Lymphangiectasis—benign and malignant. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1964, *91*, 1104–1113.
30. VIRCHOW, R., cited by Laurence, K. M.[13]

Vol. 116, No. 3

BRONCHOGRAPHY IN CHILDHOOD ASTHMA*

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DESPITE dietary and environmental control measures and hyposensitization, many asthmatic children continue to have recurrent, severe wheezing attacks. These attacks often seem to be related to antecedent or co-existing pulmonary infections. Many of these children show chronic changes of peribronchial thickening and hyperaeration. Some demonstrate persistent focal abnormalities on serial chest roentgenograms. With the knowledge that asthmatic conditions may improve after resection of localized bronchiectasis,[2] many physicians request bronchography in an attempt to identify an underlying focal area of pulmonary inflammatory disease.

This report describes our experience and observations on the value of bronchography in childhood asthma.

MATERIAL AND METHOD

Seventy-one right, left or bilateral bronchograms were obtained on 57 asthmatic children ranging in age from 10 months to 13 years.

Most bronchograms were obtained under general anesthesia with halothane mixed with nitrous oxide and oxygen. A few earlier studies employed ether anesthesia and a few older children were studied under local anesthetic techniques. Although most studies were performed with an oily contrast agent (dionosil), aqueous agents such as hytrast, aqueous dionosil and barium suspension (broncobar) were used in 40 per cent of the examinations. All of the patients were problems in regard to recurrent co-existing pulmonary inflammatory disease. No study was performed during, or shortly after, an acute respiratory tract infection. Steroids and atropine sulfate were usually administered prior to bronchography.

Preliminary roentgenograms were evaluated for hyperaeration, peribronchial cuffing, chronic focal abnormalities and collapse. Bronchographic studies were evaluated for static luminal abnormalities and filling defects.

RESULTS

Perhaps because of critical patient selection, all showed some sign of chronic bronchitis on plain roentgenograms and at bronchography. We have arbitrarily divided the bronchographic findings into 3 groups (Table 1). The first group includes 5 of the more common findings of chronic bronchitis in childhood asthma, seen in 35 to 93 per cent of the patients. Although more common in occurrence, they were less specific. For example, diffuse spasm

TABLE I

BRONCHOGRAPHIC FINDINGS OF CHRONIC BRONCHITIS IN 57 CHILDREN WITH ASTHMA AND RECURRENT CO-EXISTING PULMONARY INFLAMMATORY DISEASE

Bronchographic Findings	Per Cent
1. Spasm, truncular or diffuse	93
2. Retained mucus secretions	88
3. Irregular bronchial dilatation	79
4. Mucus plugging	37
5. Mucosal irregularities	35
Less Common Bronchographic Signs	
6. Patchy bronchiolar filling	19
7. Dilated mucous gland duct filling	12
Rare Bronchographic Signs	
8. Bronchial mucosal atrophy	2
9. Bronchiolectasis	2

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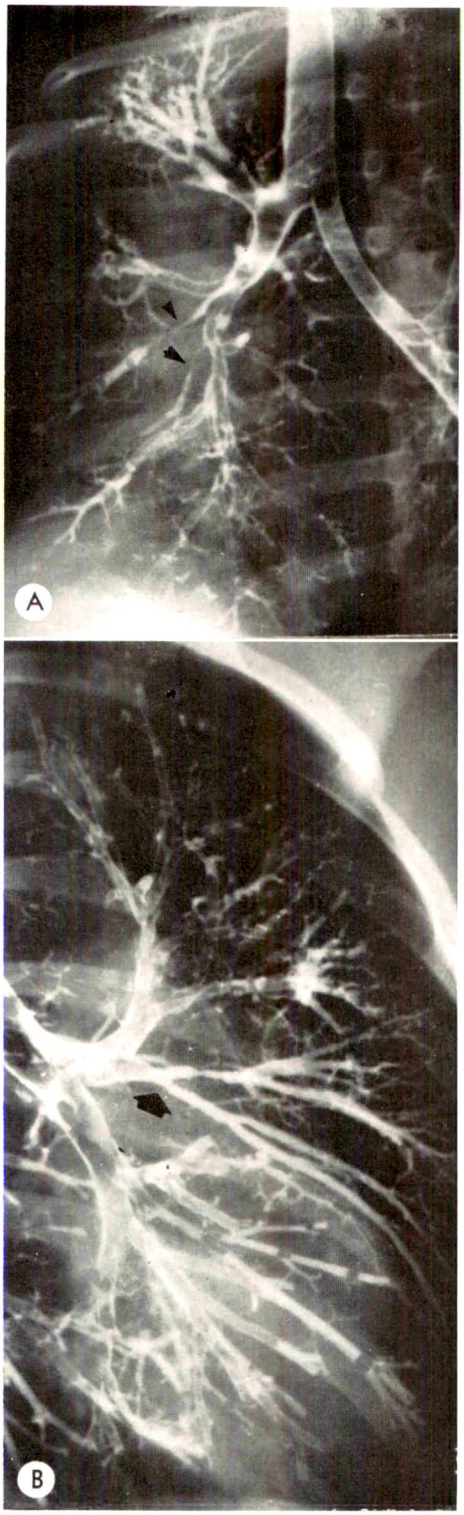


FIG. 1. (*A*) Localized spasm of peripheral segmental bronchi (small arrows) in a 13 year old male. (*B*) Localized area of narrowing at bronchial bifurcation of truncular spasm (arrow) in a 7 year old female.

and retained secretions were found in many nonasthmatic children undergoing bronchography under general anesthesia for recurrent pulmonary disease.

1. *Spasm.*

The most common finding was bronchial spasm. Nearly all cases showed diffuse spasm, *i.e.*, gradually tapering areas of narrowing along smaller airways with wider distal diameters (Fig. 1*A*). A more specific finding in asthmatics was truncular spasm. This appeared as a "pair of pants" with constriction along the parent and daughter branches at bifurcations (Fig. 1*B*). This phenomenon is related to constriction of the abundant smooth muscle bundles present at these division points.[4]

2. *Secretions.*

Retained secretions were second in frequency. This was the most common of the many signs related to the abnormal and abundant mucus present in asthmatic children. In spite of careful premedications and patient preparation, most studies showed evidence of retained bronchial secretions. They appeared as bubbles within the contrast coated lumen. Pooled secretions would prevent the suction effect needed for good peripheral filling and would lead to mucus plugging and bronchial wall irregularities.[1] In regard to adult bronchitis, Trapnell and Gregg[10] considered intraluminal lucencies to represent air bubbles mixing with the contrast agent secondary to more peripheral mucus obstruction (Fig. 2*A*). In any event, this should not be confused with air bubbles seen as a technical artifact when vigorously prepared barium suspensions are used (Fig. 2*B*).

3. *Dilatation.*

Irregular bronchial dilatation (Fig. 3*A*) was demonstrated as a loss of normal tapering of the airway lumen and appeared more widespread and milder than fixed bronchiectasis.[3] Although

this was not related to any visible segmental collapse, it could have represented another manifestation of more peripheral airway obstruction.

4. *Mucus plugging.*

Mucus plugging of small airways was noted with less than one-half the incidence of the first 3 findings. It was seen as an abrupt termination of contrast material filling due to obliterated small bronchi and bronchioles (Fig. 3*B*).[3] The sharp cut-off of the column of contrast medium was not necessarily as far down as the actual point of obstruction. A solid column cut-off indicated obstruction, whereas a tubular column cut-off represented inadequate filling.[10]

5. *Mucosal irregularity.*

Bronchial mucosal irregularities occurred at about the same frequency as mucus plugging (Fig. 3*C*). In most cases they represented collections of mucus impacted against bronchial walls,[1] rather than contrast medium filling around localized areas of mucosal destruction.[4]

Less common findings were more specific for chronic bronchitis.

6. *Patchy bronchiolar filling.*

Patchy bronchiolar filling represented over-filling in some areas separated by adjacent areas of peripheral obstruction and non-filling.[4] It may also represent localized loss of normal protective mechanisms of ciliary activity and the associated mucus plug which ordinarily retards peripheral filling. Patchy bronchiolar filling is a more accurate term than the more popular description of "patchy alveolarization" (Fig. 4*A*).[3]

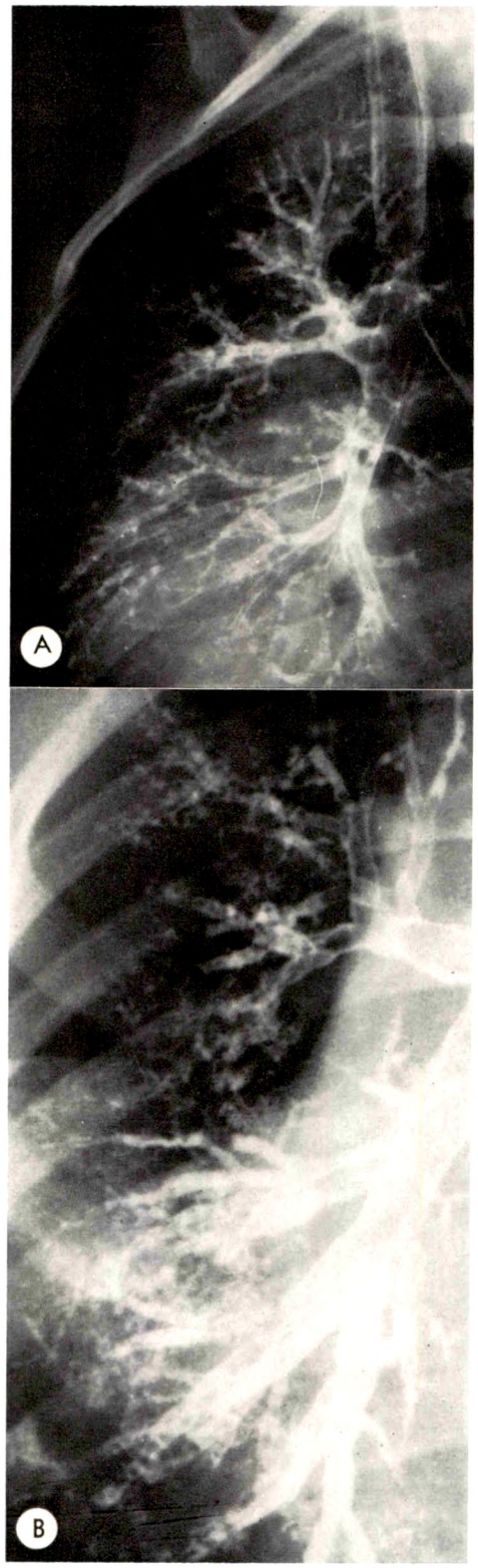


FIG. 2. (*A*) Multiple filling defects in bronchi due to retained mucus secretions in a 3 year old boy. (*B*) Multiple lucencies in proximal bronchi representing air bubble artifacts produced during preparation of an aqueous barium solution. Retained secretions are seen peripherally in an 8 year old female.

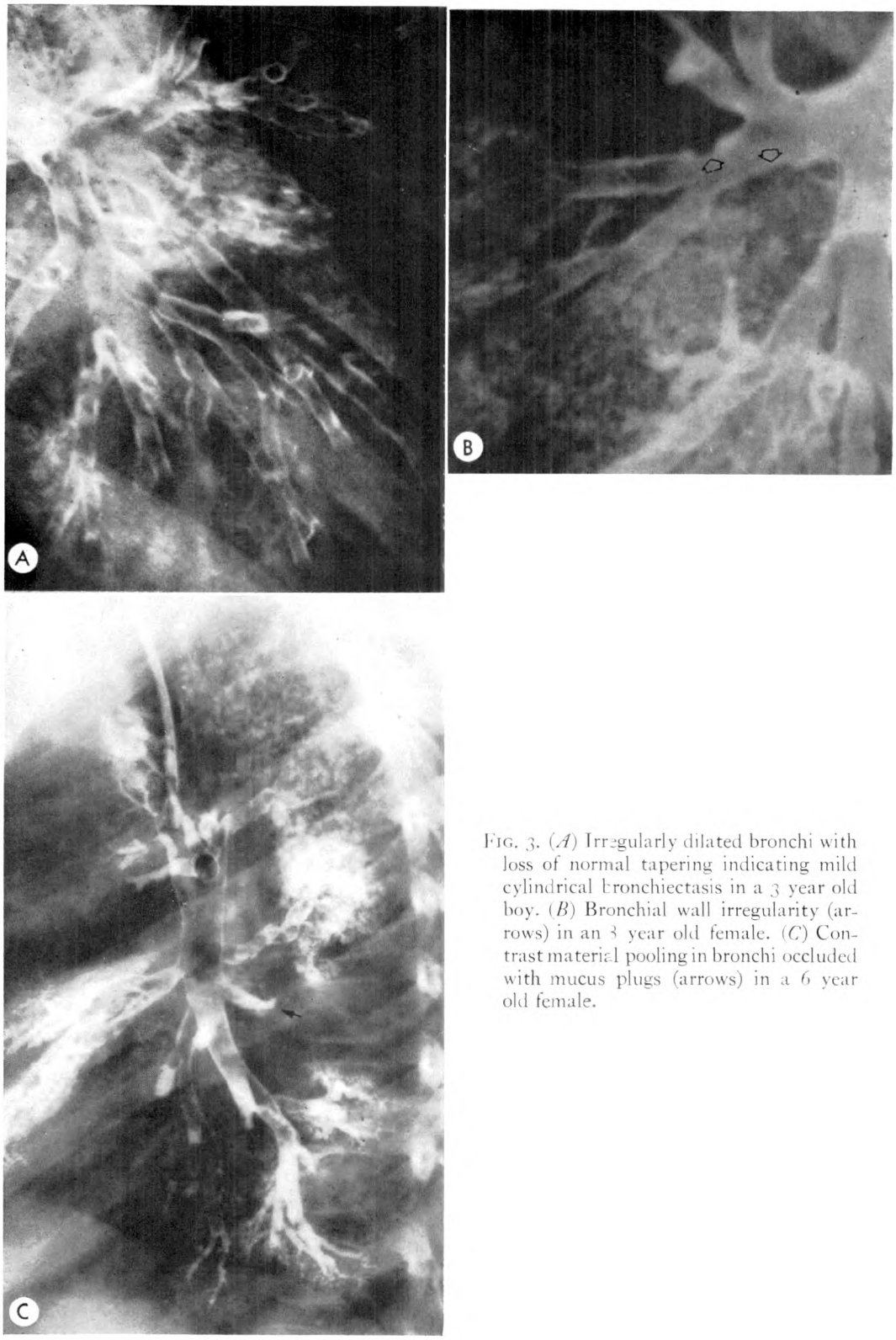


FIG. 3. (*A*) Irregularly dilated bronchi with loss of normal tapering indicating mild cylindrical bronchiectasis in a 3 year old boy. (*B*) Bronchial wall irregularity (arrows) in an 8 year old female. (*C*) Contrast material pooling in bronchi occluded with mucus plugs (arrows) in a 6 year old female.

This patchy peripheral filling should be differentiated from the technical error illustrated in Figure 4*B*. Diffuse "alveolarization" is related to generalized overventilation with low viscosity contrast material.

7. *Mucous gland duct filling.*

Dilated mucous gland duct filling is said to be characteristic of chronic bronchitis in adults. It is usually seen along the undersurfaces of lobar bronchi. In the 2 cases illustrated, it can be seen in segmental bronchi along both the lower and upper surfaces (Fig. 5, *A* and *B*). These represent contrast medium filling of the dilated necks of enlarged mucous glands.

8. *Mucosal atrophy.*

With mucosal atrophy, the thin coating provided by an aqueous medium (hytrast) presents a corrugated appearance along intrapulmonary bronchi of hypertrophied underlying smooth muscle rings (Fig. 6*A*). The same phenomenon along an extrapulmonary mainstem bronchus would demonstrate accentuation of the bronchial cartilage rings.[9]

9. *Bronchiolectasis.*

Bronchiolectasis is a rare finding in adults as well as children (Fig. 6, *B* and *C*). It is the pools and spikes described by Reid;[6] the pools being dilated bronchioles proximal to an obstruction and the spikes representing dilated bronchioles with filling of more peripheral terminal bronchioles. This finding is evidence of diffuse advanced disease, reflecting fibrosis and irreversible destruction.

BRONCHIECTASIS AND COLLAPSE

Although only selected patients had bronchograms, localized areas of obvious bronchiectasis or chronic segmental collapse were found in only 12 children, or 21 per cent. Ten of these involved the right middle lobe and were apparent on preliminary chest roentgenograms. One was in the lingular segment and was also evident on preliminary roentgenograms. One patient presented with chronic collapse of the left lower lobe which was detectable on the plain roentgenograms, but not readily apparent because of the chronic nature and compensatory overinflation of the adjacent lung (Fig. 7, *A* and *B*).

COMPLICATING SEGMENTAL COLLAPSE

As reported earlier, a 45 per cent incidence of complicating segmental loss of

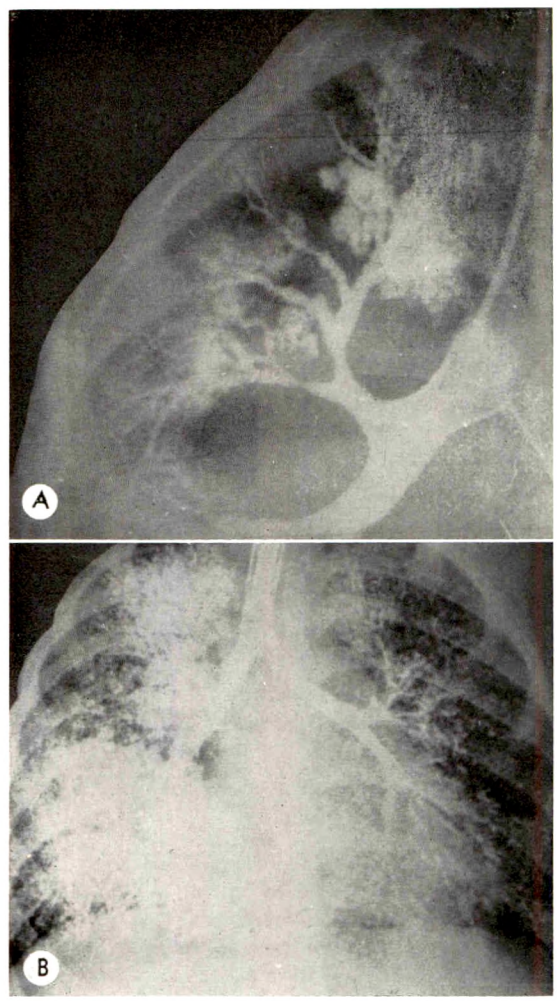


FIG. 4. (*A*) Patchy peripheral dispersion ("alveolarization") of contrast material related primarily to obstruction of adjacent small bronchi and bronchioles in a 10 year old female. (*B*) Diffuse bronchiolar filling due to improper bronchographic technique in a 2 year old female.

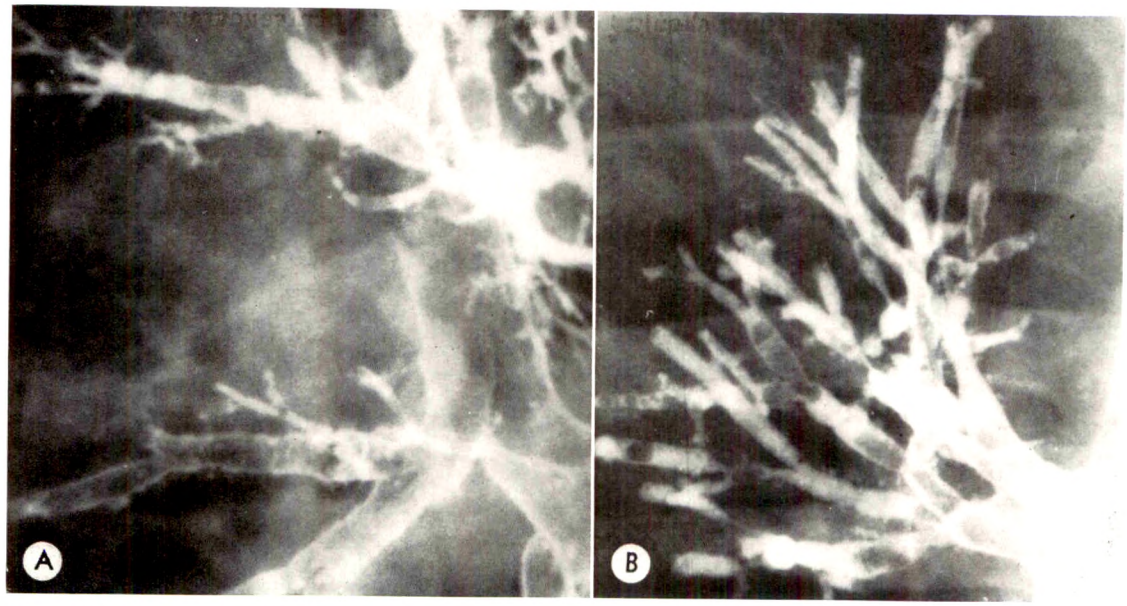


FIG. 5. Dilated mucous gland ducts filled with contrast material along (*A*) right middle lobe bronchi (8 year old male) and (*B*) superior and inferior aspects of the upper lobe bronchi (12 year old male).

volume was found in children undergoing bronchography under general anesthesia.[7] This was related to the highly diffusible anesthetic gas mixture used which allowed the alveoli to empty faster than they could be filled by airways compromised by both contrast medium coating and bronchoconstriction. In addition, collateral filling was hampered by the many adjacent mucus plugs present. Subsequent segmental collapse distorted the pulmonary architecture, making evaluation more difficult and further diminishing pulmonary gas exchange.

CONCLUSIONS

Widespread roentgenographic abnormalities of chronic bronchitis were evident in asthmatic children with recurrent pulmonary infection. They could be found on conventional chest roentgenograms as peribronchial cuffing, interstitial thickening, and generalized hyperaeration. Diffuse abnormalities were also seen at bronchography. Abnormal mucus production resulted in bubbly filling defects and mucus plugging of bronchi as well as dilated mucous gland duct filling and bronchial wall irregularities. Bronchospasm was demonstrated in both diffuse and localized truncular forms. Bronchial wall inflammation produced irregular dilatation and occasional bronchiectasis and bronchiolectasis. Mucosal damage resulted in patchy bronchiolar filling and surface changes of atrophy.

Many of the bronchographic abnormalities were mild and correlated well with the diffuse conventional roentgen findings. Only the more severely involved showed evidence of prominent pulmonary destruction and impaired mucociliary drainage. When bronchography revealed isolated destruction which might be amenable to surgical resection, it confirmed the abnormalities of segmental collapse and/or cystic changes evident on earlier conventional studies. Bronchography offers an excellent means for study of the pathophysiology of chronic bronchitis in childhood asthma, but only rarely will it provide new information necessary for specialized treatment. Consideration must be given to the complications of contrast media and general anesthesia in regard to any anticipated yield from bronchography.

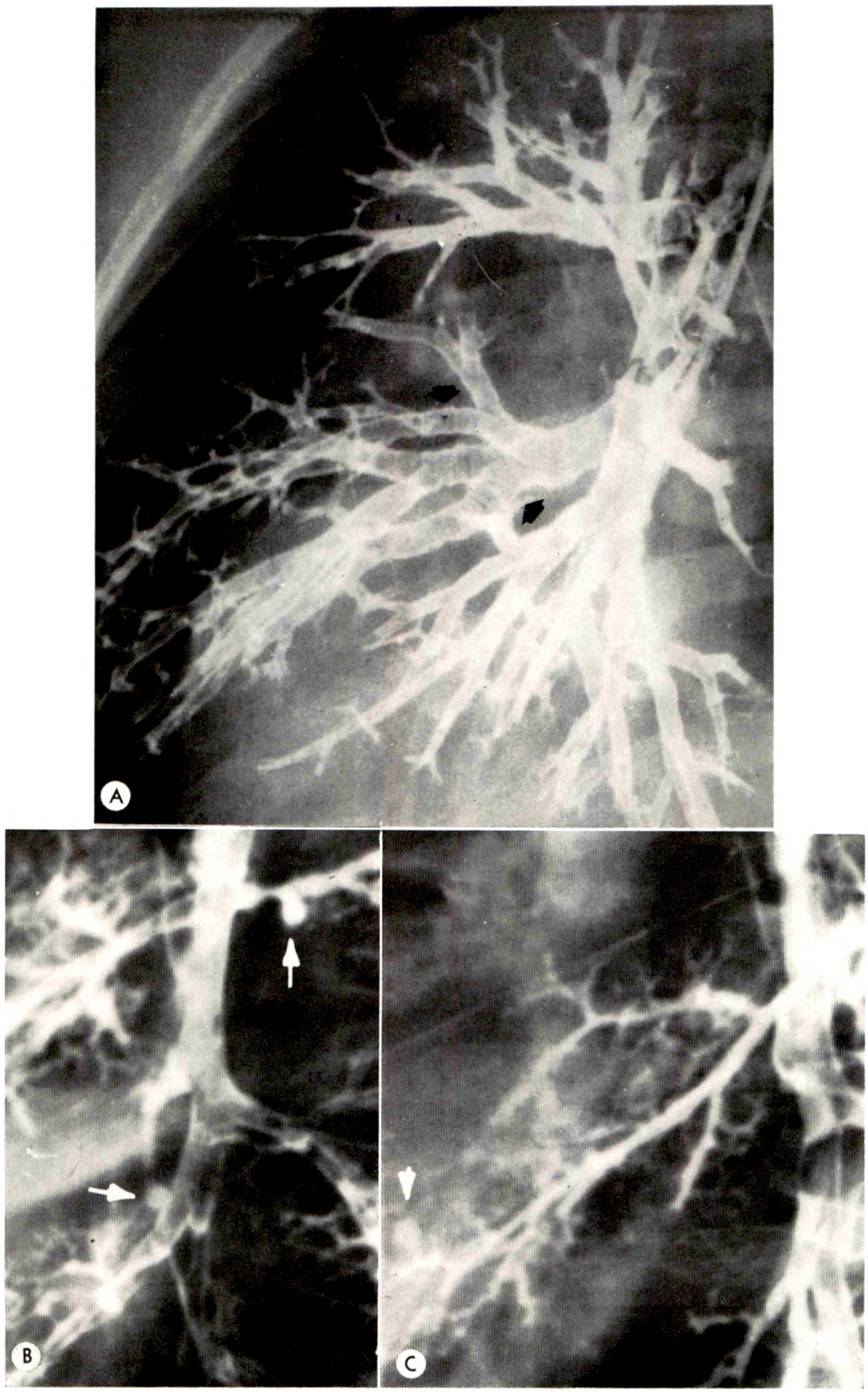


Fig. 6. (*A*) Coating of atrophic mucosa with accentuation of muscle bundles (arrows) in an 11 year old male. (*B* and *C*) Filling of dilated bronchioles proximal to inflammatory obstruction and fibrosis (arrows).

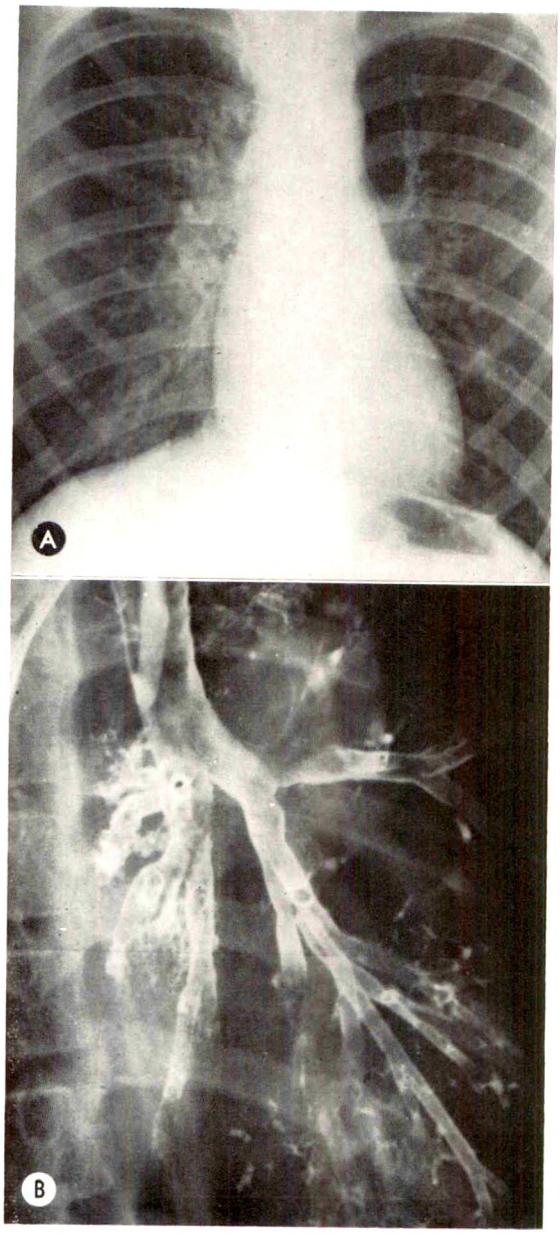


FIG. 7. (*A*) An 11 year old male asthmatic with prominent interstitial markings on the right, a hyperlucent left lung, and an indistinct retrocardiac density. (*B*) Bronchogram demonstrates chronic left lower lobe collapse with bronchiectasis (left posterior oblique projection).

SUMMARY

It was common for children with bronchial asthma and the problem of recurrent co-existing pulmonary inflammatory disease to demonstrate many of the diffuse changes attributed to chronic bronchitis at bronchography.

It was unusual for advanced localized abnormalities to be present without being evident on routine chest roentgenograms.

Complicating segmental loss of volume associated with general anesthesia added to the difficulty in performing bronchographies.

Since only diffuse changes are usually demonstrated, bronchography in childhood asthma should be reserved for special situations.

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REFERENCES

1. CHRISTOFORIDIS, A. J. RSNA Refresher Course on Bronchography, 1969.
2. DEES, S. C., and SPOCK, A. Right middle lobe syndrome in children. *J.A.M.A.*, 1966, *197*, 8–14.
3. FRASER, R. G., and PARÉ, J. A. P. Diagnosis of Diseases of the Chest. W. B. Saunders Company, Philadelphia, 1970.
4. FREIMANIS, A. K., and MOLAR, W. Chronic bronchitis and emphysema at bronchography. *Radiology*, 1960, *74*, 194–205.
5. GREGG, I., and TRAPNELL, D. H. Bronchographic appearance of early bronchitis. *Brit. J. Radiol.*, 1969, *42*, 132–139.
6. REID, L. M. Correlation of certain bronchographic abnormalities seen in chronic bronchitis with pathological changes. *Thorax*, 1955, *10*, 199–204.
7. ROBINSON, A. E., HALL, K. D., YOKOYAMA, K. N., and CAPP, M. P. Pediatric bronchography: problem of segmental loss of volume. I. Retrospective study of 165 pediatric bronchograms. *Invest. Radiol.*, 1971, *6*, 89–94.
8. SAMMONS, B. P., PISCHOTTE, W. O., and WILLIAMS, J. K. Bronchographic sign of chronic bronchitis. *Radiology*, 1957, *69*, 389–392.
9. SIMON, G. Radiological changes in chronic bronchitis. *Brit. J. Radiol.*, 1959, *32*, 292–304.
10. TRAPNELL, D. H., and GREGG, I. Some principles of interpretation of bronchograms. *Brit. J. Radiol.*, 1969, *42*, 125–131.

Vol. 116, No. 3

MEDIASTINAL LYMPHADENOPATHY IN BUBONIC PLAGUE*

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ENLARGEMENT of peripheral lymph nodes is the hallmark of bubonic plague. In their classical description of lymphadenopathy in plague, Albrecht and Ghon[1] divided buboes into 3 pathologically distinct types: (a) primary buboes of the first order, those draining the area of infection; (b) primary buboes of the second order, lymph nodes located adjacent to the originally invaded lymph nodes and infected via the lymphatics; and (c) secondary buboes, lymph nodes infected via the blood stream secondary to septicemia.

Enlargement of the mediastinal lymph nodes was noted at postmortem examination in the pneumonic plague epidemic in Manchuria in 1910–1911.[7] Koulecha[4] believed that these enlarged lymph nodes displayed the pathologic changes of primary buboes of the second order. Occasional mention of enlarged mediastinal lymph nodes in nonpneumonic plague has appeared in the literature;[2] however, there has been a paucity of roentgenographic studies of mediastinal lymphadenopathy in both pneumonic and nonpneumonic plague.

The roentgenographic studies of 2 cases of mediastinal lymph node enlargement in bubonic plague are reported.

REPORT OF CASES

CASE I. A 3 year old boy developed fever and chills on June 23, 1969, 3 days after playing with a dead chipmunk near his home in Jemez Springs, New Mexico. During the ensuing week, in spite of therapy with parenteral penicillin and oral ampicillin,[6] he continued to be febrile and developed a severe nonproductive cough. On June 30, an enlarged left inguinal lymph node and bilateral axillary lymphadenopathy were noted, and he was hospitalized. Admission chest roentgenograms demonstrated enlarged mediastinal lymph nodes, which enlarged further during hospitalization (Fig. 1, *A–D*). His cerebrospinal fluid was normal on admission, but on July 3, a pleocytosis of 658 white blood cells (50 per cent polymorphonuclears) was present; no organism was cultured from the cerebrospinal fluid. The enlarged inguinal lymph node was excised and cultured on July 2, and the patient was begun on chloramphenicol intravenously. An organism subsequently identified as *Yersinia pestis* was recovered from the lymph node. Following the presumptive identification of this organism on July 4, tetracycline and streptomycin were added to his regimen. *Y. pestis* was not isolated from the blood and throat cultures taken on July 2 or from tracheal aspiration obtained on July 4. The patient recovered gradually, but on July 12 he developed symptoms of congestive heart failure and was digitalized. His subsequent recuperation was uneventful.

CASE II. An 8 year old boy developed fever on May 30, 1970, 5 days after he had close contact with several ground squirrels, while on a camping trip. He also had received numerous insect bites on that trip. Bilateral supraclavicular and femoral lymphadenopathy developed over the ensuing 3 days. He was given penicillin as an outpatient on May 31 and was hospitalized on June 3 because of his worsening condition. On June 4, he was begun on chloramphenicol followed by a slow but progressive clinical improvement. *Y. pestis* was isolated from a blood culture obtained prior to administration of chloramphenicol. Throat culture revealed "normal flora;" no sputum cultures were obtained. Chest roentgenograms taken during the illness demonstrated enlarge-

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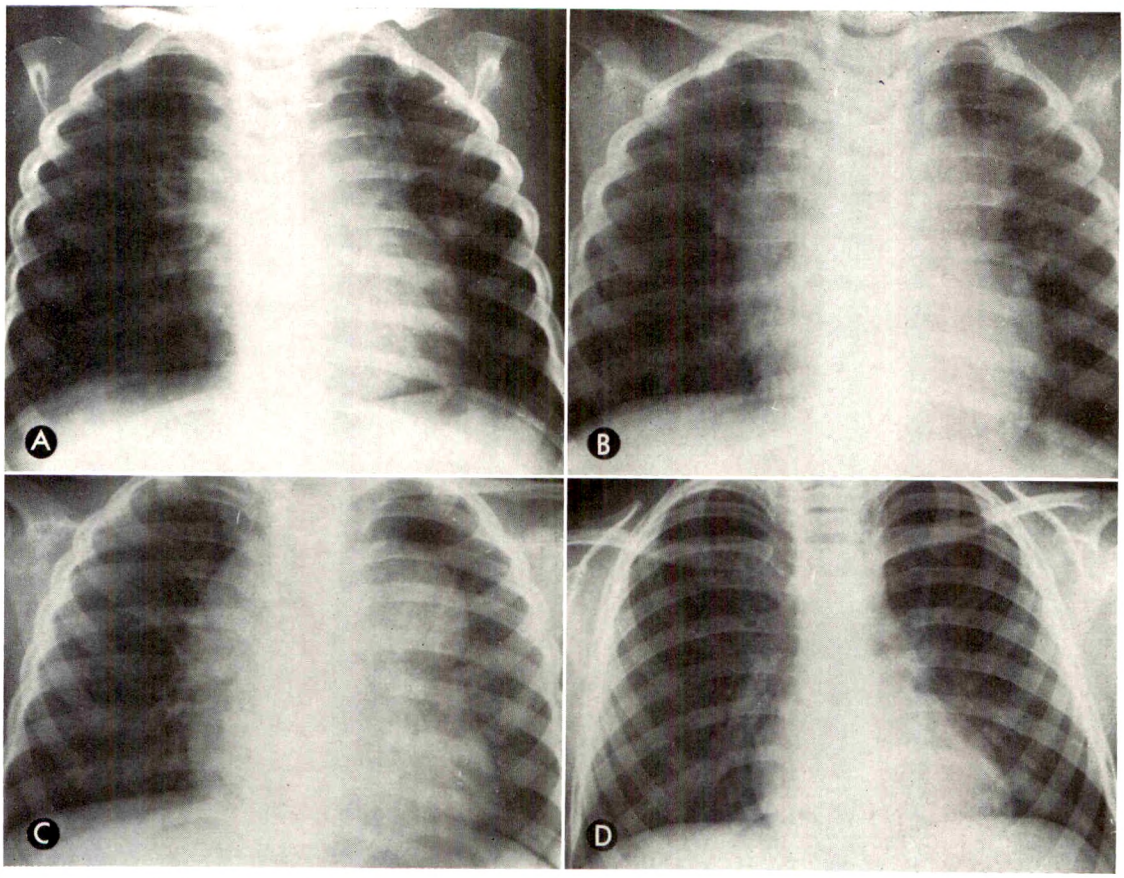


FIG. 1. Case 1. (*A*) *July 3, 1969.* (*B*) *July 5, 1969.* (*C*) *July 17, 1969.* Marked enlargement of the hilar lymph nodes which progressed to massive enlargement over the ensuing 2 week period. (*D*) *October 11, 1969.* The follow-up roentgenogram demonstrates complete clearing of the lymphadenopathy. (*A* and *D* also published in *Medicine*, 1970, *49*, 476, and reproduced with permission of the authors[6] and The Williams & Wilkins Co., Baltimore, Md.)

ment of the right hilar lymph nodes and pulmonary infiltrates (Fig. 2, *A–C*). His recovery was uneventful.

DISCUSSION

Case 1 contracted classical bubonic plague, and Case II was unusual in that multiple peripheral lymphadenopathy was evident early in his illness. Multiple lymphadenopathy, although uncommon, has been described in bubonic plague and was seen by one of the authors (J. P.) during an epidemic in Java in 1968.[5] Both patients eventually developed lymphadenopathy in multiple lymph node areas, including the mediastinal lymph nodes. The question arises whether the enlarged mediastinal lymph nodes were related to the development of secondary pneumonic plague (primary bubo of the second order) or were infected secondary to a bacteremia (secondary bubo). The negative blood culture on Case 1 was obtained about 2 hours after chloramphenicol was started; a single negative culture could also suggest either that the ampicillin therapy suppressed the organism, or that he had intermittent bacteremia rather than septicemia. Both patients had parenchymal infiltrates, although these were more marked in Case II. Nonspecific pulmonary infiltrates are frequently seen in bubonic plague and are probably due to atelectasis. The sputum culture was obtained after therapy was

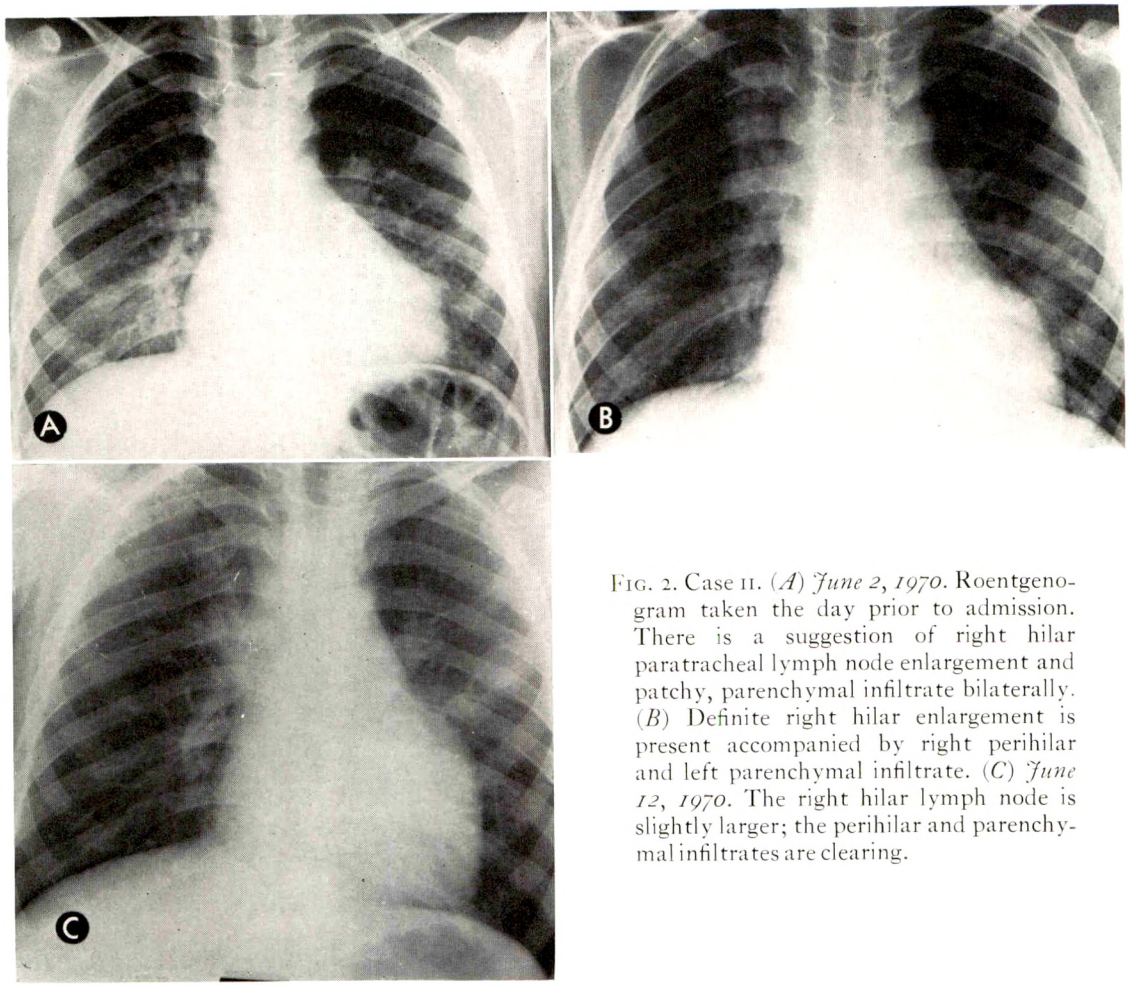


Fig. 2. Case II. (*A*) *June 2, 1970.* Roentgenogram taken the day prior to admission. There is a suggestion of right hilar paratracheal lymph node enlargement and patchy, parenchymal infiltrate bilaterally. (*B*) Definite right hilar enlargement is present accompanied by right perihilar and left parenchymal infiltrate. (*C*) *June 12, 1970.* The right hilar lymph node is slightly larger; the perihilar and parenchymal infiltrates are clearing.

begun in Case I and sputum culture was not attempted in Case II, so that secondary plague pneumonia cannot be excluded as the cause of the pulmonary infiltrate. However, because of the relatively benign course of the pulmonary infiltrates and the absence of primary pneumonic cases among unprotected medical personnel caring for patients, the pulmonary infiltrates probably did not represent frank plague pneumonia.

The mediastinal lymph nodes probably were infected secondary to the septicemia or bacteremia in both cases. The existence of bacteremia was suggested by the onset of meningitis in Case I and was demonstrated by the positive blood culture in Case II. Thus, these enlarged lymph nodes are most likely secondary buboes, according to Albrecht and Ghon's classification.

CONCLUSION

Bubonic plague should be considered in the differential diagnosis of acutely ill patients with enlargement of the mediastinal lymph nodes.

This is particularly true in plague-endemic areas, but with the speed of modern transportation, all such cases should be questioned about recent travel to endemic areas.[3]

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REFERENCES

1. Albrecht, H., and Ghon, A. Denkschr. Akad. Wiss., Wien, 1900, *66*, 581. In: Plague. Edited by R. Pollitzer. World Health Organization Monograph, 1954, *22*, 206–207.
2. Finegold, J. J. Pathogenesis of plague. *Am. J. Med.*, 1965, *45*, 549–554.
3. Kartman, L., Goldenberg, M. I., and Hubbert, W. D. Recent observations on epidemiology of plague in United States. *Am. J. Pub. Health*, 1966, *56*, 1554–1569.
4. Koulecha, G. S. Morbid anatomy. Report on International Plague Conference, Manila, 1912, 151–155.
5. Poland, J. D., Chin, T. D. Y., Golub, B. P., Barnes, A. M., Hudson, B. W., Goldenberg, M. I., and Schoof, H. F. Investigation of plague outbreak in Central Java, Indonesia. Proceedings of Twenty-third International Northwestern Conference on Diseases in Nature Communicable to Man, 1968, 135.
6. Reed, W. P., Palmer, D. L., Williams, R. C., Jr., and Kisch, A. L. Bubonic plague in southwestern United States. *Medicine*, 1970, *49*, 465–486.
7. Strong, R. P. Morbid anatomy. Report on International Plague Conference, Manila, 1912, 135–142.

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POSTERIOR MEDIASTINAL "PSEUDO-MASS" OF THE NEWBORN*

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A RECENT case brought home to us the consequences of erroneous interpretation of mediastinal shadows in the newborn.

In the hope of sparing infant patients and other radiologists future discomfort, we offer this report.

REPORT OF A CASE

A 2 day old full term Caucasian male was sent for a chest roentgenogram because his mother had pulmonary tuberculosis. A posterior left mediastinal mass was suggested in both the frontal and lateral projections (Fig. 1, *A* and *B*). Barium esophagogram showed no displacement of the esophagus (Fig. 1*C*) and coned down views revealed no abnormalities of the spine.

Exploratory thoracotomy was performed at another institution with the tentative diagnosis of neuroblastoma; no tumor was found.

Comment. Examination of the roentgenograms (Fig. 1, *A–C*) led many radiologists to suggest the presence of a posterior mediastinal mass. The combination of a left mediastinal mass on the frontal roentgenogram (Fig. 1*B*), which was not recognized as the "ductus bump,"[1] and an apparent posterior mediastinal mass on the lateral roentgenogram, led to exploratory thoracotomy. The appearance in the lateral view, however, was due to a gentle anterior curvature of the tracheal air shadow and the lower lobe continuation of the left main stem bronchus combined with the adjacent densities of the scapulae and axillary soft tissues (Fig. 2, *A* and *B*). We have termed this appearance the posterior mediastinal "pseudo-mass" of the newborn.

INVESTIGATION

We reviewed 63 consecutive newborn lateral chest roentgenograms to determine how commonly the "pseudo-mass" is demonstrated. We considered that a roentgenogram showed a "pseudo-mass" either if the configuration of the airway strongly suggested the presence of posterior mediastinal mass or if the combination of scapular and soft tissue densities combined with the airway shadow to suggest a mass density. We found that 21 of the 63 cases reviewed showed a posterior mediastinal "pseudo-mass" (Fig. 3, *A* and *B*).

DISCUSSION

This experience underlined the importance of recognizing a "pseudo-mass" of the posterior mediastinum. In a newborn with the roentgenographic finding of a posterior mediastinal mass density, but with no bony abnormality or esophageal deviation, this entity must be considered.

Although we found a "pseudo-mass" in one-third of newborn lateral chest roentgenograms, the finding is rarely noted clinically since the frontal roentgenogram is completely normal. The shadow of the "pseudo-mass" became important in our case because the frontal roentgenogram showed a corresponding mass density, subsequently recognized as a "ductus bump." Berdon *et al.*[1] described the ductus bump in 1965 as a left mediastinal mass in the newborn, seen only in the frontal roentgenogram. They postulated that visualization of the large ductus arteriosus was a reflection of normally slow neonatal adjustment to extrauterine life, the ductus remaining patent during the first 24 hours of life. In the series of 63 newborn chest examinations reviewed, we found one additional ductus bump.

Other variations in the contour of the tracheal air column have been described. Chang *et al.*[2] reported lateral deviation or

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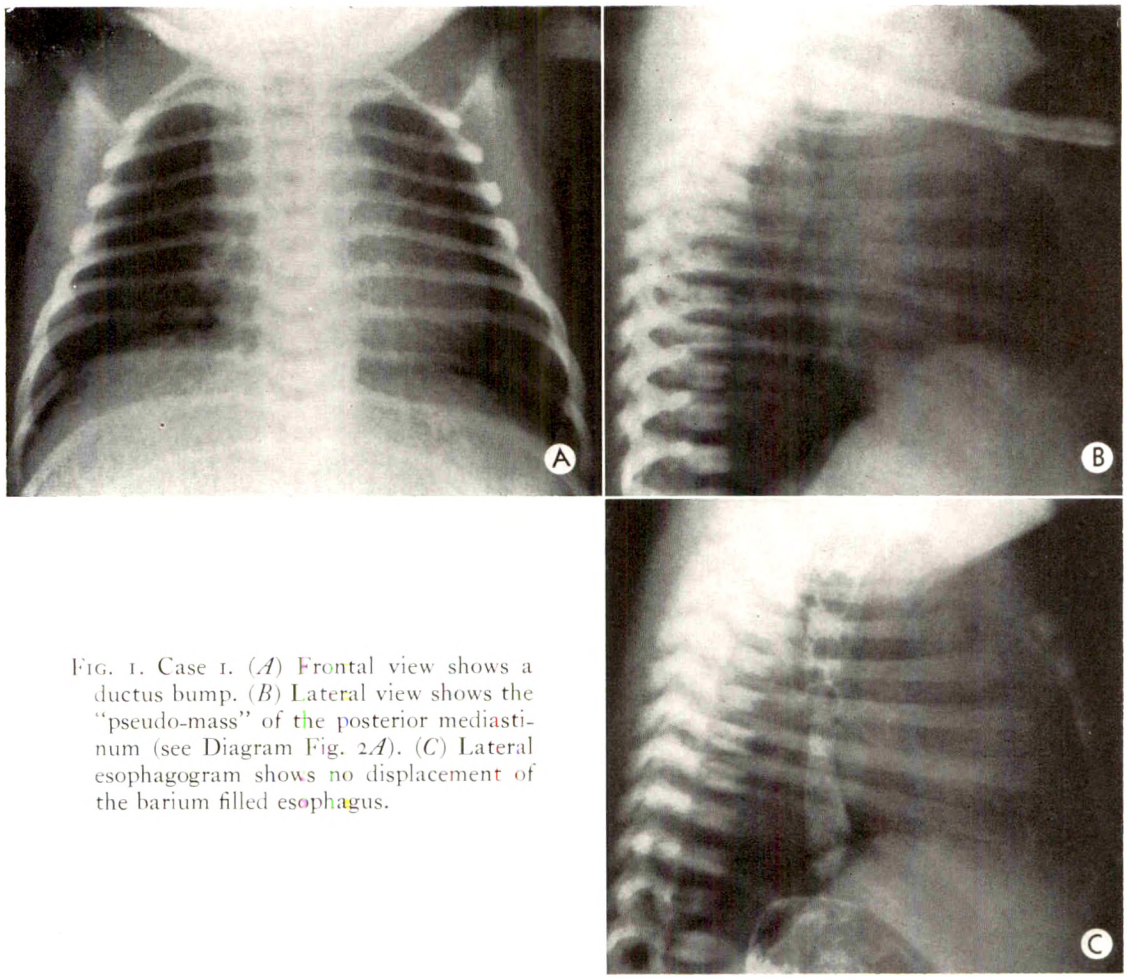


FIG. 1. Case 1. (*A*) Frontal view shows a ductus bump. (*B*) Lateral view shows the "pseudo-mass" of the posterior mediastinum (see Diagram Fig. 2*A*). (*C*) Lateral esophagogram shows no displacement of the barium filled esophagus.

buckling of the tracheal air column at or just above the thoracic inlet as a normal finding on frontal chest roentgenograms of infants and children. Swischuk[5] recently studied another common deformity of the trachea in infants. He reported that the anterior tracheal indentation seen in the lateral projection was vascular in origin, confirming this angiographically as the innominate artery. The anterior curvature of the trachea and left lower lobe bronchus seen on lateral roentgenograms, delineating the "pseudo-mass," is a normal configuration unrelated to these other variants.

In view of the incidence of neuroblastoma discovered on routine chest roentgenograms, critical interpretation of the newborn chest roentgenogram is essential. Heimberger and Battersby,[3] reporting their experience with mediastinal tumors in infants and children, found that one-third of 42 patients with mediastinal tumors had no symptoms at the time of diagnosis. The mass was detected as an incidental finding on chest roentgenography. In the newborn, neuroblastoma is the most common solid tumor of the posterior mediastinum.[4] Duplication of the foregut or neurenteric cyst is the second most common mass in this region. With this in mind, the radiologist must nevertheless be wary of combinations of normal anatomic densities which may simulate mediastinal abnormalities and lead to unnecessary surgical exploration.

SUMMARY

In a significant proportion of newborns, the lateral chest roentgenogram appears to show a posterior mediastinal mass.

The fortuitous superimposition of scapu-

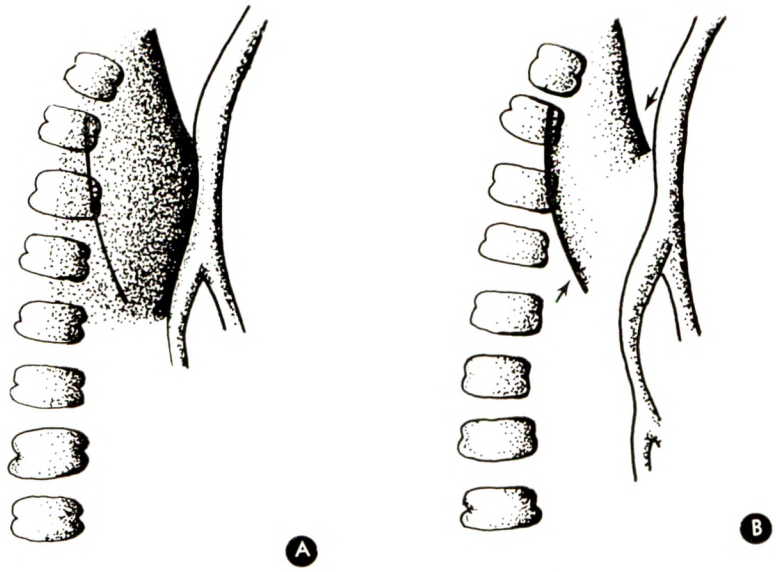


Fig. 2. Diagrams of the lateral chest roentgenogram demonstrating the composition of the "pseudo-mass" of the posterior mediastinum. (*A*) Apparent posterior mediastinal mass (see Fig. 1*B*; and 3, *A* and *B*). (*B*) The "pseudo-mass" consists of shadows formed by scapulae (arrows) and axillary soft tissues in conjunction with the tracheal and left bronchial airways.

lar and axillary soft tissue shadows and a gentle anterior curvature of the adjacent tracheo-bronchial air column account for the "pseudo-mass."

These findings are correctly disregarded when the frontal roentgenogram is completely normal.

Occasionally, a localized mediastinal bulge in the frontal projection due to the ductus is also seen. Simultaneous occur-

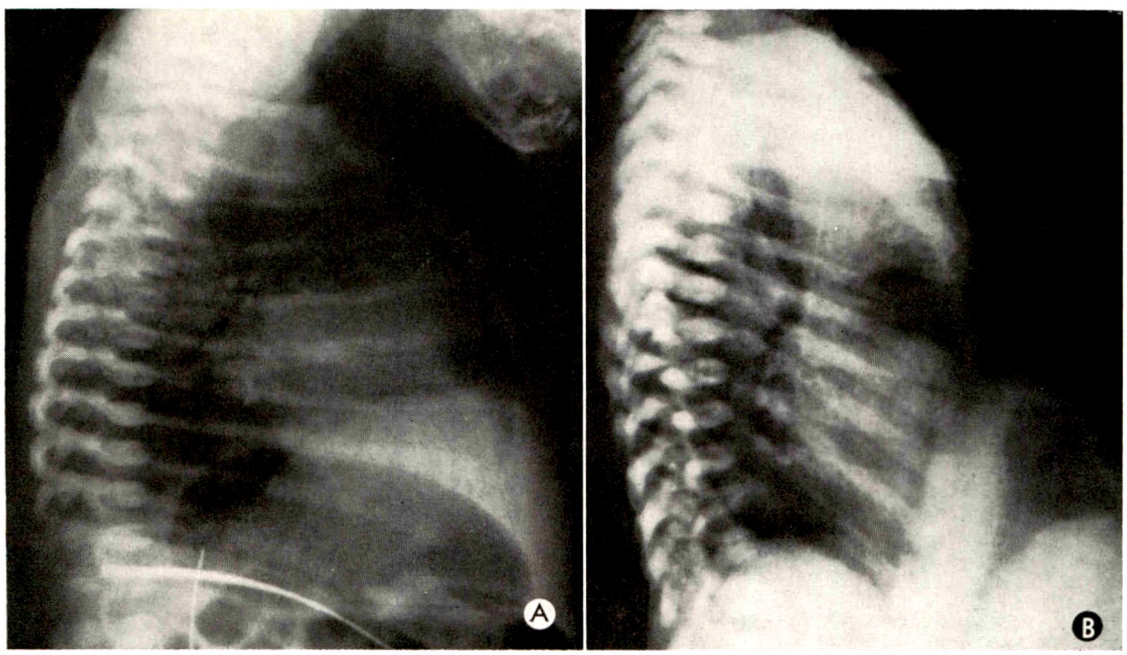


Fig. 3. (*A*) Case II. Premature baby with incidental finding of "pseudo-mass" in the posterior mediastinum. (*B*) Case III. Newborn with an omphalocele and incidental finding of the posterior mediastinal "pseudo-mass."

rence of these unrelated shadows may result in formidable diagnostic overevaluation and surgery.

If a "pseudo-mass" is considered in the differential diagnosis of posterior mediastinal shadows in the newborn, it can easily be distinguished roentgenologically from a true posterior mediastinal mass.

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REFERENCES

1. BERDON, W. E., BAKER, D. H., and JAMES, L. S. Ductus bump: a transient physiologic mass in chest roentgenograms of newborn infants. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1965, *95*, 91–98.
2. CHANG, L. W., LEE, F. A., and GWINN, J. L. Normal lateral deviation of trachea in infants and children. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1970, *109*, 247–251.
3. HEIMBERGER, I., and BATTERSBY, J. S. Primary mediastinal tumors of childhood. *J. Thoracic & Cardiovasc. Surg.*, 1965, *50*, 92–103.
4. HOPE, J. C., and KOOP, C. E. Differential diagnosis of mediastinal masses. *Pediat. Clin. North America*, 1959, *6*, 379–412.
5. SWISCHUK, L. E. Anterior tracheal indentation in infancy and early childhood: normal or abnormal? AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1971, *112*, 12–17.

LACTOBEZOAR*

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A FORMULA of powdered milk mixed with an inadequate amount of water may cause formation of a coagulum in the stomach of an infant. This foreign body has been termed "lactobezoar."

The mass may lead to upper gastrointestinal symptoms.

The present report is an example of a symptomatic lactobezoar which resolved spontaneously.

REPORT OF A CASE

A 4½ month old male infant was admitted to the Children's Hospital of the District of Columbia with a 1½ day history of vomiting and diarrhea.

On physical examination, a large, firm abdominal mass was found in the left upper quadrant of the abdomen. The mass was movable and appeared to be separate from the liver. The patient's temperature was 104° F. The physical examination was otherwise unremarkable.

The original roentgenograms of the abdomen showed a large, mottled, soft tissue density in the left upper quadrant, extending to the midportion of the abdomen and crossing the midline. The upper border of the mass was outlined by a crescent of air, which appeared to be in the fundus of the stomach. A repeat roentgenogram of the abdomen, after instillation of 50 ml. of air into the stomach via a nasogastric tube, clearly outlined a characteristic bezoar filling the infant's stomach (Fig. 1).

The patient was started on intravenous fluids with a plan to remove the bezoar through a gastrotomy. The following morning, the large abdominal mass was no longer palpable. Repeat roentgenogram of the abdomen, as well as an upper gastrointestinal series, were normal.

In retrospect, a careful feeding history showed that the infant's formula had been changed 2 days prior to hospitalization. A powdered milk formula had been substituted for one which contained evaporated milk and water. The powdered milk formula had been prepared haphazardly. Water was added to the powder and the mixture was stirred with an egg beater until a consistency of thick cream was obtained. The mixture was then fed to the baby.



FIG. 1. An air contrast study of the stomach shows the characteristic mottled appearance of a lactobezoar.

DISCUSSION

In 1959, Wolf and Bruce[2] reported a case of lactobezoar in an 18 day old infant which was apparently caused by a powdered milk formula mixed with an inadequate amount of water. The lactobezoar was removed through a gastrotomy. Wolf and Davis,[3] in 1963, reported 2 additional cases of lactobezoar secondary to concentrated formula which resolved spontaneously. They emphasized that the continued use of incorrectly prepared pow-

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dered milk products can form abdominal masses which may lead to upper gastrointestinal symptoms. Levkoff *et al.*,[1] in 1970, reported lactobezoar and gastric perforation in an 11 day old premature infant associated with multiple gastric ulcers and pneumatosis of the stomach. These complications occurred during the recovery phase of hyaline membrane disease. In this infant, the relationship of the multiplicity of findings is not clear.

If the reported cases of lactobezoar are analyzed together with our own, 2 common features are apparent: (1) a concentrated powdered milk formula was fed each infant; and (2) hospitalization and the discovery of the lactobezoar was accompanied by diarrhea and/or vomiting. In the opinion of the authors, the pathogenesis of lactobezoar in infancy is the result of a vicious cycle. The concentrated formula probably precipitates the diarrhea and/or vomiting. This produces a diminution of the extracellular fluid volume (dehydration). To replenish the extracellular fluid volume, water is absorbed from the gastrointestinal tract (including the stomach). A lactobezoar is thus formed and by mechanical obstruction of the stomach causes the vomiting to persist.

Wolf and Davis[3] stress the importance of careful instruction of parents in reconstituting powdered milk formulas. The authors would add the following observations:

1. A gastric bezoar in an infant should immediately alert the physician to the possibility of a lactobezoar. A careful history of what the infant has been fed and how the formula was prepared should then be obtained.

2. Once the diagnosis of lactobezoar has been established, then conservative therapy should be instituted. In 3 of 5 infants, the administration of intravenous fluids resulted in rapid spontaneous resolution.

SUMMARY

The case report of a $4\frac{1}{2}$ month old infant with a lactobezoar secondary to feeding him an incorrectly reconstituted powdered milk formula is described. A review of the literature showed 4 similar cases.

In addition to an improperly prepared formula, diarrhea and/or vomiting were common features of this disorder. On this basis, the possible role of dehydration in the pathogenesis of lactobezoar is discussed.

The most important conclusion stresses conservative management. With the use of intravenous fluids, the lactobezoar should resolve spontaneously.

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REFERENCES

1. LEVKOFF, A. H., GADSDEN, R. H., HENNIGAR, G. R., and WEBB, C. M. Lactobezoar and gastric perforation in neonate. *J. Pediat.*, 1970, *77*, 875–877.
2. WOLF, R. S., and BRUCE, J. Gastrotomy for lactobezoar in newborn infant. *J. Pediat.*, 1959, *54*, 811–812.
3. WOLF, R. S., and DAVIS, L. A. Lactobezoar: foreign body formed by use of undiluted powdered milk substance. *J.A.M.A.*, 1963, *184*, 782.

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BILE DUCT ANOMALIES ASSOCIATED WITH DUODENAL ATRESIA; PARADOXICAL PRESENCE OF SMALL BOWEL GAS*

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IN THE newborn, the roentgenographic finding of a dilated duodenum with no gas beyond is invariably diagnostic of duodenal atresia. The finding of duodenal dilatation with gas beyond implies partial duodenal obstruction.

In 1969, Astley[2] reported 3 newborns with duodenal atresia whose abdominal roentgenograms revealed small bowel gas; autopsy in each demonstrated bifurcation of the terminal portion of the hepatopancreatic duct, one limb entering proximal to and the other distal to the atresia.

We have encountered this roentgenographic pattern in 2 newborns with duodenal atresia; in 1, autopsy confirmed the existence of bile duct anomalies.

REPORT OF CASES

CASE 1. B.G., a full-term male infant, developed severe respiratory distress shortly after birth; chest roentgenogram obtained at 3 hours demonstrated a diaphragmatic hernia on the left (Fig. 1*A*).

At surgery, the entire small bowel, stomach, spleen and a portion of the transverse colon were found in the left chest; the left lung was hypoplastic. The abdominal contents were reduced and the Bochdalek hernia was repaired.

Postoperatively, there was persistent collapse of the left lung and a large left pleural effusion. A roentgenogram on the third day revealed dilatation of the first portion of the duodenum, and no gas in the remainder of the bowel (Fig. 1*B*). The same roentgenographic pattern was seen on several subsequent examinations. On the sixth day the dilated stomach and duodenum were again noted; however, gas was now present in scattered loops of small bowel (Fig. 1*C*).

The infant was maintained on the Bird respirator and with intermittent nasogastric suction; on the seventh day a catheter was placed for intravenous hyperalimentation.

On the eighth day laparotomy was performed; there was atresia of the descending duodenum. The biliary tract was grossly normal. Duodenojejunostomy and gastrostomy were performed.

The postoperative course was stormy. A purulent left pleural effusion developed. Several wound sites grew out *Candida albicans*.

On the 18th day of life he suddenly developed bradycardia and cyanosis and died.

At autopsy, the immediate cause of death was *Candida albicans* sepsis. Extensive hemorrhage was present in both adrenals. There was empyema of the left chest. The repair of the Bochdalek hernia was intact. The mesentery was unfixed and the cecum lay free in the midabdomen. The duodenojejunostomy site was intact. The proximal duodenum was markedly dilated and ended blindly 1.5 cm. distal to the pylorus. There was a 7 mm. gap between the blind ends of the duodenum which were connected by a fine band of fibrous tissue. Pancreatic tissue occupied the bed beneath the atretic ends.

The biliary and pancreatic ducts were examined both by postmortem cholangiography and by gross dissection (Fig. 2, *A–C*; and 3). The common bile duct bifurcated at its distal end into 2 branches: (1) the shorter branch (5 mm. long) opened into the duodenum distal to the atresia; and (2) the longer branch (15 mm. long) opened into the duodenum proximal to the atresia. Neither duct terminated in a mucosal papilla. The dorsal pancreatic duct joined the ventral pancreatic duct to form the main pancreatic duct, which then entered the longer branch of the common bile duct at right angles. The accessory pancreatic duct was not identified.

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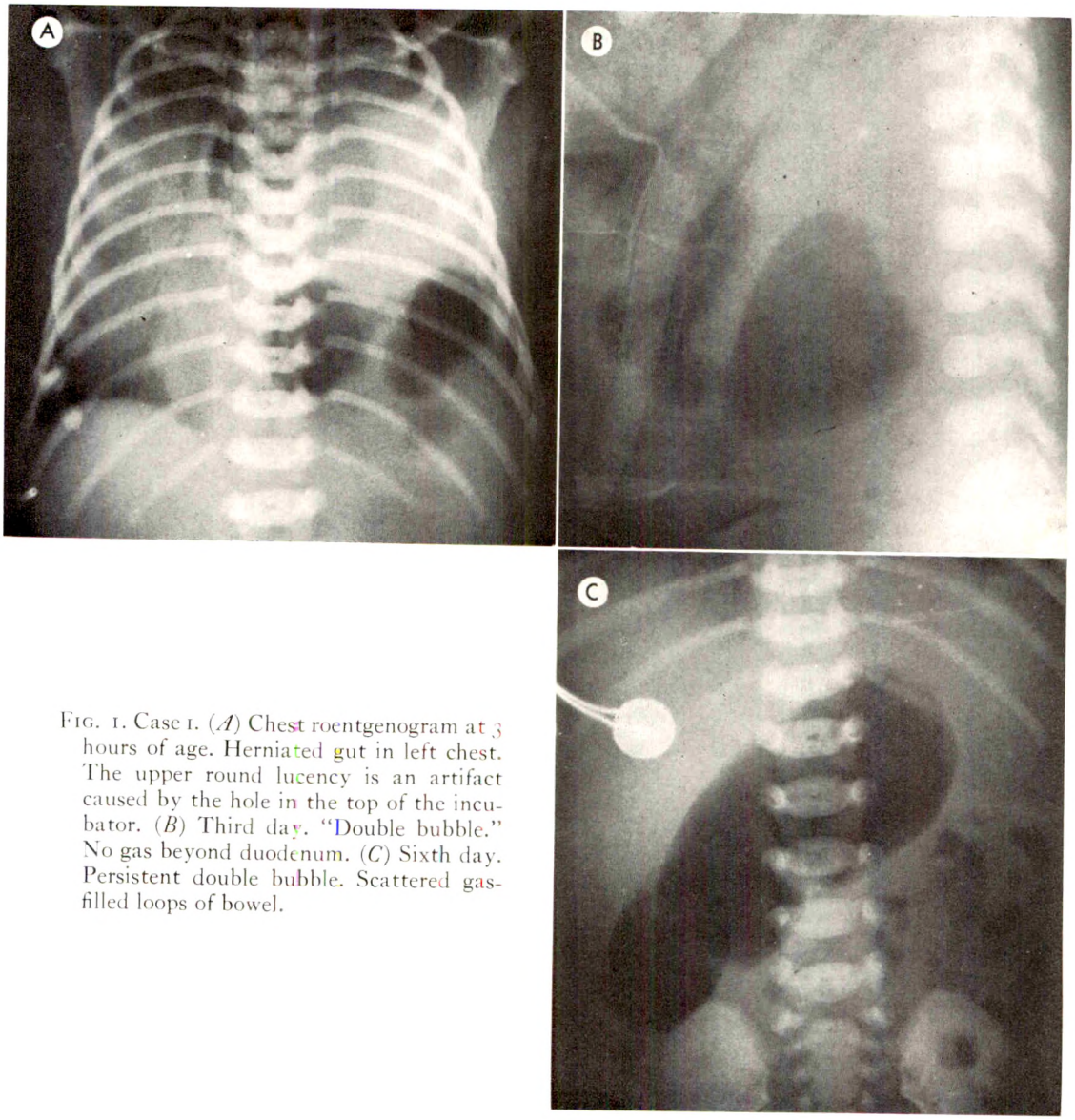


FIG. 1. Case I. (*A*) Chest roentgenogram at 3 hours of age. Herniated gut in left chest. The upper round lucency is an artifact caused by the hole in the top of the incubator. (*B*) Third day. "Double bubble." No gas beyond duodenum. (*C*) Sixth day. Persistent double bubble. Scattered gas-filled loops of bowel.

Several other congenital anomalies were noted: (1) hypoplasia of the left lung; (2) hyperplasia of the right kidney (which weighed 27 grams, compared to the normal left kidney which weighed 17 grams); and (3) accessory adrenal tissue in the mesentery.

CASE II.* M.F., a 1,930 gram premature female infant with Down's syndrome, was noted to have large amounts of bile stained gastric aspirate on the first day of life. A roentgenogram showed a fluid level in the stomach and slight gaseous distention of the proximal duodenum; there was scattered gas throughout the rest of the bowel (Fig. 4*A*). On Day 2 there was a "double bubble" and a small amount of gas was present in the small bowel (Fig. 4*B*). Barium meal demonstrated complete obstruction of the second portion of the duodenum (Fig. 4*C*).

At laparotomy the duodenum was seen to end in a blind pouch at about the junction of the second and third portions. There was a separation between the blind pouch and the blind-ending distal duodenum. The small bowel was collapsed and no intraluminal gas was visible.

* Kindly contributed by Dr. Walter Berdon and Dr. John Schullinger, Babies Hospital, New York, N. Y.

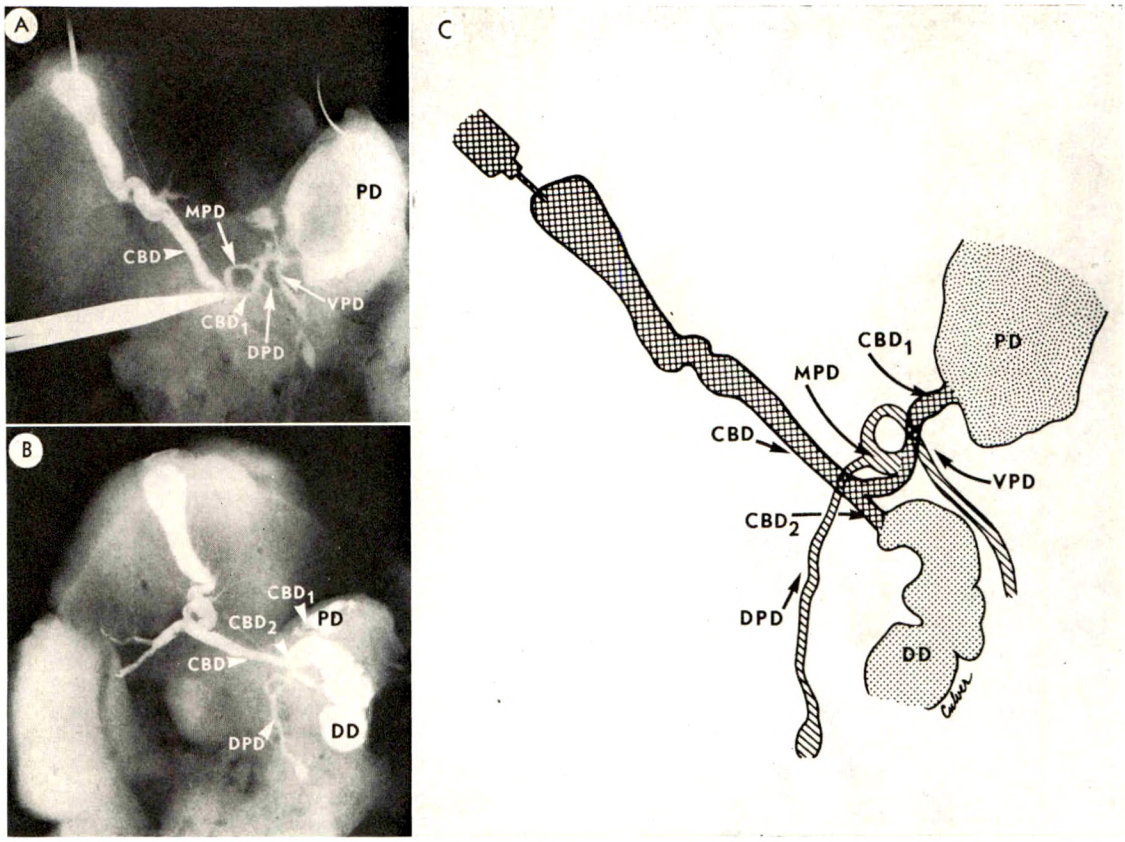


FIG. 2. Case 1. Postmortem cholangiograms. (*A*) Drainage from common bile duct is primarily into the duodenum distal to atresia. A small amount of contrast material enters the proximal duodenum via another branch (obscured in this projection). (*B*) Bile duct clamped at point of entry into distal duodenum. The branch to the proximal duodenum is now clearly shown. (*C*) Composite tracing (semischematic, because of slightly differing projections of *A* and *B*). PD = Duodenum proximal to atresia; DD = Duodenum distal to atresia; CBD = Common bile duct; CBD_1 = Branch of common bile duct to proximal duodenum; CBD_2 = Branch of common bile duct to distal duodenum; MPD = Main pancreatic duct; VPD = Ventral pancreatic duct; and DPD = Dorsal pancreatic duct.

The colon was normal in position and contained meconium. The liver and gallbladder were normal. Duodenojejunostomy was performed.

The infant's postoperative course was uneventful and she was discharged in good condition on the 46th day.

DISCUSSION

In the human fetus there is a temporary interruption of the lumen of the duodenum that occurs as the result of epithelial proliferation during the 30th to 60th days of gestation.[11] Vacuoles form within the epithelial plug and coalesce. Recanalization progresses in a cephalocaudad direction, the segment adjacent to the jejunum acquiring its lumen last. Initially the duodenal lumen is recanalized into 2 parallel channels. Into each, the hepatopancreatic duct and accessory pancreatic ducts acquire an opening. Thus, at an early stage of development, 4 duct orifices enter a short segment of the second portion of the duodenum. Subsequently 1 terminal branch of the hepatopancreatic duct and 1 terminal branch of the accessory pancreatic duct regress.[3,10] The zone in which these complex events occur, corresponding to the ultimate sites of the major and minor papillae, measures $\frac{1}{8}$ mm. in length in the 35 day human fetus.[3]

Incomplete recanalization has generally

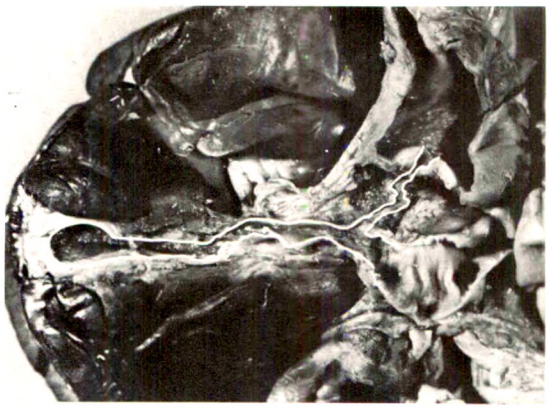


FIG. 3. Case 1. Dissection of bile ducts and duodenum. Bifurcation of common bile duct clearly shown. Pancreatic tissue occupies the interval between the blind ends of the duodenum.

been considered the cause of duodenal atresia and stenosis. Boyden *et al.*[3] suggested that the local "traffic jam" associated with the entry of the hepatopancreatic and accessory pancreatic ducts impeded recanalization of this short segment of the duodenum. In support of this concept, they cited the observation that the site of atresia occurred at the level of the major papilla in 83 per cent of the documented cases of duodenal atresia. In the remainder, the point of obstruction lay distal to this region.

Duodenal atresia may be associated with aberrations of the branching pattern of the hepatopancreatic ducts. Karpa[6] described a newborn with duodenal atresia in whom terminal branches of the hepatopancreatic ducts entered the duodenum above and below the atresia. Subsequent reports by Katz[7] (1 patient), Astley[2] and ourselves (Case 1) bring to 6 the number of patients in whom this complex of anomalies has been demonstrated at autopsy. Persistence of the primitive branching pattern of the hepatopancreatic ducts satisfactorily accounts for the anomalous duct insertions in all these cases (Fig. 5, *A–D*).

In the newborn, the roentgenologic pattern of duodenal dilatation accompanied by gas in undilated loops of small bowel has been considered diagnostic of partial duodenal obstruction (Table 1). It is now evident that this pattern may be encountered with duodenal atresia, if there is a persistent bifid termination of the hepatopancreatic ducts. Although air was not detected in the bile ducts in the 6 patients studied roentgenographically, air presumably entered the hepatopancreatic ducts via a duct orifice rendered incompetent by distention of the obstructed proximal duodenum. Air in bile ducts has been demonstrated roentgenographically[4] and gaseous distention of the gallbladder has been found at surgery in newborns with duodenal atresia.[9]

Contrast studies are generally not needed when typical clinical and roentgenographic features of duodenal atresia are present. Such examinations are valuable in the evaluation of partial duodenal obstruction, particularly to exclude midgut volvulus. In 1 of our patients (Case II) barium meal demonstrated the typical appearance of duodenal atresia; the bile ducts were not opacified.

Since bypass operations are performed to relieve the duodenal obstruction, duct anomalies are generally not detected during surgery for duodenal atresia. Operative cholangiography has not been performed in the 3 patients with anomalous ducts who underwent laparotomy and bypass surgery (Astley's Case 1, our Cases 1 and II). Preoperative intravenous cholangiography could be attempted, but this examination is not feasible in the newborn. In 1 of Astley's cases and in 1 of ours (Case 1) postmortem cholangiography vividly demonstrated the duct system and served as a guide to dissection.

TABLE I

CAUSES OF PARTIAL DUODENAL OBSTRUCTION IN THE NEWBORN

Duodenal stenosis
Duodenal diaphragm (incomplete)
Duplication of duodenum (intramural, intramucosal)
Ladd's bands
Midgut volvulus
Tumor of duodenum
Hematoma of duodenum
Retroperitoneal tumor
Annular pancreas
Preduodenal portal vein

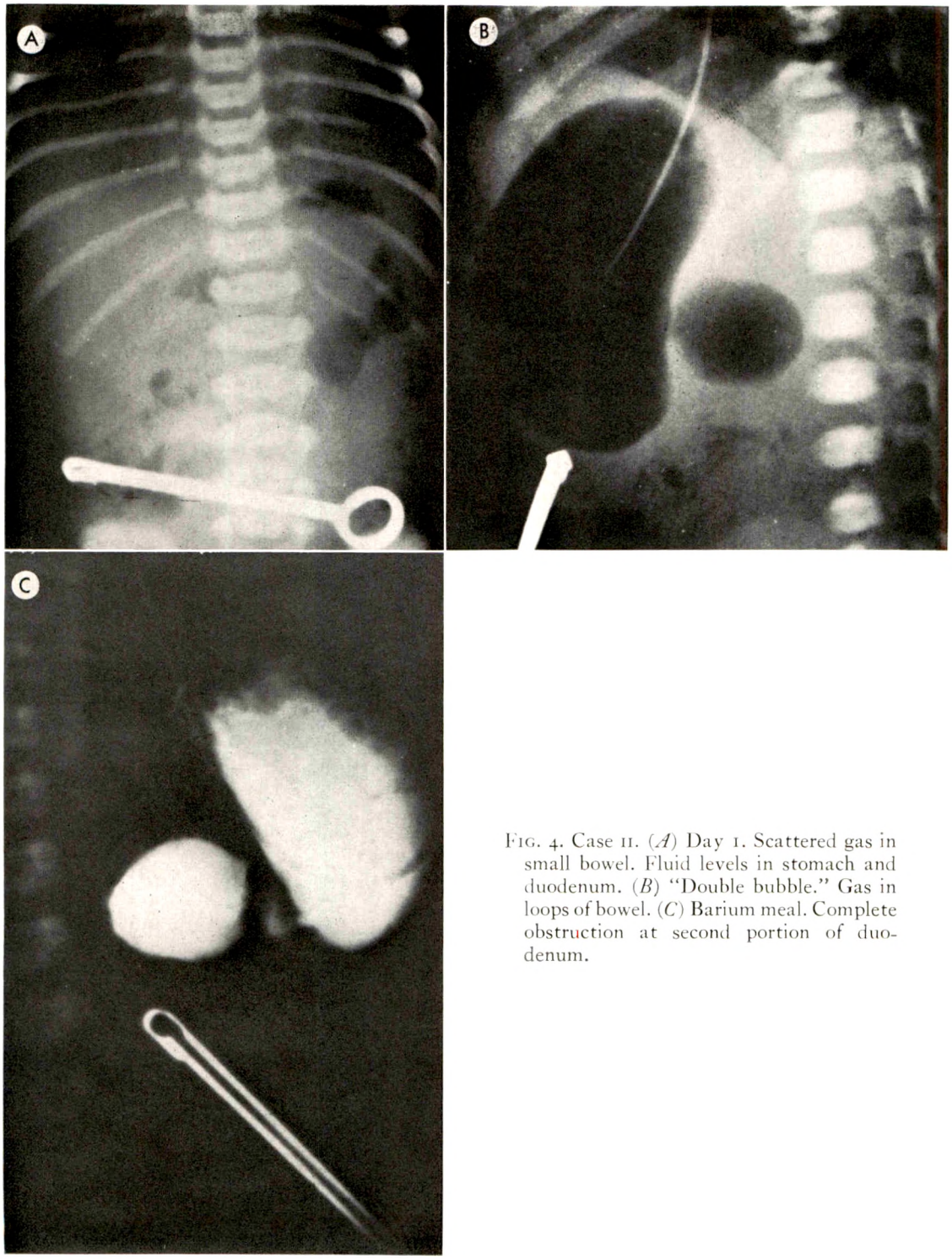


Fig. 4. Case II. (*A*) Day 1. Scattered gas in small bowel. Fluid levels in stomach and duodenum. (*B*) "Double bubble." Gas in loops of bowel. (*C*) Barium meal. Complete obstruction at second portion of duodenum.

The unique coincidence of duodenal atresia and diaphragmatic hernia in 1 of our patients is worthy of mention. This combination of anomalies was not encountered in several extensive reviews of congenital duodenal obstruction.[5,8,12] Diaphragmatic hernia through the foramen of Bochdalek has been attributed to defective

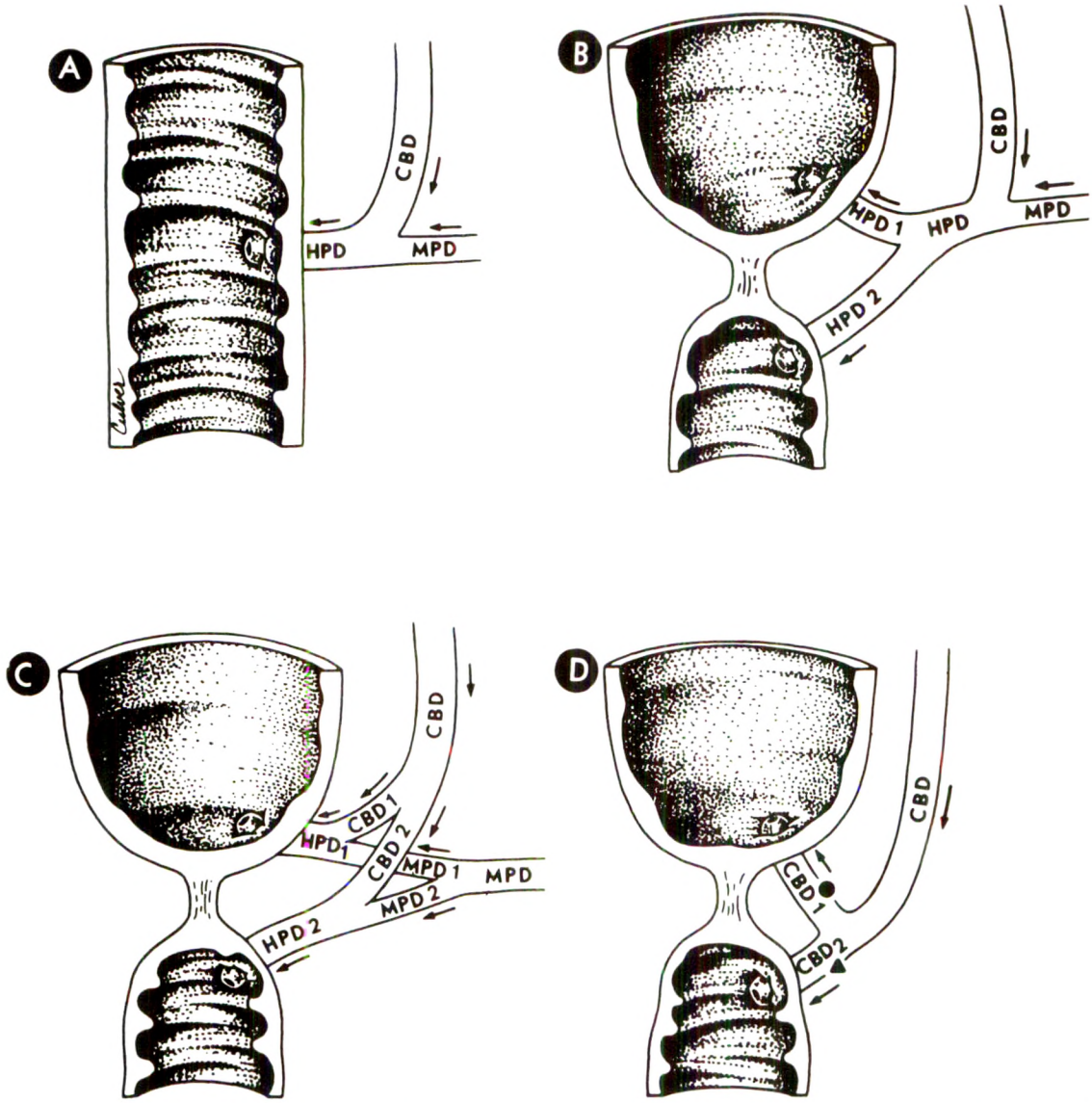


FIG. 5. Anomalies of bile and pancreatic ducts associated with duodenal atresia in 6 reported cases. (*A*) Normal branching pattern. In 60 per cent the main pancreatic duct (MPD) and common bile duct (CBD) join to form hepatopancreatic duct (HPD) which enters the duodenum. In 40 per cent, CBD and MPD enter the duodenum separately. (*B*) Three patients (Astley,[2] 1969). Bifurcation of HPD, 1 branch to proximal duodenum (HPD_1), other branch to distal duodenum (HPD_2). (*C*) One patient (Katz,[7] 1930). Bifurcation of CBD and MPD. CBD_1 and MPD_1 join to form branch to proximal duodenum (HPD_1). CBD_2 and MPD_2 join to form branch to distal duodenum (HPD_2). (*D*) Two patients (our Case 1 and Karpa,[6] 1906). Bifurcation of CBD, one branch to proximal duodenum (CBD_1), other to distal duodenum (CBD_2). In our patient, MPD entered CBD_1 (point of entry indicated by dot). In Karpa's patient, MPD entered CBD_2 (indicated by triangle).

closure of the pleuroperitoneal membrane. Closure normally occurs shortly after the 7th week of gestation,[1] corresponding to the time period during which duodenal recanalization occurs.

SUMMARY

Two newborns with duodenal atresia presented with the roentgenographic findings of duodenal dilatation and gas in the small bowel—a pattern which suggested

partial small bowel obstruction.

In one patient, a bifid termination of the common bile duct (one limb entering above, the other below the point of atresia) was found at autopsy. A similar anomaly was probably present in the other patient but could not be confirmed.

A total of 6 patients have now been described in whom bifid termination of the hepatopancreatic ducts was associated with duodenal atresia; Boyden's theory satisfactorily accounts for this complex of anomalies.

The unique coincidence of diaphragmatic hernia (Bochdalek) and duodenal atresia was present in 1 of our patients.

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REFERENCES

1. Arey, L. B. Developmental Anatomy. Seventh edition. W. B. Saunders Company, Philadelphia, 1965, pp. 284–294.
2. Astley, R. Duodenal atresia with gas below obstruction. *Brit. J. Radiol.*, 1969, *42*, 351–353.
3. Boyden, E. A., Cope, J. G., and Bill, A. H. Anatomy and embryology of congenital intrinsic obstruction of duodenum. *Am. J. Surg.*, 1967, *114*, 190–202.
4. Frates, R. E. Incompetence of sphincter of Oddi in newborn. *Radiology*, 1965, *85*, 875–877.
5. Fonkalsrud, E. W., de Lorimer, A. A., and Hays, D. M. Congenital atresia and stenosis of duodenum: review compiled from members of surgical section of American Academy of Pediatrics. *Pediatrics*, 1969, *43*, 79–83.
6. Karpa, P. Zwei Fälle von Dünndarmatresie. *Virchows Arch. path. Anat. Physiol.*, 1906, *185*, 208–226.
7. Katz, K. Atresie des Duodenums mit Verdoppelung des Ductus choledochus und pancreaticus. *Virchows Arch. path. Anat. Physiol.*, 1930, *278*, 290–294.
8. Nixon, H. H., and Tawes, R. Etiology and treatment of small intestinal atresia: analysis of series of 127 jejunoileal atresias. *Surgery*, 1971, *69*, 41–51.
9. Rosenblatt, M. S. Discussion of paper by Boyden, E., Cope, J. G., and Bill, A. H.[3]
10. Schwengler, R. A. R., and Boyden, E. A. Development of pars intestinalis of common bile duct in human fetus, with special reference to origin of ampulla of Vater and sphincter of Oddi. I. Involution of ampulla. *Anat. Rec.*, 1937, *67*, 441–467.
11. Tandler, J. Zur Entwicklungsgeschichte des menschlichen Duodenums in frühen Embryonalstadium. *Morph. Jahrb.*, 1900, *29*, 187–216.
12. Young, P. G., and Wilkinson, A. W. Abnormalities associated with neonatal duodenal obstruction. *Surgery*, 1968, *63*, 832–836.

POSTOPERATIVE INTUSSUSCEPTION IN CHILDREN*

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INTESTINAL obstruction is a common occurrence in the postoperative patient.

The purpose of this paper is to call attention to the frequency of intussusception as a postoperative complication of the pediatric patient and its nonspecific clinical and roentgen presentation.

CLINICAL MATERIAL

Intussusception has been noted in 4 postoperative patients at Riley Hospital for Children in the last 2 years (Table 1). This does not include patients with the initial diagnosis of intussusception, who developed recurrent disease after reduction. Three of the 4 had surgical procedures involving the abdominal cavity; 1 had revision of ventriculo-atrial shunt tubing in the neck. All had clinical symptoms of mechanical small bowel obstruction, with onset of symptoms 2 to 7 days after the initial surgical procedure. Vomiting was noted in all, and most had abdominal distention and active bowel sounds. Crampy abdominal pain was present in the 1 child over 1 year of age. The classical symptoms of ileocolic intussusception: vomiting, restlessness, and hematochezia, were seen in only 1 patient. None had a palpable abdominal mass. Abdominal roentgenograms showed definite mechanical small bowel obstruction in 3 children, and probably obstruction in the fourth (Fig. 1, *A* and *B*). Mass lesions and other roentgen signs of intussusception were not detected.

Laparotomy was performed 1 to 10 days

TABLE I

SUMMARY OF CLINICAL AND ROENTGEN DATA ON THE 4 PATIENTS WITH POSTOPERATIVE INTUSSUSCEPTION

Age and Sex	Initial Diagnosis	Initial Operation	Onset of Symptoms Post-operative	Signs/Symptoms	Interval Between Initial Symptoms and Next Operation	Roentgenograms	Operation
10 mo. F	Hepatic cyst	Resection of left lobe of liver	7 da.	Vomiting, distention; bowel sounds present; stools normal on previous day	1 da.	KUB—low small bowel obstruction; no mass	Reduction of ileocolic intussusception
5 mo. M	Hydronephrosis with prostatic valves	Bilateral cutaneous ureterostomies (extraperitoneal)	2 da.	Vomiting, distention; bowel sounds present; few loose stools; no hematochezia	3 da.	KUB—low small bowel obstruction; no mass	Reduction of ileo-ileal intussusception
7 yr. M	Myelomeningocele with neurogenic bladder	(1) Ileal conduit for urinary diversion (2) Release of adhesions from first surgery 16 days later	4 da. after second laparotomy	Crampy abdominal pain, vomiting, stools normal; bowel sounds present	10 da.	KUB—high small bowel obstruction; no mass; barium meal examination confirmed above	Reduction of jejuno-jejunal intussusception
5 mo. F	Hydrocephalus	Revision of ventriculo-atrial shunt in neck	6 da.	Vomiting, distention; "currant jelly" stools; bowel sounds present	1 da.	KUB—probable small bowel obstruction; barium enema examination shows ileocolic intussusception	Reduction of ileocolic intussusception

KUB = kidney, ureter, bladder.

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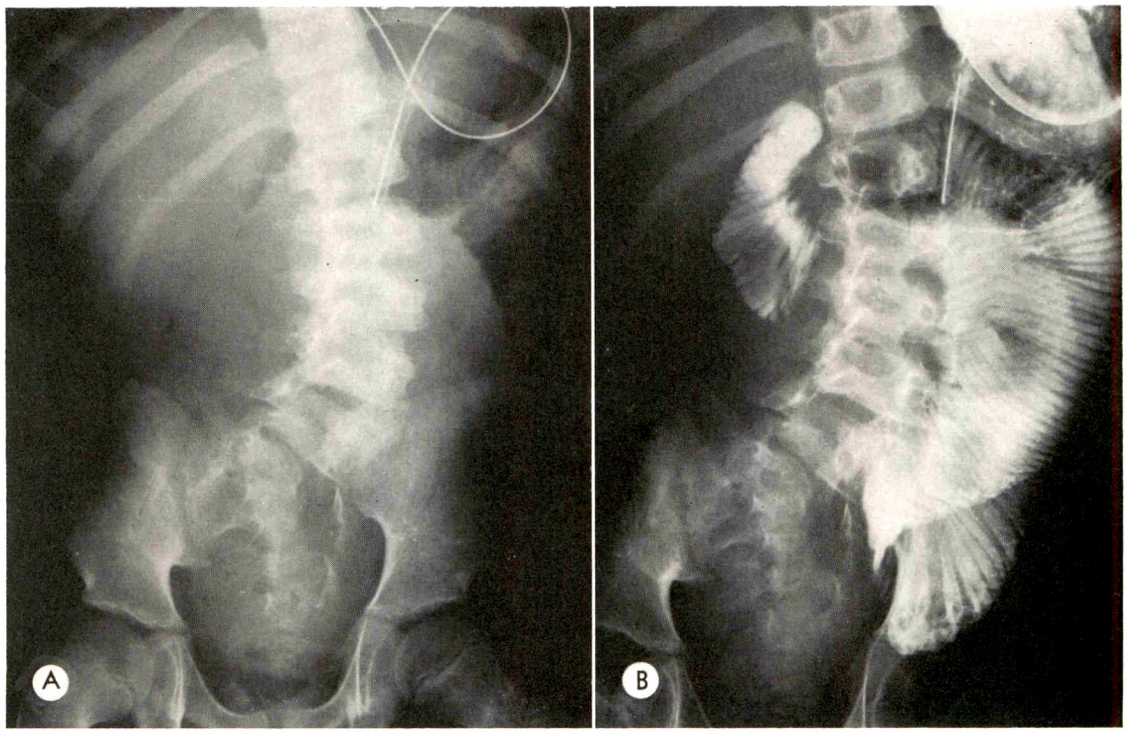


Fig. 1. (*A*) Abdominal roentgenogram in a 7 year old male 14 days after laparotomy for lysis of intestinal adhesions incident to urologic surgery. Dilated small bowel suspicious of high small bowel obstruction is noted. (*B*) Small bowel examination the same day confirms high small bowel obstruction, with the character of the obstruction not evident. A jejuno-jejunal intussusception was reduced at laparotomy.

after the onset of symptoms. Intussusception was found in all cases. Two cases were ileocolic, one jejuno-jejunal, and one ileo-ileal. Bowel resection was not required, and all patients did well after surgical reduction of the intussusception.

DISCUSSION

Intussusception is a recognized postoperative complication in pediatric surgery with an estimated frequency of 1 case per year in a children's hospital.[6] In one series[12] it was responsible for 13 per cent of postoperative bowel obstructions in children. Most cases occur after abdominal surgery, but it has been reported after extraabdominal surgical procedures,[4] as was noted in 1 patient in our series. The vast majority of the reported cases have been ileo-ileal intussusceptions,[1,7,11,12,15] but the ileocolic type has also been seen.[6,7]

Clinical indications of postoperative ileo-ileal intussusception are nonspecific, *i.e.*, only signs of mechanical small bowel obstruction are present. Delay in definitive surgery is frequent because the diagnosis of adhesions with subsequent obstruction is the foremost consideration.[12] If ileocolic intussusception occurs the clinical signs, particularly hematochezia, may suggest the diagnosis of intussusception, as 1 of our cases demonstrated.

Roentgenograms in the cases reported here showed evidence of mechanical small bowel obstruction in all cases, with definite signs in 3 of the 4. Similar roentgen findings have been noted in other series.[7,11] Roentgen evidence specific for intussusception was not present, and mass lesions were not detected on the roentgenograms. Rigler and Godfrey[14] describe a "bicycle seat" sign of gas proximal to an ileo-ileal intussusception, and Foulds and Beamish[5] refer to a similar "nipple" of gas at the same site. Contrast studies may show identical narrowing of the barium column as it enters the intus-

suscepted loop.[2] These roentgen signs, which are specific for intussusception, were not present in our patients. Others[9] have commented on the frequent lack of specific signs of intussusception on abdominal roentgenograms.

The prognosis of postoperative ileo-ileal intussusception is not as grave as in those patients with specific causes of the same entity, *e.g.*, Meckel's diverticulum.[15] Gangrene was not present in any of our cases and was reported in only 3 of 40 cases recently reviewed.[6]

Postoperative intussusception in the adult is related to the type of operation: jejunogastric intussusception after gastrectomy,[10] and cecocolic intussusception following appendectomy.[3] The etiology of intussusception in the pediatric patient is obscure; one theory is that it is related to disorganized peristalsis following recovery from ileus.[7] A relationship of chronic renal disease with postoperative intussusception has been noted by Ravitch,[13] but the reason is unclear.

The postoperative child with bowel obstruction differs considerably from a comparable adult. Metabolic changes are profound,[12] intussusception is frequent, and occult gastrointestinal anomalies may be present.[8] The pediatric patient who will respond to conservative measures is infrequent.[12] If mechanical obstruction is indicated by clinical signs and confirmed by roentgen studies, further observation should not be prolonged before performing a laparotomy. If there is clinical or roentgen doubt of the presence of obstruction, oral barium studies are necessary. If symptoms of ileocolic intussusception are present, a diagnostic barium enema examination may be performed.

SUMMARY

Intussusception, especially of the ileo-ileal type, is a frequent cause of bowel obstruction in the postoperative child.

The clinical and roentgen signs of this complication are those of mechanical small bowel obstruction without specific evidence of intussusception.

Aggressive therapy, *i.e.*, laparotomy, is indicated in the postoperative child with definite bowel obstruction after a brief period of conservative management.

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REFERENCES

1. Brown, P. M., and Thornfeldt, R. Intussusception in early postoperative period. *A.M.A. Am. J. Dis. Child.*, 1964, *107*, 297–299.
2. Carlson, H. C. Small intestinal intussusception: an easily misunderstood sign. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *110*, 338–339.
3. Danielson, K. S. Cecocolic intussusception—postoperative complication of appendectomy. *New England F. Med.*, 1969, *280*, 35–36.
4. Dodrill, F. D., and Benson, C. D. Coarctation of aorta with both subclavian arteries arising from distal segment complicated by postoperative intussusception. *Surgery*, 1962, *51*, 809–813.
5. Foulds, D. M., and Beamish, W. E. Plain film diagnosis of acute ileo-ileal intussusception. *J. Canad. A. Radiologists*, 1970, *21*, 178–180.
6. Guttman, F. M., Ducharme, J. C., and Collin, P. P. Intussusception after major abdominal operations in children. *Canad. J. Surg.*, 1970, *13*, 427–433.
7. Hays, D. M. Intussusception as postoperative complication in pediatric surgery. *Surg., Gynec. & Obst.*, 1961, *112*, 583–589.
8. Kark, W. Postoperative intestinal obstruction in infants and children. *South African M. J.*, 1961, *35*, 211–213.
9. LeVine, M., Schwartz, S., Katz, I., Burko, H., and Rabinowitz, J. Plain film findings in intussusception. *Brit. J. Radiol.*, 1964, *37*, 678–681.
10. Mason, L. B., Williams, R. W., and Marshburn, E. T. Retrograde jejuno-gastric intussusception following gastrectomy. *A.M.A. Arch. Surg.*, 1960, *81*, 485–491.
11. McGovern, J. B., and Gross, R. E. Intussusception as postoperative complication. *Surgery*, 1968, *63*, 507–513.
12. Raffensperger, J. G., and Baker, R. J. Postoperative intestinal obstruction in children. *A.M.A. Arch. Surg.*, 1967, *94*, 450–459.
13. Ravitch, M. M. Intussusception in Infants and Children. Charles C Thomas, Publisher, Springfield, Ill., 1959.
14. Rigler, L. G., and Godfrey, H. W. Roentgen diagnosis of ileo-ileal intussusception. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1958, *79*, 837–842.
15. Stevenson, E. O. S., Hays, D. M., and Snyder, W. H. Postoperative intussusception in infants and children. *Am. J. Surg.*, 1967, *113*, 562–566.

CHOLEDOCHAL CYST DEMONSTRATED BY I^{131} ROSE BENGAL SCANNING*

REPORT OF A CASE

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CHOLEDOCHAL cyst is a sharply defined dilatation of the common bile duct. A definitive diagnosis can be made if oral cholecystography or intravenous cholangiography is successfully accomplished. Silberman and Glassner's[3] review of the literature revealed only 3 of 405 cases of cystic dilatation of the bile duct demonstrated by oral cholecystography; they added 2 new cases demonstrated by intravenous cholangiography. Nonvisualization of the cyst or gallbladder was even postulated as one of the diagnostic features by some authors.[2]

The purpose of this communication is to describe a case in which I^{131} rose bengal scanning established the diagnosis of choledochal cyst. The fact that none of the previously reported cases of choledochal cyst have been demonstrated by means of I^{131} rose bengal scanning prompted us to describe this case.

REPORT OF A CASE

An 11 year old Japanese female presented with a right upper abdominal mass. She had been well until 2 months previously, when she experienced abdominal pain of 5 days' duration. Three weeks prior to admission, she noticed an abdominal mass in the right upper quadrant and an acholic stool. Two weeks later she felt a severe abdominal pain in the right hypochondrium.

On physical examination the patient appeared well nourished and normally developed. A large, 8×14 cm., nontender mass was palpable below the right costal margin. The mass was felt to be hard without cystic fluctuation.

The urine gave a ++ test for bilirubin. The hematocrit was 39.5 per cent; the blood white cell count was 5,800 per $mm.^3$, with normal differentials. The total serum protein was 7.5 gm. per 100 ml., with an albumin-globulin ratio of 1:3. The thymol turbidity test was 2.2 units, zinc turbidity test 3.7 units, the icteric index 20, the glutamic oxalacetic transaminase 381 units, the glutamic pyruvic transaminase 246 units, the alkaline phosphatase 41 King-Armstrong units, and the lactic acid dehydrogenase 561 units. The total bilirubin was 3.7 mg., with the direct bilirubin 2.4 mg. per 100 ml.

During hospitalization the mass seemed to increase in size, and the patient became icteric. On the eighth hospital day the icteric index was 36, the total bilirubin 6.2 mg. per 100 ml. with direct bilirubin 4.6 mg.

A survey roentgenogram of the abdomen revealed a large soft tissue mass occupying the right upper quadrant. The kidneys and psoas muscle outlines seemed to be preserved. On celiac angiography the common and proper hepatic arteries, the portal vein, and the normal-sized gallbladder were displaced upward by an extrahepatic avascular mass (Fig. 1, *A* and *B*). There was no deviation of the abdominal aorta on aortography. The intravenous pyelographic phase of the angiography was normal. The upper gastrointestinal examination revealed compression and marked anterior displacement of the second portion of the duodenum (Fig. 2, *A* and *B*).

On the ninth hospital day an excretory hepatoscintigraphy using I^{131} rose bengal was performed. Immediately after the intravenous administration of 250 μc I^{131} rose bengal, a large mass lesion around the porta hepatis was recognized as a negative finding (Fig. 3*A*). Three hours later there was minimal activity in the space. An 18 hour scan disclosed the mass in question, filled with the rose bengal excreted from the liver (Fig. 3*B*).

On the eleventh hospital day surgery was performed. A choledochal cyst containing 1,200 ml. of bile was removed and choledochojejunostomy (Roux-Y) was accomplished. Immediate normalization of the laboratory findings occurred, and her postoperative course was uneventful.

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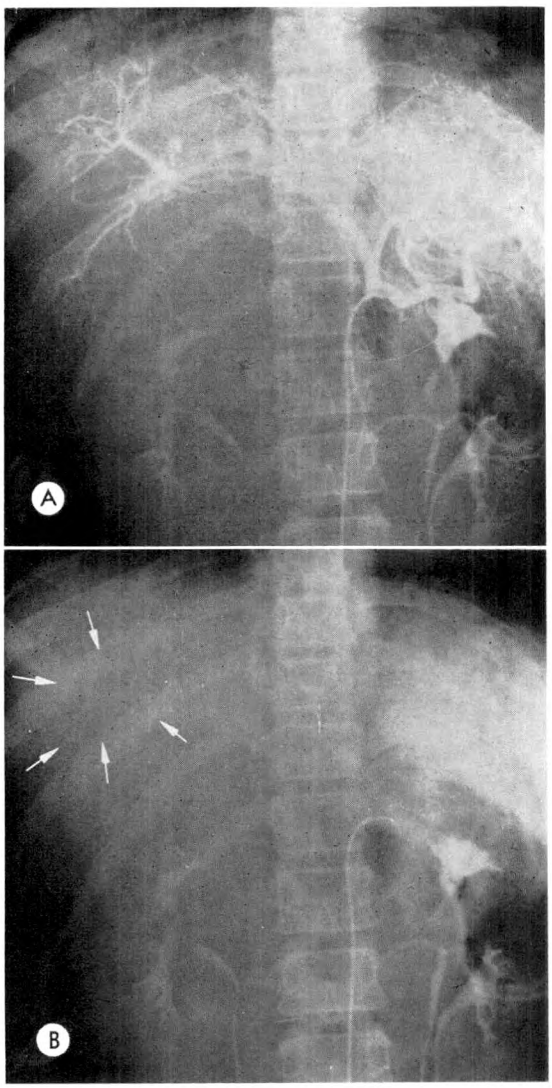


FIG. 1. Celiac angiograms. (*A*) The common and proper hepatic arteries are displaced upward and the gastroduodenal artery medially in the arterial phase. (*B*) The gallbladder (arrows) is shown in the capillary phase.

DISCUSSION

This case represents a typical presentation of choledochal cyst. The correct diagnosis was made by a radioiodinated rose bengal scan preoperatively. The usefulness of I^{131} rose bengal liver scans in the differential diagnosis of jaundice has been previously described.[1] The superiority of the rose bengal scan to the cholecystographic method in visualizing the gallbladder has also been described.[1] Unless there is a complete obstruction to bile flow, the isotope will be found in the small bowel. Nontoxicity of the substance and the higher yield of the bile duct visualization are highly favorable in jaundiced patients. Those pa-

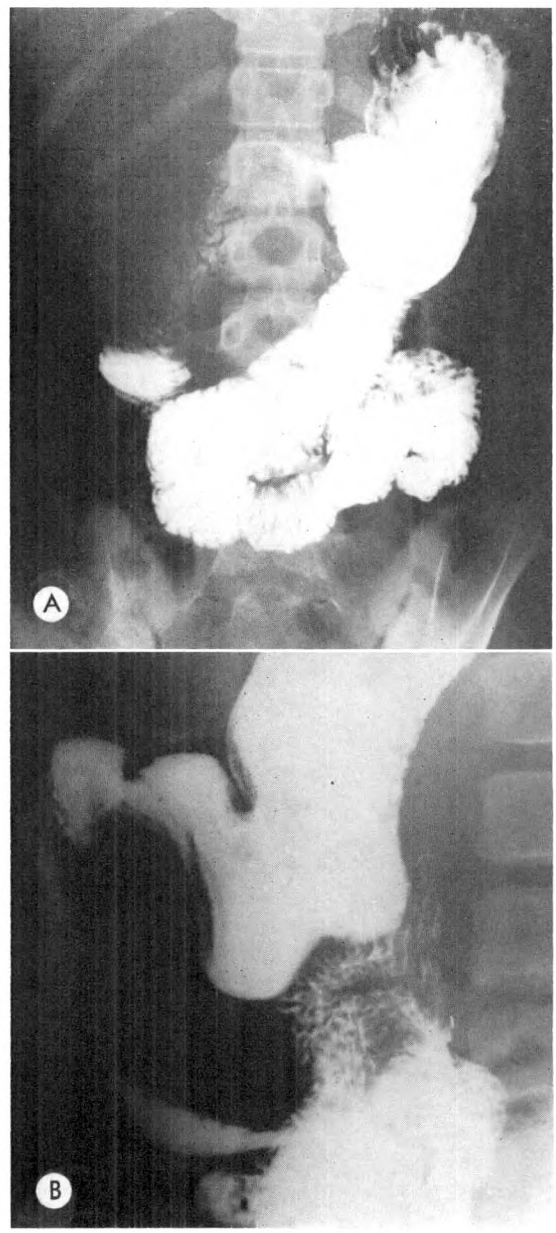


FIG. 2. Upper gastrointestinal series. The second portion of the duodenum is markedly flattened and displaced anteriorly in (*A*) the frontal projection, and (*B*) the lateral projection.

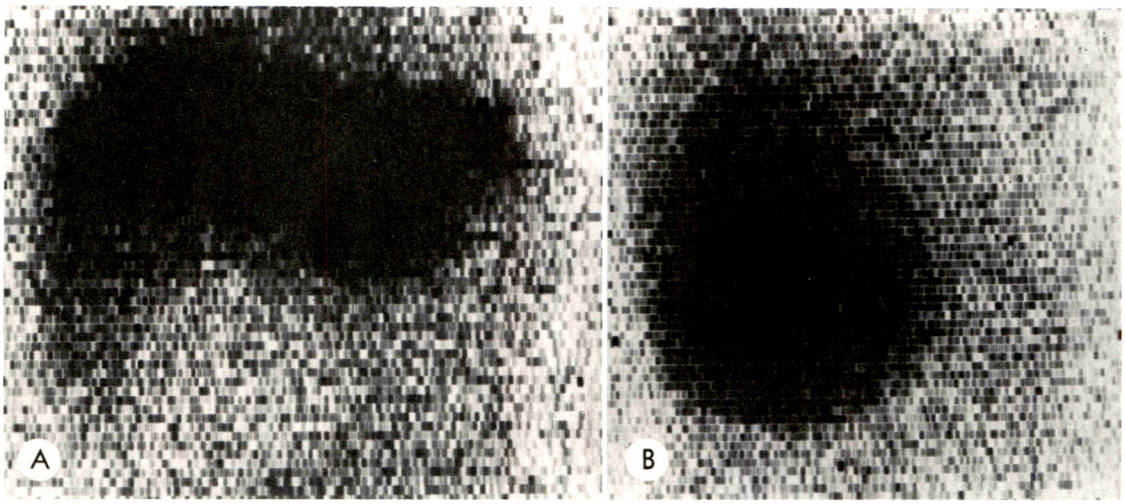


Fig. 3. I^{131} rose bengal scans. (*A*) A round mass is shown as negative immediately after the injection. (*B*) The mass is filled with radioactive rose bengal, 18 hours later.

tients with a poorly functioning liver should not be exposed to the risk of renal shutdown by using multiple doses of contrast material in visualizing the gallbladder.[4]

According to Wise,[5] if the serum bilirubin value is above 4 mg. per 100 ml., opacification of the biliary ducts by intravenous cholangiography may be expected in only 9 per cent of the cases, and, if the bilirubin level rises, the probability of opacification decreases. These facts are quite compatible with the statement that a cholecystographic examination during the quiescent phase between intermittent jaundice in choledochal cyst may produce a higher percentage of specific diagnoses.[3]

In the present case, I^{131} rose bengal scanning was performed during the attack of jaundice, when the serum bilirubin was acutely increasing, yet a perfect visualization of the choledochal cyst was obtained.

SUMMARY

A case of choledochal cyst is reported in an 11 year old female.

The correct diagnosis was made on I^{131} rose bengal scanning.

The value of the procedure is discussed in a comparison with the cholecystographic techniques.

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REFERENCES

1. Eyler, W. R., Schuman, B. M., DuSault, L. A., and Hinson, R. E. Radioiodinated rose bengal liver scan as aid in differential diagnosis of jaundice. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1965, *94*, 469–476.
2. Shiraki, K., and Okamoto, Y. Idiopathic common bile duct dilatation: report of 9 cases in reference to symptoms and x-ray diagnosis in infancy and childhood. *J. Pediatric Practice* (in Japanese), 1962, *25*, 292–303.
3. Silberman, E. L., and Glassner, T. S. Roentgen features of congenital cystic dilatation of common bile duct: report of two cases. *Radiology*, 1964, *82*, 470–475.
4. Whitehouse, W. M. Roentgenology of biliary tract. In: Alimentary Tract Roentgenology. Edited by A. R. Margulis and H. J. Burhenne. Volume 2. C. B. Mosby Company, St. Louis, 1967.
5. Wise, R. E. Intravenous cholangiography. In: Alimentary Tract Roentgenology. Volume 2. Edited by A. R. Margulis and H. J. Burhenne. C. V. Mosby Company, St. Louis, 1967.

CLINICAL APPLICATIONS OF CISTERNOGRAPHY IN THE PEDIATRIC AGE GROUP*

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THE concept of subarachnoid injection of a radiopharmaceutical for the evaluation and imaging of cerebrospinal fluid (CSF) has recently received widespread attention.[2,3,7] We have utilized this diagnostic technique in children for the evaluation of hydrocephalus, to determine the patency of CSF diversionary shunts, and for evaluation of cerebrospinal fluid leaks and abnormal communications with the CSF space (meningomyelocele, subdural hygroma, and spinal syrinx).

The purpose of this paper is to review our experience with cisternography in children emphasizing certain technical and clinical considerations.

METHODOLOGY

Cisternography, when properly performed, is a relatively innocuous and extremely valuable diagnostic procedure.[4,11] The site of injection and the choice of radiopharmaceutical depend upon the indications for the study and the questions to be answered.

There is no one "best" radiopharmaceutical at present for cisternography. A "good" radiopharmaceutical is one that is confined to the cerebrospinal fluid space during the time of the study, but is rapidly eliminated from the body after images are obtained.[11] It should have a high photon yield per rad patient dose and should cause no alteration of the CSF dynamics. Energy of the gamma photon emission should be in the proper range for diagnostic images on detectors in current use (100–400 kev., optimally 120–140 kev.).[5] The pharmacologic and chemical properties of the tracer should not cause significant changes in the meninges, nerve roots, spinal cord, and brain. Movement of the radiopharmaceutical should mimic CSF flow.

To investigate patients with hydrocephalus or abnormal CSF space communications, delayed images must often be obtained at 24, 48 and even 72 hours. For these studies, the radionuclide labels of I^{131}, Yb^{169} and In^{111} are most appropriate because of their physical half life and acceptable radiation dose to the patient. Studies obtained to detect CSF leaks can be accomplished with Tc^{99m} labelled radiopharmaceuticals. Considerations of reduction of patient radiation dose make Tc^{99m} the preferred radionuclide in certain studies because of the relatively short physical (6 hours) half life. CSF diversionary shunts may be injected with Tc^{99m} pertechnetate if patency of the apparatus is the only consideration.[6]

For cisternographic imaging, the radiopharmaceutical should be confined to the CSF space for an adequate time period. Higher molecular weight substances (serum albumin, M.W. 70,000, and DTPA complex, M.W. 600) have been utilized because they do not rapidly transfer across the subarachnoid membrane.[10] An advantage of the DTPA chelate is that it is removed from the body as a glomerular substance once it leaves the CSF space.

Improper injection of the radiopharmaceutical is the most common cause of inadequate and misleading cisternographic studies. Subdural and epidural injections and

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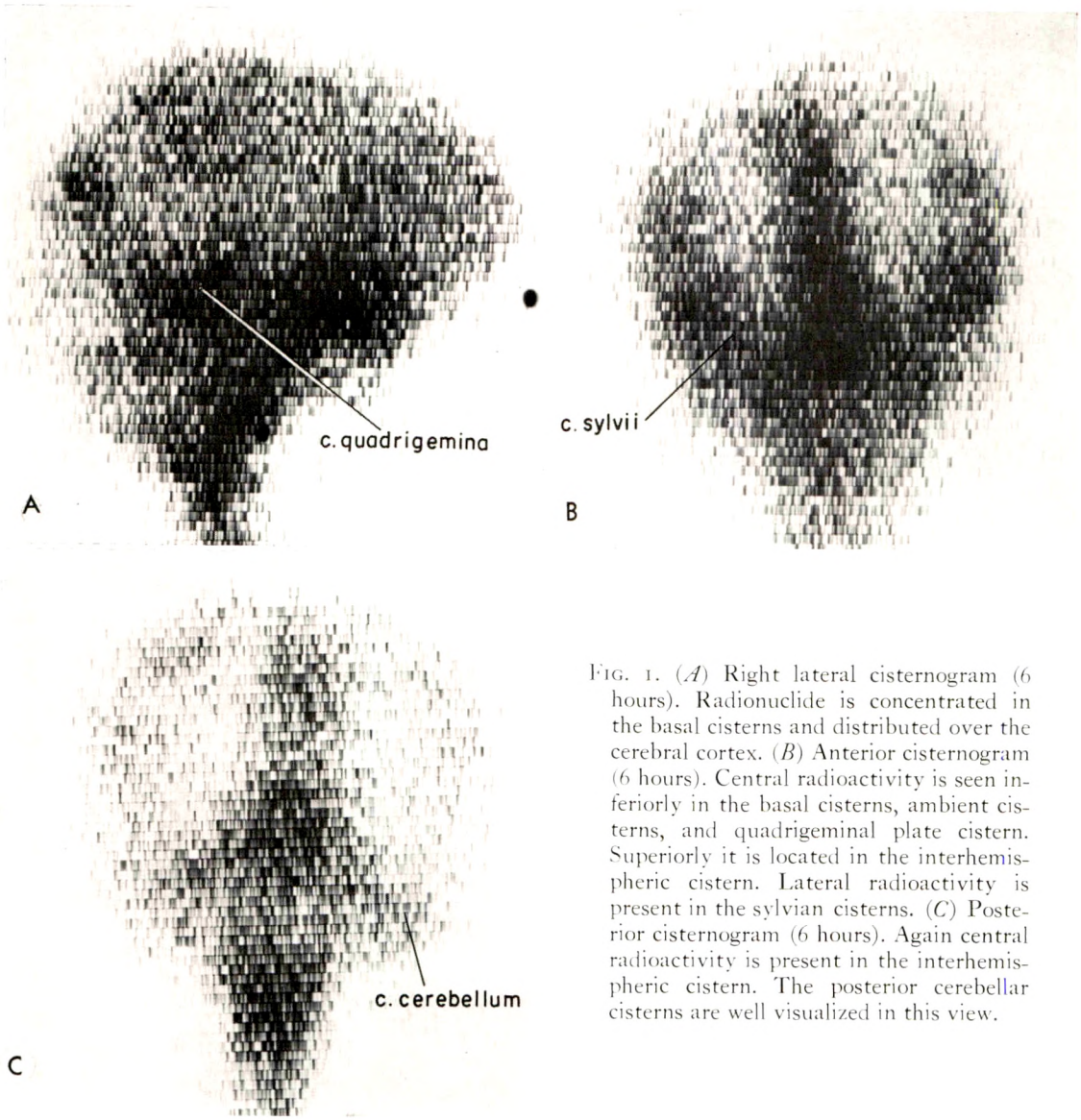


Fig. 1. (*A*) Right lateral cisternogram (6 hours). Radionuclide is concentrated in the basal cisterns and distributed over the cerebral cortex. (*B*) Anterior cisternogram (6 hours). Central radioactivity is seen inferiorly in the basal cisterns, ambient cisterns, and quadrigeminal plate cistern. Superiorly it is located in the interhemispheric cistern. Lateral radioactivity is present in the sylvian cisterns. (*C*) Posterior cisternogram (6 hours). Again central radioactivity is present in the interhemispheric cistern. The posterior cerebellar cisterns are well visualized in this view.

significant subarachnoid extravasation at the injection site invalidate the examination. To minimize this problem, we do not undertake cisternographic studies until several days after a lumbar puncture and at least a week after a pneumoencephalography. For the lumbar puncture a small bore spinal needle is preferred. Several methods have been advocated to decide early in the study whether or not a proper injection has been made. We have found it practical to image the spinal area approximately 30-60 minutes after injection to detect extravasation at the injection site or cessation of radiopharmaceutical movement. Monitoring transfer from the subarachnoid space into the blood or urine is an even more sensitive method.

Multiple views at selected time intervals are necessary to document CSF movement. Entry of the radiopharmaceutical into the ventricles is abnormal and is the critical observation in patients with communicating hydrocephalus. Decision as to whether or not radioactivity is in the lateral ventricles depends upon recognition of the nor-

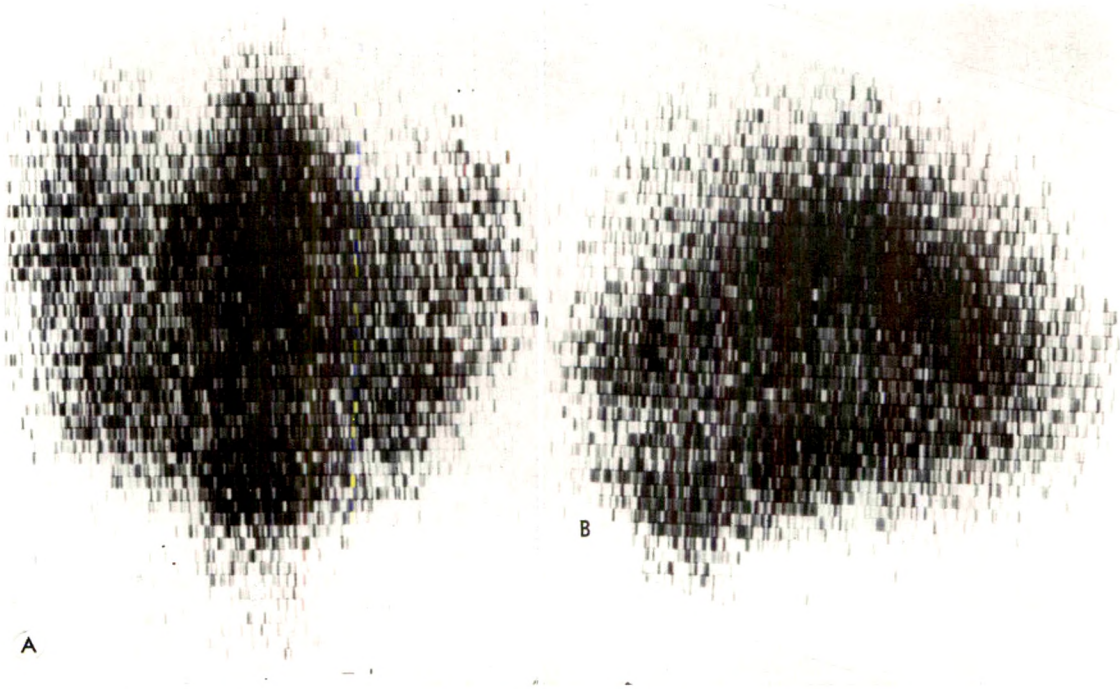


Fig. 2. (*A*) Anterior cisternogram (6 hours). Abnormal broad band of central accumulation of radiopharmaceutical. This radionuclide is in the lateral ventricles. (*B*) Right lateral cisternogram (6 hours). "C" shaped configuration of radioactivity represents radiopharmaceutical concentration in the lateral ventricles.

mal configuration of the radionuclide in the cisterns. Therefore, routine lateral, anterior and posterior views are obtained (Fig. 1, *A–C*). Normally, a linear band of radioactivity is seen centrally. When there is entry of the radiopharmaceutical into the ventricles, this central radioactivity appears wider than the normal linear band and is somewhat heart shaped in the anterior view and C shaped in the lateral view (Fig. 2, *A* and *B*). Occasionally, special views such as the vertex view must be obtained to establish that a patient has ventricular activity (Fig. 3, *A* and *B*).

After ventricular activity is seen, retention of activity or stasis in the ventricles must be determined. Those patients with radioactivity remaining in the lateral ventricles at 24 hours appear to have better results from CSF diversionary shunts than patients with transient ventricular activity.[7]

In determining the function and patency of shunts, flow of the radiopharmaceutical from the distal shunt tip is evidence of a functioning shunt (Fig. 4).[1] Radioactive material injected into the lumbar area can enter the lateral ventricles due to their enlargement even in the presence of a diversionary shunt that is not functioning. This entry of the radiopharmaceutical into the ventricles may be due to simple diffusion, but probably reflects participation of the ventricle in CSF absorption.

To detect CSF leaks, greater reliance should be placed upon nasal pack counts than upon images. The amount of radioactivity necessary to cause a significantly increased count is far less than that necessary to create a detectable abnormality on the cisternographic image. However, when the abnormality is seen on the cisternogram, additional anatomic information is gained.

We obtained multiple images to document migration of the radioactive material and have correlated the patterns of migration at time intervals of 2, 6 and 24 hours. With

specific indications, images are obtained at 48 and 72 hours. The flow pattern of radioactivity may be so characteristic that a diagnosis can be made on the basis of the cisternogram.

Normally, a radiopharmaceutical introduced into the lumbar subarachnoid space will migrate to the basal cisterns and be imaged there on the 2 hour study. At 6 hours, the radionuclide should reach the cerebral hemispheres and is often to the parasagittal area. The 24 hour image normally demonstrates homogeneous distribution of activity over the cerebral hemispheres accompanied by little or no activity in the basal cisterns. It is not as often concentrated in the area of the arachnoid granulations as in adults.

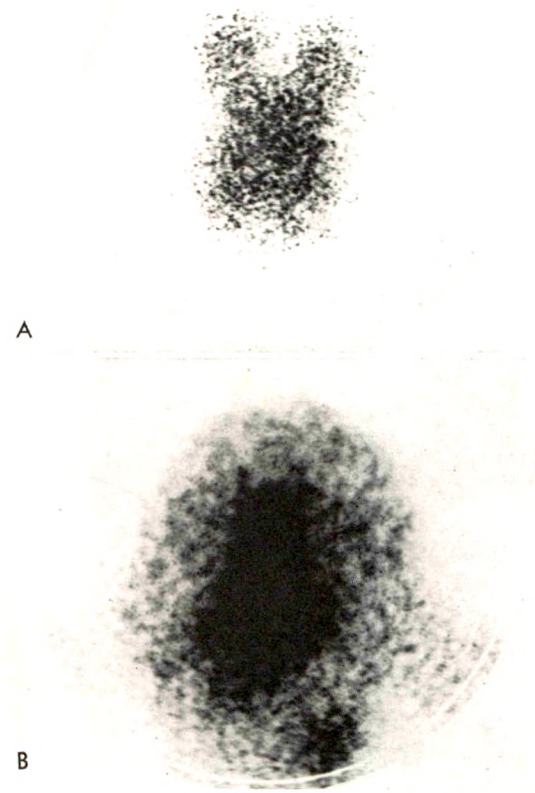


Fig. 3. (*A*) Vertex cisternogram (2 hours). Radionuclide present in the anterior and temporal horns of the lateral ventricles. No cisternal activity is seen peripherally. (*B*) Vertex cisternogram (6 hours). Peripheral activity in cerebrospinal fluid (CSF) space around the cerebral hemispheres. Central radionuclide accumulation is in the lateral ventricles.

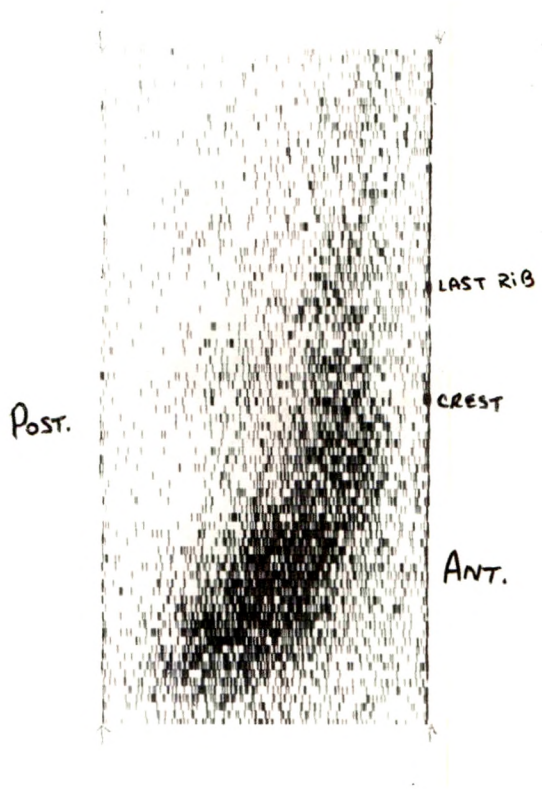


Fig. 4. Lateral cisternogram (abdominal view over distal shunt tip; anterior aspect on reader's right). Flow of radiopharmaceutical from CSF space out distal shunt confirms shunt patency.

Serial urine and plasma samples to monitor absorption of the radiopharmaceutical from the CSF space and elimination from the body should also be obtained.

CLINICAL EXPERIENCE AND REPORT OF CASES

Over one-half of the cisternograms requested on children in our institution were obtained for evaluation of hydrocephalus.[11] In communicating hydrocephalus (obstructive type) there is extraventricular obstruction to the cerebrospinal fluid flow, usually in the basal subarachnoid space or over the cerebral hemispheres. The cisternographic

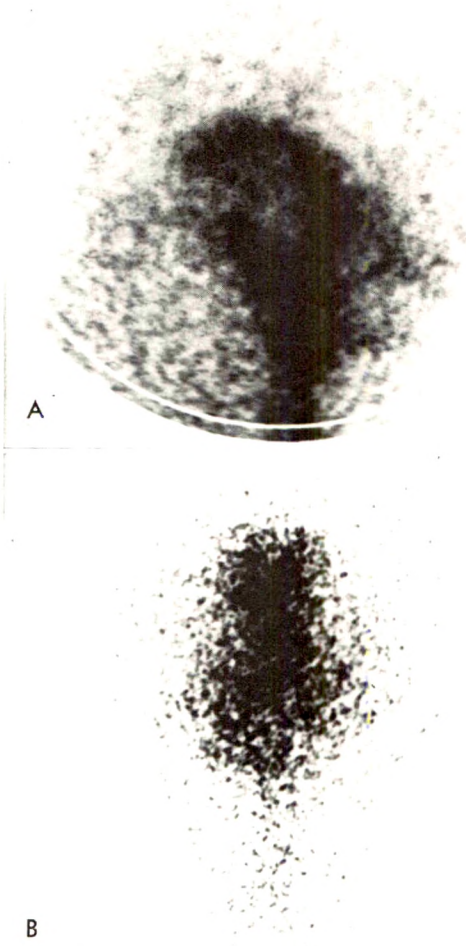


Fig. 5. Case I. (*A*) Lateral and (*B*) anterior cisternogram (6 hours). "C" shaped concentration of radioactivity centrally is in the lateral ventricles diagnostic of communicating hydrocephalus.

hallmark of communicating hydrocephalus is entry of the radionuclide into the lateral ventricles.[4,10] In addition, there may be no radioactivity over one or both cerebral hemispheres (Fig. 5, *A* and *B*; and 6, *A* and *B*).

Case I. B.R., (JHH 110 50 21), a 13 year old girl, was admitted to the Johns Hopkins Hospital at age 11 because of the neurologic and ocular manifestations of craniopharyngioma. The tumor was so large that total excision could not be performed. Instead, the tumor mass was aspirated and a catheter placed between the tumor sac and the oropharynx.

Six years after surgery, the patient developed bacterial meningitis. The cisternography was performed to determine if there was communicating hydrocephalus. It showed radioactivity in the ventricles at 6 hours, indicating communicating hydrocephalus (Fig. 5, *A* and *B*).

Case II. S.H., (JHH 130 89 18), a 22 month old male infant, was admitted to the Johns Hopkins Hospital for evaluation of large head size and delayed development. The cisternography was performed to determine if the child had communicating hydrocephalus (Fig. 6, *A* and *B*). It showed ventricular activity at 6

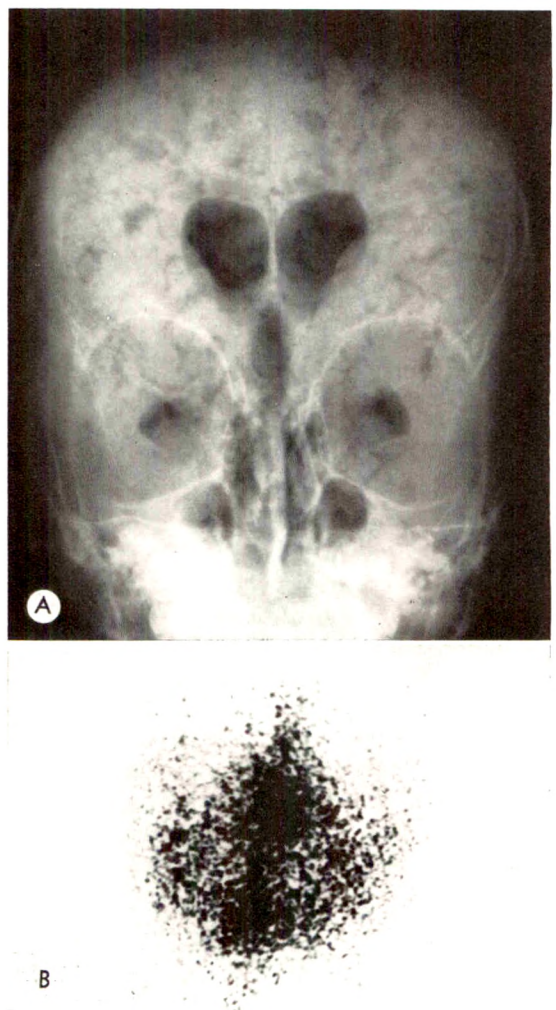


Fig. 6. Case II. (*A*) Anteroposterior pneumoencephalogram (PEG) and (*B*) anterior cisternogram at 6 hours. Dilated lateral ventricles are shown by PEG. The ventricular entry of radiopharmaceutical is characteristic of communicating hydrocephalus.

hours and indicated communicating hydrocephalus.

In patients with obstructive hydrocephalus, the cisternogram may appear normal because failure of the radiopharmaceutical to enter into the lateral ventricles is normal.[11] If the accompanying increased intracranial pressure is great enough, the basal cisterns may be obliterated, causing obstruction to cerebrospinal fluid flow peripherally.

The second most common indication for cisternography is evaluation of cerebrospinal fluid leak caused by trauma or surgery. We do not rely upon scanning to reveal the source of the leak, but use cotton pledgets placed in the ears and nostrils which are counted in a well chamber. If the scan happens to show the source of the leak, additional confirmatory and anatomic information is gained (Fig. 7).

Case III. N.M., (JHH 133 32 77), a 16 year old girl with Cruzon's disease, underwent plastic surgery for reconstruction of her face. Her convalescence was complicated by cerebrospinal rhinorrhea. The cisternography was performed to determine the site of the leak. It demonstrated an anterior leak which was later shown to be through the cribriform plate (Fig. 7).

Another area in which cisternography has proven useful is the evaluation of shunt patency in patients with communicating hydrocephalus.[12] The radiopharmaceutical

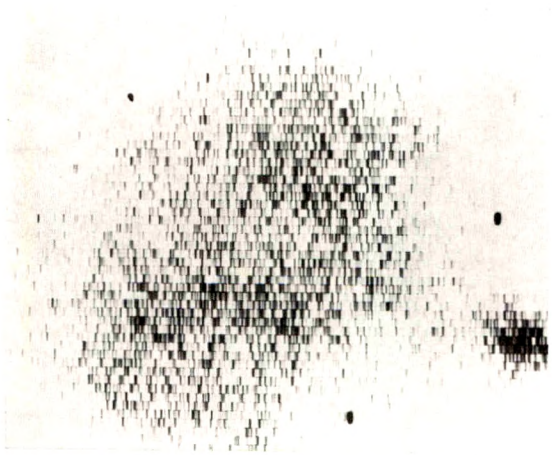


Fig. 7. Case III. Lateral cisternogram (24 hours). Anterior accumulation of radioactivity demonstrates CSF leak through the cribriform plate which was later surgically repaired.

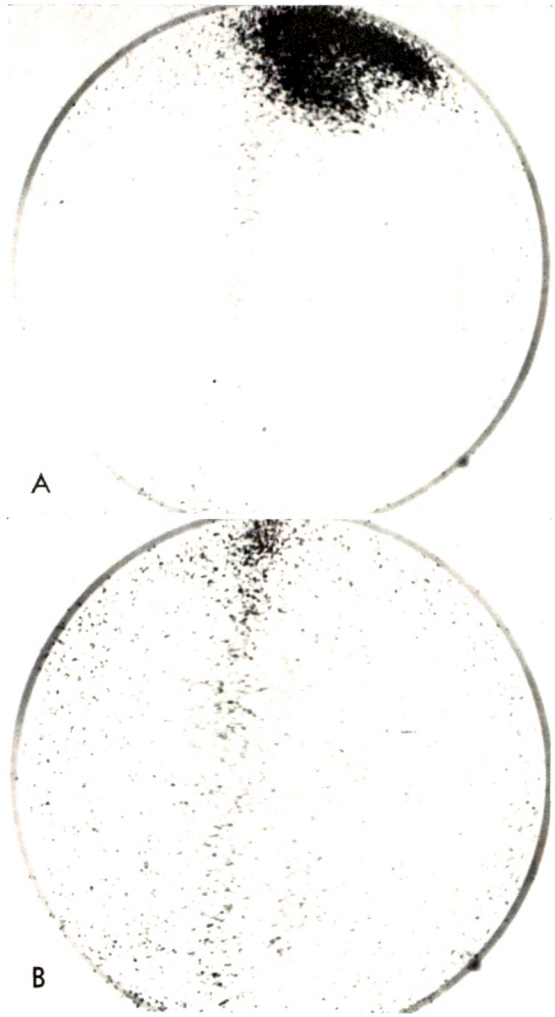


Fig. 8. Case IV. (*A*) Lateral cisternogram of vertebral column and cranium after lumbar injection of radiopharmaceutical. Spinal and lateral ventricular activity is seen. (*B*) Cisternogram over distal shunt tip (abdominal peritoneum). Radioactivity is present in the lumbar spine area but there is no activity over the distal shunt tip. The cisternogram confirms the impression of an obstructed CSF diversionary shunt.

is injected into the lumbar subarachnoid space. Imaging is performed over the distal shunt tip, either the peritoneal area or the heart, depending upon which type of shunt has been performed (Fig. 8, *A* and *B*).

Case IV. R.B., (JHH 129 82 64), a $1\frac{1}{2}$ year old boy, was admitted to the Johns Hopkins Hospital with a 6 month history of increasing head size and 11 month history of seizures. A pneumoencephalogram demonstrated hydrocephalus. Because of persistent increase in in-

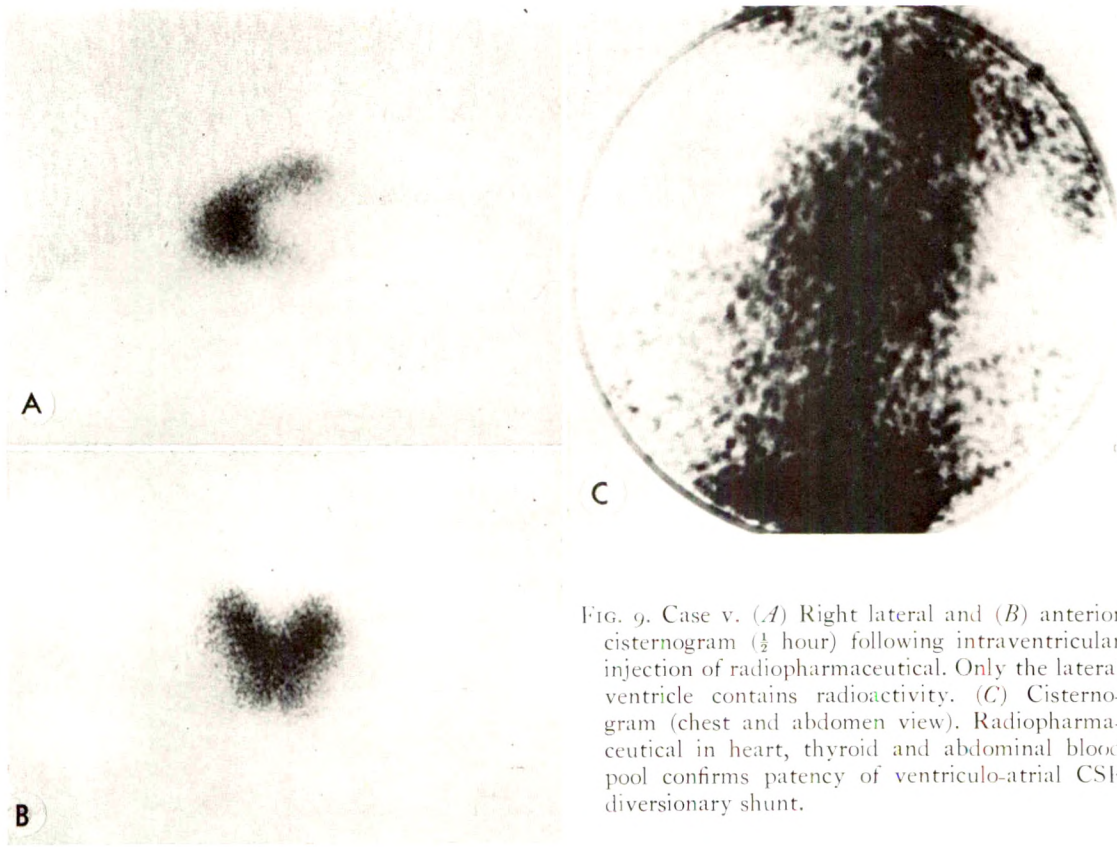


Fig. 9. Case v. (*A*) Right lateral and (*B*) anterior cisternogram (½ hour) following intraventricular injection of radiopharmaceutical. Only the lateral ventricle contains radioactivity. (*C*) Cisternogram (chest and abdomen view). Radiopharmaceutical in heart, thyroid and abdominal blood pool confirms patency of ventriculo-atrial CSF diversionary shunt.

tracranial pressure, a lumbar subarachnoid-peritoneal shunt was performed. The cisternography was done to evaluate shunt function (Fig. 8, *A* and *B*).

In obstructive hydrocephalus the radiopharmaceutical must be introduced into the cerebral ventricle and images made in the area of the distal shunt tip (Fig. 9, *A–C*).

Case v. B.B., (JHH 134 55 06), a 2 week old boy, was referred for evaluation of rapidly increasing head circumference. Cranial angiography demonstrated a mass in the occipital region. Air injected into the mass outlined a large cyst. A pneumoencephalography (PEG) showed no connection between the cyst and the ventricular system. It was concluded that the child had Dandy-Walker syndrome with marked cerebellar hypoplasia and obstruction of the 4th ventricular outlet.

A ventriculo-peritoneal shunt was performed. Intraventricular injection of Tc^{99m} as pertechnetate resulted in visualization of the cerebral ventricles. Subsequent imaging over the distal shunt tip showed radioactivity in the heart, thyroid, and abdominal blood pools (Fig. 9, *A–C*).

DISCUSSION

The cisternogram is not intended to compete with the pneumoencephalogram in terms of anatomic information, but yields physiologic information about flow patterns of the cerebrospinal fluid.[3] From the altered flow patterns, diagnoses can be inferred. Cisternography can be performed with only mild sedation, whereas general anesthesia is preferred for pneumoencephalography in children.

We have used both the rectilinear scanner and the gamma camera as imaging devices. The reduction in time needed to obtain an image using the gamma camera had been an advantage. Rectilinear scanners have optimum resolution at the focal plane and give better anatomic detail

of central structures such as the ventricles.

We have employed a number of different radiopharmaceuticals for cisternography including I^{131}HSA, Tc^{99m} serum albumin, as well as In^{111} and Yb^{169}DTPA chelate. The radiation dose to the spinal cord and nerve roots increases if there is obstruction to CSF flow in the spinal cord proximal to the injection site.[9] Radiation dose to the brain is greater in patients with hydrocephalus than normal subjects which must be considered when choosing a radiopharmaceutical. The unique information gained from this study makes the radiation dose to these structures acceptable with any of the above radiopharmaceuticals.

Cisternography is also being used in our laboratory to evaluate porencephalic and subarachnoid cysts, subdural hematomas, hygromas and meningomyeloceles. We anticipate that CSF imaging will be helpful in establishing the diagnosis and helping in the management of patients with these conditions.

SUMMARY

The introduction of a radiopharmaceutical into the lumbar subarachnoid space and subsequent imaging of the CSF pathways has proven useful in children for the evaluation of hydrocephalus, CSF leaks, diversionary shunts, and abnormal communications with the cerebrospinal fluid space.

Cisternography is safe, simple, and has low morbidity.

The procedure is accurate and we believe that it will become more widely used.

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REFERENCES

1. Atkinson, J. R., and Foltz, E. L. Intraventricular RISA as diagnostic aid in pre- and post-operative hydrocephalus. *J. Neurosurg.*, 1962, *19*, 159–166.
2. Brocklehurst, G. Use of radio-iodinated serum albumin to study CSF flow. *J. Neurol., Neurosurg. & Psychiat.*, 1968, *31*, 162–168.
3. DiChiro, G. Movement of cerebrospinal fluid in human beings. *Nature*, 1964, *204*, 290–291.
4. DiChiro, G. Observations on circulation of cerebrospinal fluid. *Acta radiol.* (Diag.), 1966, *5*, 988–1022.
5. DiChiro, G., Ashburn, W. L., and Briner, W. H. Technetium ^{99m}Tc serum albumin for cisternography. *A.M.A. Arch. Neurol.*, 1968, *19*, 218–227.
6. DiChiro, G., and Grove, A. S. Evaluation of surgical and spontaneous CSF shunts by isotope scanning. *J. Neurosurg.*, 1966, *24*, 743–748.
7. DiChiro, G., Reames, P. M., and Matthews, W. B. RISA ventriculography and RISA cisternography. *Neurology*, 1964, *14*, 185–191.
8. Glasauer, F. E., Alker, G. J., and Leslie, W. V. Isotope cisternography and ventriculography. *A.M.A. Am. J. Dis. Child.*, 1970, *120*, 109–114.
9. Harbert, J. C., Zieger, L. S., McCullough, D. C., Ashburn, W. L., and Davidson, J. D. Spinal cord dosimetry in ^{131}I HSA cisternography. To be published.
10. James, A. E., DeLand, F. H., Hodges, F. J., III, and Wagner, H. N., Jr. Cisternography: role in evaluation of normal pressure hydrocephalus. *J.A.M.A.*, 1970, *213*, 1615–1622.
11. James, A. E., DeLand, F. H., Hodges, F. J., III, and Wagner, H. N., Jr. Cerebrospinal fluid (CSF) scanning: cisternography. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *110*, 74–87.
12. McCullough, D. C., and Luessenhop, A. J. Evaluation of photoscanning of diffusion of intrathecal RISA in infantile and childhood hydrocephalus. *J. Neurosurg.*, 1969, *30*, 673–678.
13. Overbeck, W. J. Isotope investigation of hydrocephalus. *Psychiat., Neurol., & Neurochir.*, 1968, *71*, 99–103.

FACIAL DUPLICATION*

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FACIAL duplication, a rare anomaly, is a partial expression of the normal phenomenon of twinning. The result is usually a grotesque facial malformation. The rarity and potential cosmetic correctability of this condition prompt this case report.

REPORT OF A CASE

A 6 day old female was referred to the hospital because of facial anomalies noted following birth by cesarean section for prolonged breech labor. The mother had taken multiple vitamin preparations, thiamine, amitriptyline, and one other unidentified medication during early pregnancy. The mother also had undergone extensive diagnostic examinations during the first week of pregnancy for a vague neurologic complaint. Roentgen examinations included complete myelography, brain scanning, and examinations of the skull, spine, and chest. The mother had 2 normal children by a previous marriage; the father of the patient had no other children. A second cousin has a cleft palate and lip.

On physical examination, the child appeared healthy and normal except for head and facial abnormalities (Fig. 1, *A* and *B*). The head, face, and mandible were slightly larger on the right. The eyes were widely separated and the metopic suture was 2.5 cm. wide; 2 nasal bones were palpable and the nares were 3 cm. apart. Two oral stomata were present, each containing palpable lingual tissue, one on each side of a large fleshy mass which extended from the nose to the chin. In the vertical midline of the face, a horizontal cleft was present just below the nose at the usual location of the philtrum. A hairline subsequently developed on the inferior margin of this cleft resembling eyelashes, but no underlying ocular or palpebral tissue was identified at surgery. Chromosomal analysis was normal. Roentgenograms of the child at birth demonstrated no abnormalities except for those of the head and face. Facial tomograms demonstrated asymmetry of the mandible with minimal bony duplication and duplicated maxillary structures with clear demonstration of the oral cavities communicating with the pharynx posteriorly (Fig. 2, *A* and *B*).

At age 4 months, the child was readmitted for further evaluation and initial corrective surgery. A pneumoencephalogram demonstrated an elevated, widened third ventricle

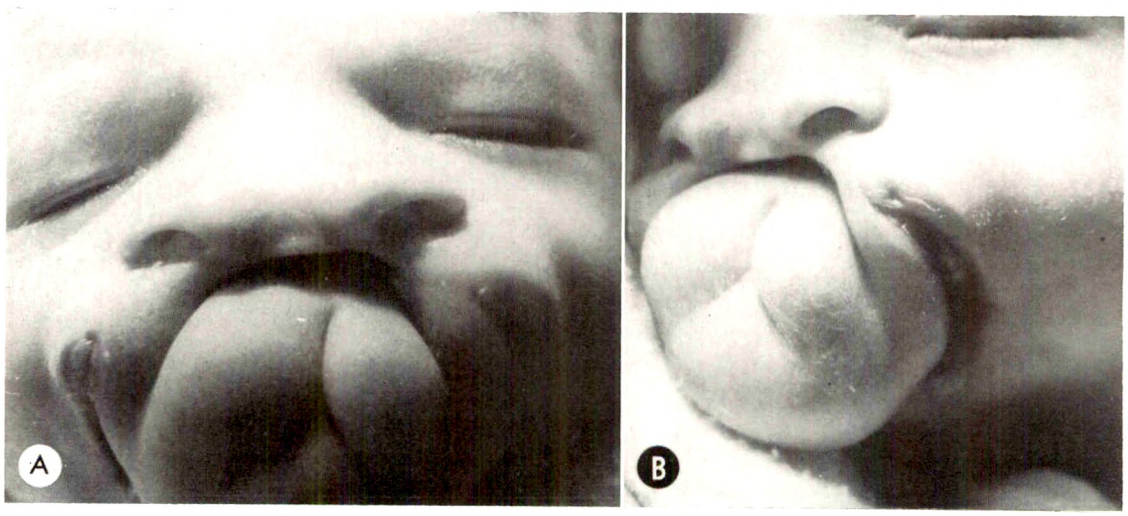


FIG. 1. (*A* and *B*) Photographs of infant demonstrating fleshy mass interposed between paired oral stomata, infranasal cleft, hypertelorism, and broad nose.

* From the Department of Radiology, University of New Mexico, School of Medicine, Albuquerque, New Mexico.

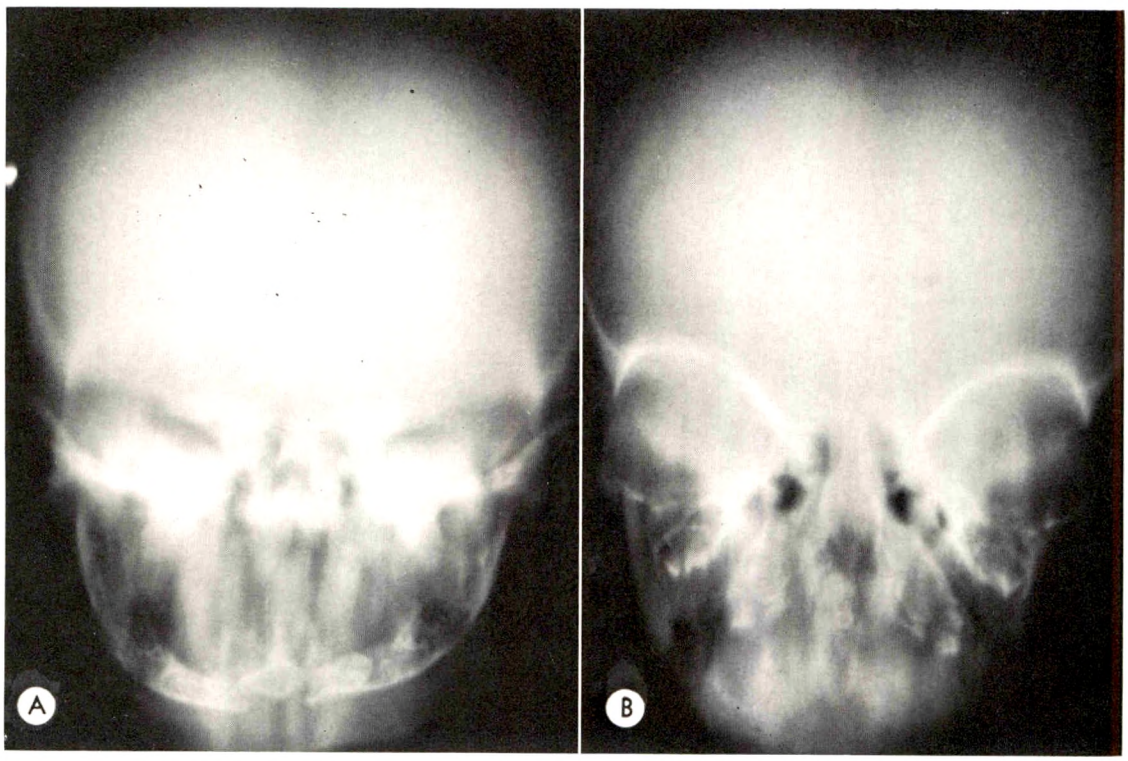


Fig. 2. (*A* and *B*) Tomograms showing paired maxillary structures and asymmetry of mandible with minimal duplication. Paired air passages to nasopharynx are evident on more anterior section.

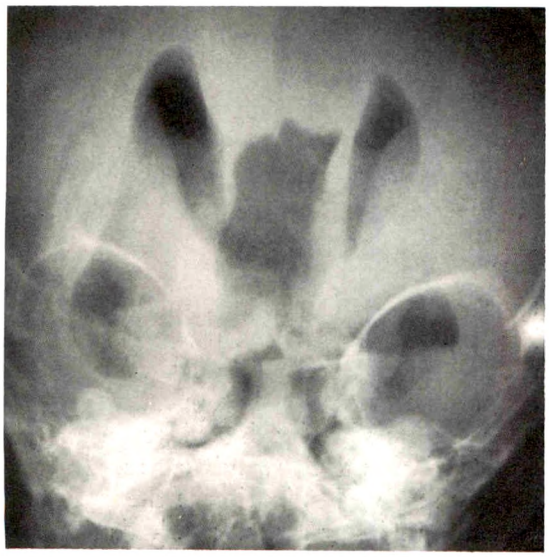


Fig. 3. Pneumoencephalogram, brow up. Frontal projection shows lateral displacement of lateral ventricles and a widened, elevated third ventricle due to agenesis of the corpus callosum.



Fig. 4. Pneumoencephalogram, brow up. Lateral view (autotomogram) shows the fourth ventricle extending from the foramen magnum to the level of C_3, indicating a probable Chiari II malformation of the hindbrain.

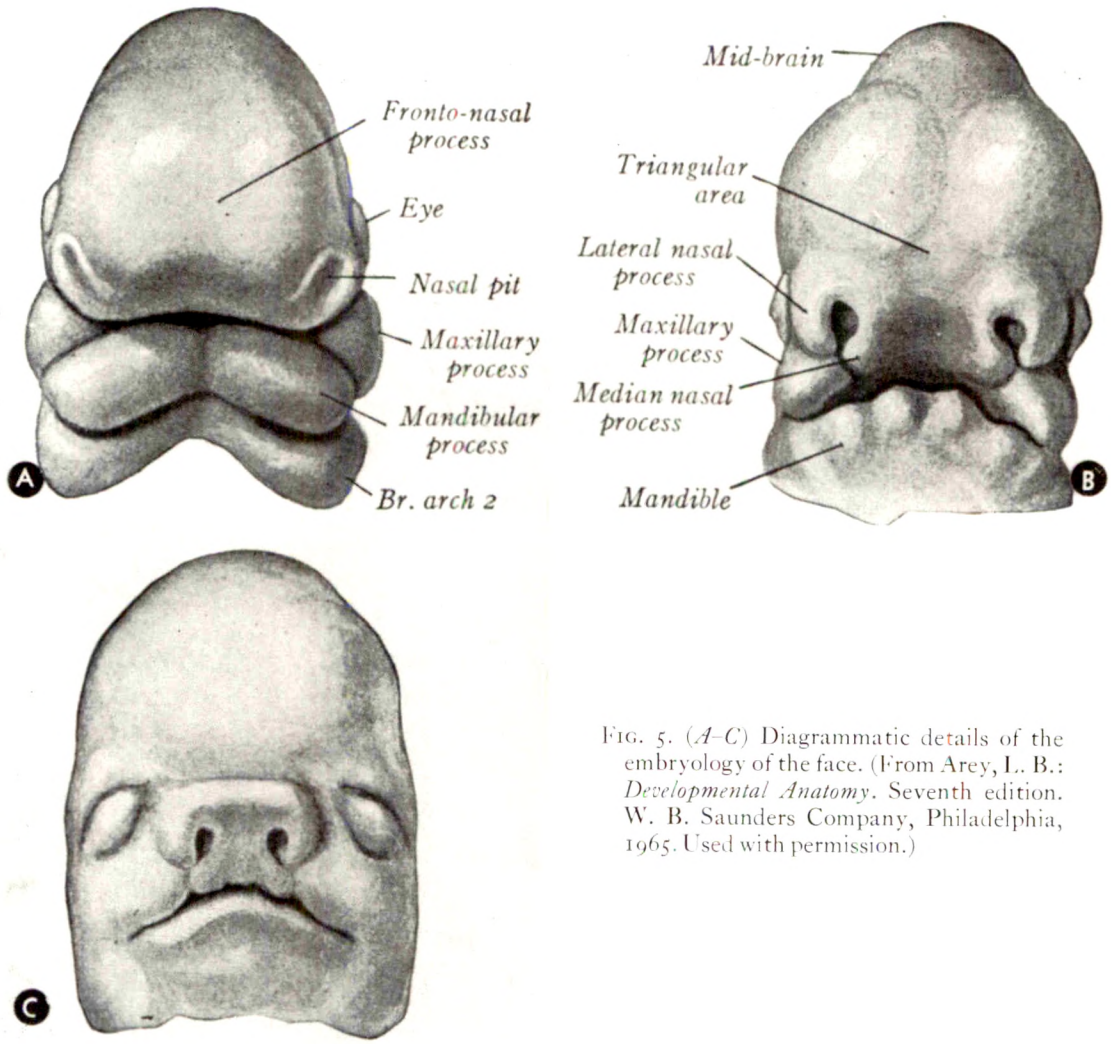


FIG. 5. (*A–C*) Diagrammatic details of the embryology of the face. (From Arey, L. B.: *Developmental Anatomy*. Seventh edition. W. B. Saunders Company, Philadelphia, 1965. Used with permission.)

and widely spaced lateral ventricles diagnostic of agenesis of the corpus callosum (Fig. 3). The fourth ventricle extended from C2–3 to the foramen magnum indicating the apparent presence of a Chiari II hindbrain malformation (Fig. 4). There was no evidence of an encephalocele. A sinogram of the infranasal cleft showed a blind-ending pouch.

DISCUSSION

Two similar cases of facial twinning have been reported previously.[1,3] The infant described by Goulian and Conway[1] underwent reconstructive surgery, but died at the age of 13 months of hydrocephalus. No intracranial abnormality other than secondary to hydrocephalus was found at autopsy. No clinical information is available on the child illustrated in Potter's textbook.[3]

Features common to infants with this type of facial duplication are hypertelorism, a broad flat nose with widely spaced nares, a transverse slit-like opening under the nose, 2 lateral oral stomata with varying amounts of fleshy tissue interposed, and (in Goulian and Conway's case and in the present study) duplication of the mandibular and maxillary structures. This duplication can be assessed best preoperatively on roentgenograms; roentgenographic details are not available for Potter's case.

The interrelationship between the intra-

cranial malformations and the facial duplication is difficult to determine; however, some relationship might exist. Two of the 3 cases, and possibly the third, have had serious abnormalities of the neural axis.

Reviewing the embryology of the face, it appears that bilateral duplication of the maxillary and mandibular processes of the first branchial arch occurred (Fig. 5, *A–C*). Fusion of the epidermal structures between the median nasal processes and the mandible left the central infranasal cleft which normally would have been part of a single oral stoma. Paired lateral oral stomata persisted, communicating with the pharynx.

Considerable plastic repair of this anomaly is possible;[1] however, the long term outlook is probably limited by the associated neurologic abnormality. Agenesis of the corpus callosum is associated with seizures, psychiatric abnormalities, mental retardation of varying degrees, and various structural anomalies of the neural axis.[4] The Chiari II malformation is generally associated with abnormalities of the spinal cord and vertebral column, although 1 case with a normal vertebral column and a normal spinal cord, except for kinking of the cervicomedullary junction and rostral direction of the upper cervical nerve roots, has been reported.[2] Pathologic proof of Chiari II malformation in this infant, who is alive and doing fairly well, is lacking, but clinically and roentgenographically no spinal cord or spinal anomaly is present.

SUMMARY

A case of facial duplication is reported.

The facial appearance is somewhat characteristic.

With the associated roentgenographic findings, the radiologist can aid the clinician in the diagnosis.

Two of 3 reported cases have had neurologic abnormalities, and cerebral pneumography is indicated.

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REFERENCES

1. Goulian, D., Jr., and Conway, H. A rare case of facial duplication. *Plast. & Reconstruct. Surg.*, 1964, *33*, 66–72.
2. Peach, B. Arnold-Chiari malformation with normal spine. *A.M.A. Arch. Neurol.*, 1964, *10*, 497–501.
3. Potter, E. L. Pathology of the Fetus and Infant. Second edition. Year Book Publishers, Inc., Chicago, 1961, pp. 222–223.
4. Toglia, J. U., and Lapayowker, M. D. Agenesis of corpus callosum. *Confinia neurol.*, 1966, *27*, 345–366.

ROENTGENOGRAPHIC FINDINGS IN FAMILIAL OSTEODYSPLASIA*

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THE roentgenographic findings of a genetic abnormality which has been previously described in the medical[1] but not in the radiologic literature are described. Four members of a sibship from a consanguineous marriage all exhibited characteristic roentgenographic and clinical appearances. An autosomal recessive pattern of inheritance is suggested.

CLINICAL FEATURES

The clinical feature for which the first member of this family was referred to us was a number of spontaneous fractures of the mandible over an interval of 9 years. There was nothing in the history to suggest systemic bone disease, and general growth and development were within normal limits. Physical examination of the patient revealed a peculiar physiognomy and striking facial features consisting of prominent brows, flattening of the nasal bridge, prominent ear lobes, dental malocclusion, and flattening of the mid-face and malar eminences (Fig. 1, *A* and *B*). She also had an evident thoracic scoliosis.

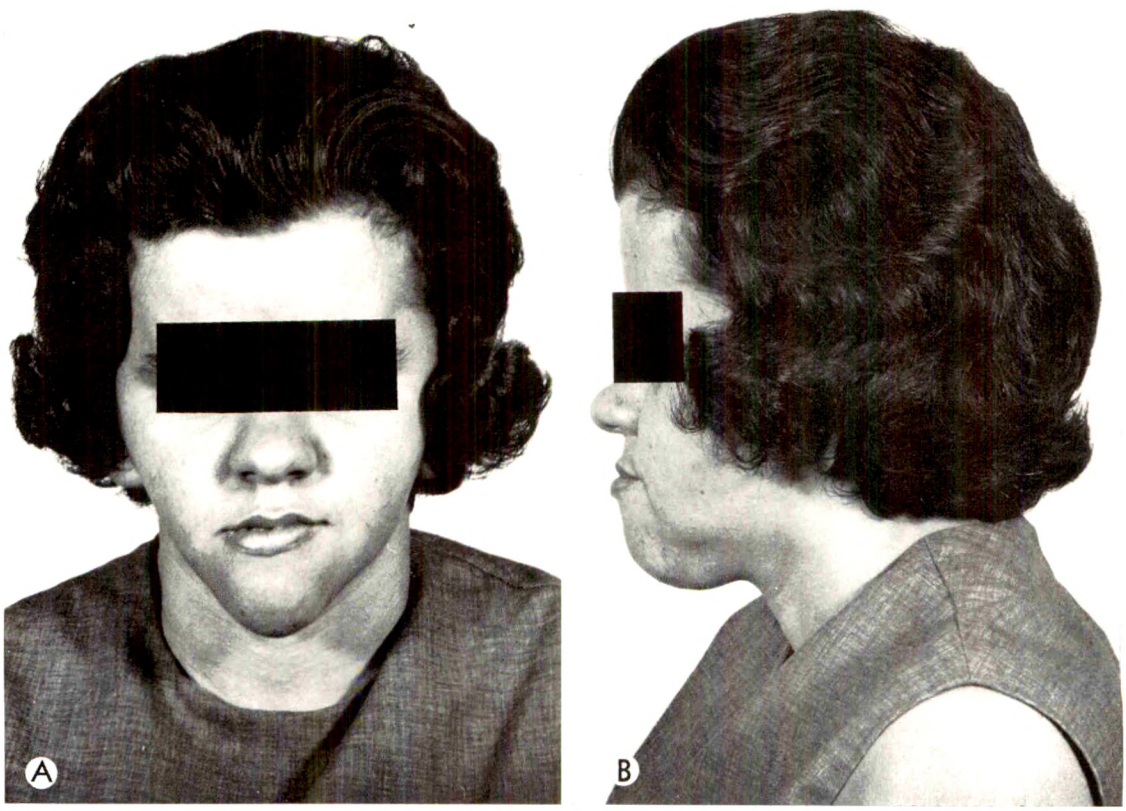


FIG. 1. (*A*) Frontal and (*B*) profile views of propositus. Note the malar and mandibular configuration.

* From the Department of Diagnostic Radiology, and National Institute of Dental Research, National Institutes of Health, Bethesda, Maryland.

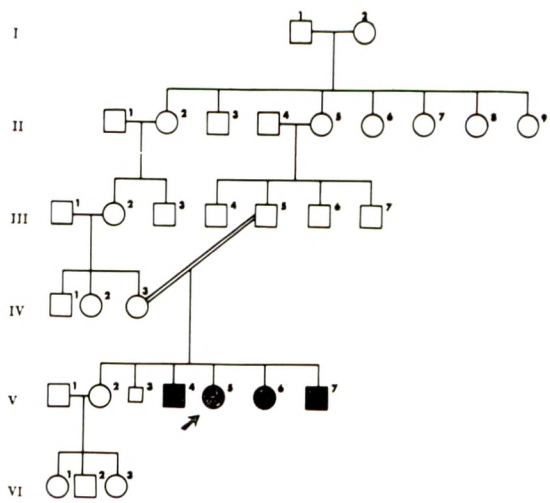


FIG. 2. Familial osteodysplasia; kindred. Darkened symbols represent affected siblings. The arrow denotes the propositus and the double line indicates the consanguineous marriage between first cousins once removed. This genealogy suggests an autosomal recessive pattern of inheritance.

Investigation of the patient's family revealed similar abnormalities of the facial structures in 3 of her 4 living sibs. As previously noted, the sibship was the product of a consanguineous marriage; the parents were first cousins once removed (Fig. 2).

TABLE I

SUMMARY OF THE ABNORMALITIES OF THE AFFECTED SIBS

	B.M.	H.M.	O.M.	D.M.
I. Clinical Findings				
A. Age	23	21	15	25
B. Sex	F	F	M	M
C. Elevated Diastolic Pressure	+	−	+	+
D. Scoliosis	+	+	+	+
E. Hyperuricemia	+	+	+	+
II. Roentgenographic Findings				
A. Facial Bones				
1. Mandibular abnormalities	+	+	+	+
2. Hypoplastic maxilla	+	+	+	+
3. Hypoplastic zygoma	+	+	+	+
B. Skull				
1. Brachycephaly	+	−	+	Roentgenograms not available for evaluation
2. Calvarial thinning	+	+	+	
3. Pointed anterior clinoids	+	+	+	
4. Hypoplastic petrous bones	+	+	+	
5. Pointed mastoids	−	+	+	
C. Spine-Pointed Spinous Processes	+	+	+	
D. Chest-Ribs Abnormal	+	+	−	
E. Pelvis				
1. Superior pubic ramus thin	+	+	−	
F. Hands—Clinodactyly	+	+	+	
G. Feet				
1. Abnormal phalanges	+	+	+	
2. Feet "paddle shaped"	+	+	−	
H. Cortical Thickening of Tubular Bones	+	+	+	

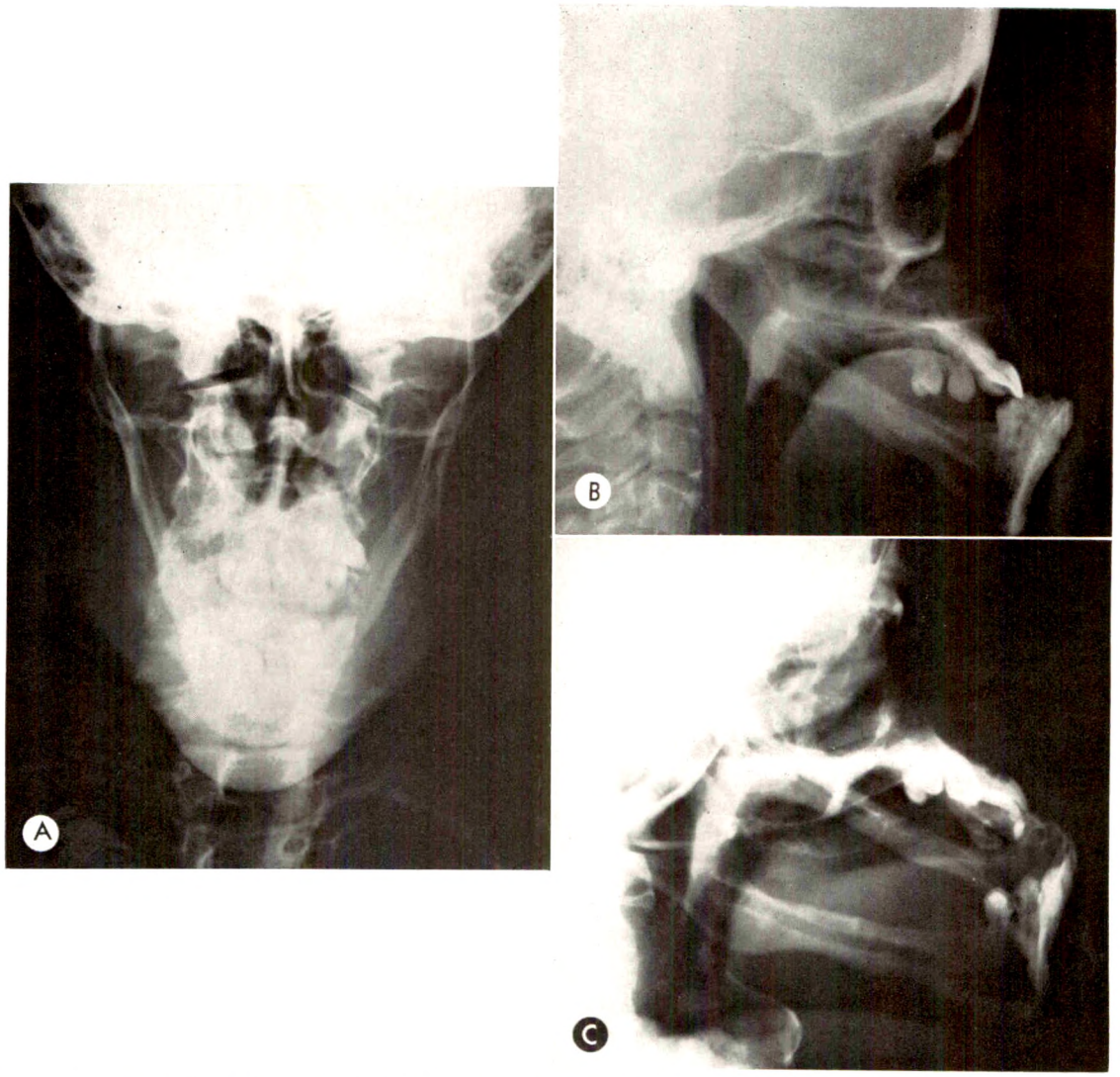


FIG. 3. (*A*) Frontal, (*B*) lateral and (*C*) oblique views of the facial structures reveal the mandible to be thick in the mental region with the ramus thin and wavy in contour. The gonial angle is excessively obtuse.

Examination of a number of the patient's relatives outside of the sibship failed to demonstrate any of the facial stigmata described.

All of the affected sibs exhibited varying degrees of the same facial findings. Each had a prognathic mandible and some degree of malocclusion, as well as a somewhat broad, flat nose. Each exhibited a V-shaped configuration to the lower face, straightening of the mandibular angle, and recession of the infra-orbital ridges in relation to the supra-orbital ridges. All affected sibs had some degree of scoliosis. A summary of the more significant abnormalities in the affected sibs is presented in Table 1. It is interesting to note that 3 of the 4 affected sibs had elevated diastolic blood pressure.

LABORATORY FINDINGS

All of the affected sibs were found to be hyperuricemic but not hyperuricosuric. No other biochemical abnormalities could be demonstrated. Specifically, there was no evidence of derangement of endocrinologic or calcium metabolism.

PATHOLOGY

A biopsy of the mandibular alveolar ridge in the patient with numerous previous, spontaneous mandibular fractures demonstrated histologically normal bone except for some sclerosis and new bone formation consistent with recent tooth extraction. A peripheral blood karyotype was also normal.

ROENTGENOGRAPHIC FINDINGS

Routine roentgenographic examinations of the skull, as well as Panorex films of the mouth reveal an unusual configuration of the mandible (Fig. 3, *A–C*). There is marked diminution in size of the maxilla and of the zygomatic bones including the malar portion thereof as well as the arch (Fig. 4). A striking feature on the plain skull roentgenograms is the calvarial thinning as well as brachycephaly. More subtle changes include a pointed configuration of the mastoids and hypoplasia of the petrous portion of the temporal bone (Fig. 5).

Three of the 4 affected sibs had roentgenograms of the spine and all 3 exhibited thoracic scoliosis; the spinous processes of some of the cervical vertebrae were small and pointed giving them a distinctly abnormal configuration. The lumbosacral spine was felt to be within normal limits. Roentgenograms of the feet revealed the proximal phalanx of the second, third, fourth and fifth digits to be relatively longer than normal while the first was thought to be probably normal. Also, the middle phalanx of the second, third, and fourth digits is relatively shorter than normal (Fig. 6, *A* and *B*).

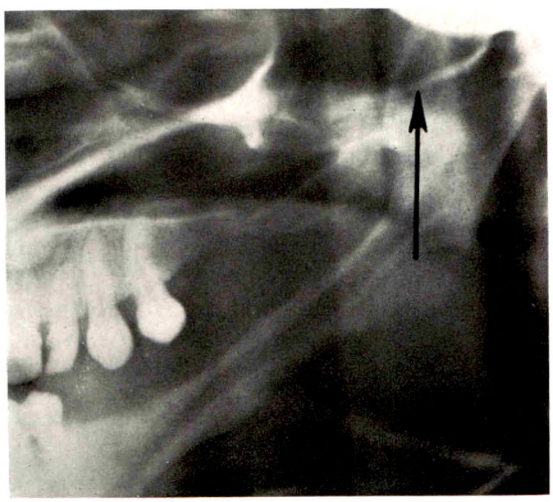


Fig. 4. Panorex view of facial bones demonstrates the hypoplastic appearance of the zygomatic arch (arrow) and of the maxilla.

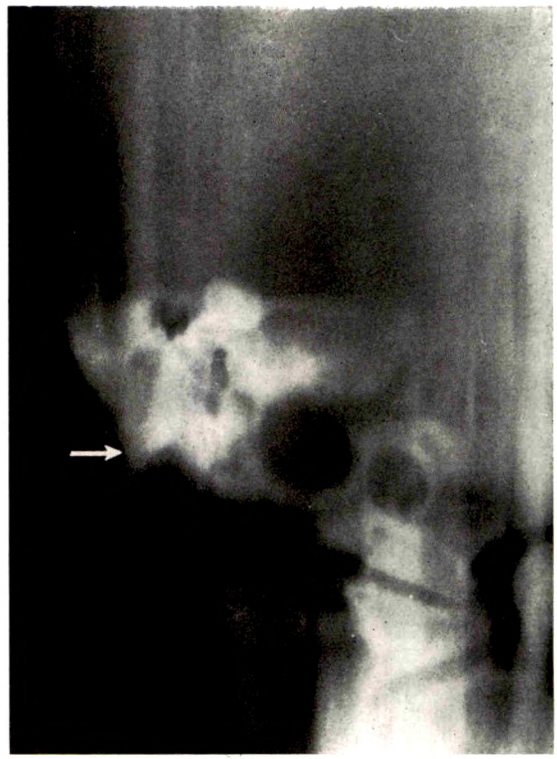


Fig. 5. Tomographic view in the anteroposterior projection. Note calvarial thinning, pointed mastoid tip and generally small size of petrous portion of temporal bone.

The pelvis revealed thinning of the superior pubic ramus bilaterally, while the ischial tuberosities were interpreted as normal (Fig. 7).

A prominent feature of this bone disorder seen throughout a number of bones of the body, but perhaps best demonstrated in the femoral shaft, is cortical thickening to an extent that compromises the medullary cavity of the bone (Fig. 8). This type of medullary encroachment by cortical thickening was noted in other bones including the phalanges of the hands.

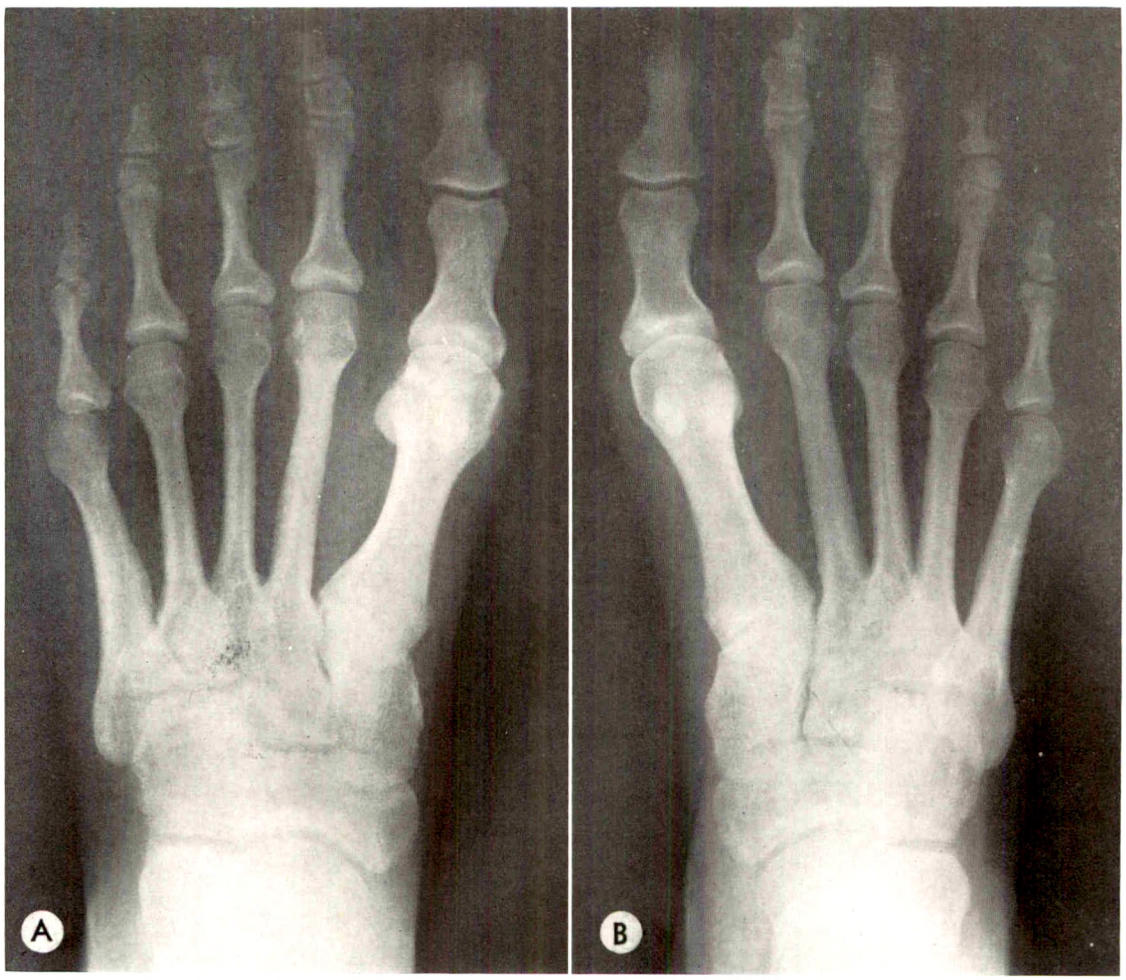


FIG. 6. (*A* and *B*) Note the disproportionate phalangeal lengths described in the text.

Chest roentgenograms in the affected siblings showed the previously described scoliosis as well as thinning of all the ribs. One of the affected sibs had 11 ribs bilaterally while another had small cervical ribs. A synchondrosis of the first and second ribs on the left was also noted in 1 of the patients.

DISCUSSION

The term "familial osteodysplasia" is proposed for the syndrome. The horizontal nature of the pedigree, the even distribution between sexes and the parental consanguinity are characteristic for an autosomal recessive pattern of inheritance. The phenotype is expressed in 4 affected homozygous sibs.

As in any bone abnormality, comparative roentgenograms of different skeletal structures should be paired with normal control roentgenograms of the same age to make variations informative. The latter was done for the mandible where absolute abnormalities of size, shape and configuration were demonstrated.

While the most characteristic abnormalities of this syndrome are found in the facial bones, there can be no doubt that it is a generalized disorder and that it affects bones of both membranous and cartilaginous origin. It is also apparent from the roentgenograms that some members of the sibship are affected to a greater extent than others. Some of the findings when seen as an isolated instance can be normal

variations; however, when taken as a constellation of findings and when repetitive in the members of the sibship, they are felt to represent abnormalities.

A review of previous dental films on the propositus revealed a similar appearance to the mandible at the time of spontaneous fracture 9 years previous to admission to this institution. There was also noted, subsequent to the fracture, apparent normal healing with good callus formation. Since these are the only previous films available for comparison, the time factor is a formidable unknown in this syndrome. It may be that, as these patients are followed, more changes will develop and/or there may be a progression of the abnormalities described herein.[4]

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of this syndrome includes a gamut of genetic disorders with malformation of facial bones. Cleidocranial dysostosis is excluded on the basis of complete clavicular development, lack of coxa vara, thinning rather than thickening of ribs centrally, and absence of the classic changes in the cranial vault.[2]

Because of the thickened bone cortex, abnormal mandibular angle and craniofacial disproportion, one might consider pycnodysostosis; however, the lack of generalized osteosclerosis and the normal terminal phalanges help to eliminate this diagnosis.

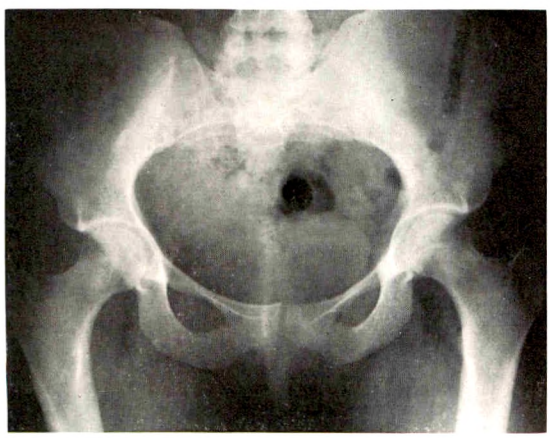


Fig. 7. The striking feature about the pelvis is the thinning of the superior pubic rami. The acetabular angles are at the upper limits of normal.

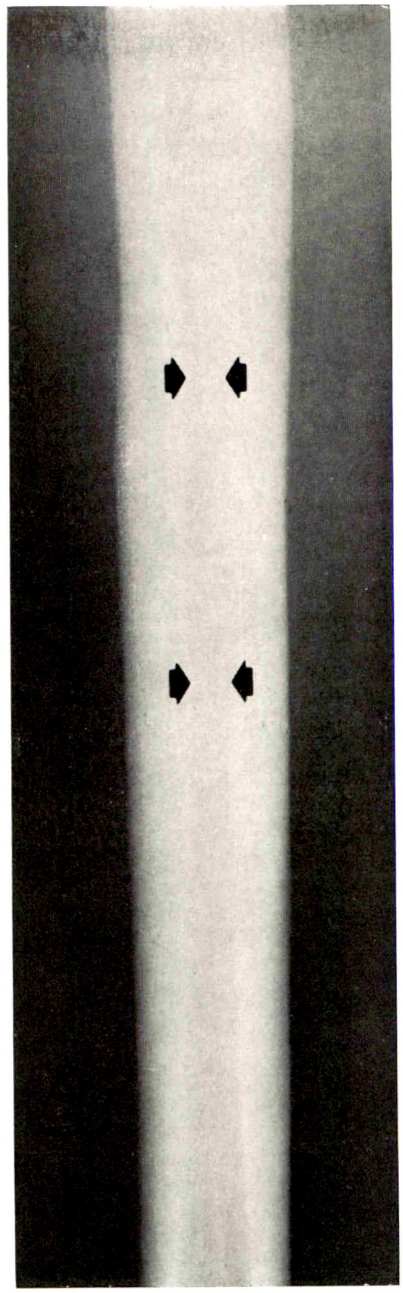


Fig. 8. Femoral cortical thickening with medullary encroachment (arrows).

Craniometaphyseal dysplasia (Pyle's disease) not only exhibits a different facies clinically, but demonstrates roentgenographic evidence of leontiasis ossea, decreased aeration of the paranasal sinuses

and mastoids, and metaphyseal flaring of the long bones.[3,6]

Mandibulofacial dysostosis (Treacher-Collins syndrome) must be considered because of the absence of zygomatic bones and associated hypoplasias of the maxilla and mandible.[5] However, the facies, especially the antimongoloid slant of the palpebral fissures and the roentgenographic findings are unlike those of familial osteodysplasia.

Other possibilities which may be considered include cranio-facial dysostosis (Crouzon's syndrome), acrocephalosyndactyly (Apert's syndrome), and oral facial-digital syndrome, but each entity has roentgenographic findings easily distinguishable from familial osteodysplasia.

In addition to the clinical and roentgenographic parameters just mentioned, it is important to note that many of the diseases or syndromes with which familial osteodysplasia may be confused exhibit abnormal teeth, cartilage, connective tissue, eyes, or intelligence, whereas these are normal in this family.

SUMMARY

The roentgenographic findings of a recently described syndrome are presented.

The syndrome is familial in nature and represents a disturbance of osteogenesis with the facial skeleton most severely affected.

The term "familial osteodysplasia" is proposed for the disorder.

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REFERENCES

1. ANDERSON, L. G., COOK, A. J., COCCARO, P. J., and BOSMA, J. F. Familial osteodysplasia. *J.A.M.A.*, 1972, *220*, 1687–1693.
2. EISEN, D. Cleidocranial dysostosis with report of four cases. *Radiology*, 1953, *61*, 21–31.
3. MORI, P. A., and HOLT, J. F. Cranial manifestations of familial metaphyseal dysplasia. *Radiology*, 1956, *66*, 335–343.
4. RUBIN, P. Dynamic Classification of Bone Dysplasias. Year Book Publishers, Inc., Chicago, 1964, pp. 65.
5. STOVIN, J. J., LYON, J. A., and CLEMMENS, R. L. Mandibulofacial dysostosis. *Radiology*, 1969, *74*, 225–231.
6. TAYBI, H. Generalized skeletal dysplasia with multiple anomalies; note on Pyle's disease. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1962, *88*, 450–457.

FAMILIAL ACROCEPHALOSYNDACTYLY (PFEIFFER SYNDROME)*

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ACROCEPHALOSYNDACTYLY (ACS) is a descriptive term applied to a group of closely related genetic diseases characterized by a premature fusion of the cranial sutures and a varied degree of syndactyly. In recent years, several distinct varieties of ACS have been separated from the classic type described by Apert[1] in 1906. Although considerable phenotypic overlay exists, these syndromes are caused by distinct genes, and correct genetic counseling depends on an accurate clinical and roentgenographic diagnosis.

This report describes the clinical and roentgenographic features in a mother and her 2 children who had a dominantly inherited, familial variety of acrocephalosyndactyly as described by Pfeiffer.[21]

REPORT OF CASES

Case I. A 30 year old mother of 3 was born with a prominent forehead, broad thumbs and toes, and webbing between the second and third toes of both feet. She did not receive any specific treatment for these abnormalities, and physical and mental development was normal. At the age of 20 years, the patient delivered a child (Case II) that resembled the mother in several respects. A second child, born when the mother was 21 years of age, was normal. The third child (Case III) was born when the mother was 25 years old, and this child also had anomalies of the head and extremities similar to those of the mother. One year later, the mother experienced a single grand mal seizure. An electroencephalogram was said to have been abnormal and she was treated with Dilantin and Gemonil without recurrence of the seizures. The mother complained of pain in her hips, elbows, shoulders, and hands which was not associated with swelling or redness, but was aggravated by the cold. Neither the parents nor other members of the patient's family had similar malformations of the skull or extremities.

On physical examination, the skull was decreased in anteroposterior diameter and the forehead was wide and prominent. The circumference of the head measured 51 cm. and the inner canthal distance 40 mm. The eyes protruded slightly, and the patient wore eyeglasses because of hyperopia (Fig. 1, *A* and *B*). Hypoplasia of the maxillary region with a "parrot-beaked" nose was apparent. The palate was narrow and the alveolar ridges were wide. The teeth were irregularly placed and some of the anterior teeth were missing. Duplicated upper canines had been removed previously. Supination of the forearms was limited and cubitus valgus was present. Brachydactyly, broad thumbs, and radially deviated terminal phalanges were noted in the hands. A painful bony nodule was present on the distal phalanx of the right thumb and there was a moderate clinodactyly of the fifth fingers. Dermatoglyphics were normal. The great toes were broad, and there was a partial soft-tissue syndactyly that involved the second and third toes, bilaterally. In general, all the long bones of the arms and hands appeared short. Height of the patient was 152 cm. Routine clinical laboratory values of the blood chemistry were unremarkable.

Roentgenographic Findings (Fig. 2–5). The roentgenograms of the skull demonstrated the typical features of premature fusion of the cranial sutures, with increased height and flattening of the frontal and occipital bones, and shortened anterior and posterior diameters. Minimal increase in the convolutional markings of the skull, upward slanting of the lesser sphenoid wings, a deepened sella turcica, and a small posterior fossa were also shown. An apparent assimilation of the atlas at the base of the skull was noted. Maxillary hypoplasia was pronounced and resulted in prognathism.

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§ Recipient of a Career Development Award from the National Institute of Child Health Development, USPHS.



FIG. 1. Case 1. (*A* and *B*) Views of face and head showing maxillary hypoplasia, prominence of the eyes, wide forehead, and increased inner canthal distance.

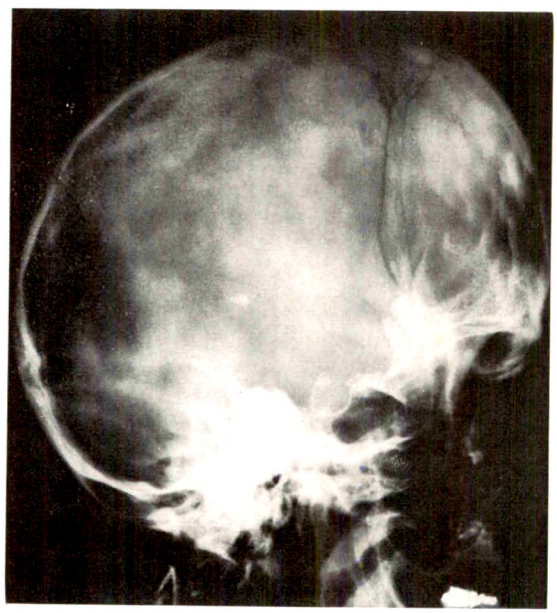


FIG. 2. Case 1. Lateral view of the skull showing typical changes of acrocephaly. The size of the skull is increased and the frontal and occipital bones are flat. The depth of the sella turcica is increased. Pronounced maxillary hypoplasia is present.

The roentgenograms of the hands showed a generalized brachydactyly. The greatest degree of shortening was in the middle phalanges. especially those of the index and little fingers, Complete fusion of the proximal and distal phalanx of the left thumb and partial fusion of the interphalangeal joint of the right thumb were noted. The thumbs were broad and deviated radially. The articular cartilage of all the interphalangeal and metacarpophalangeal joints was narrowed. No fusion of the carpal bones was demonstrated. A bone island was present in the distal left ulna. Significantly, no osseous syndactyly was demonstrated.

The roentgen findings in the feet were a generalized brachyphalangia with absence of the middle phalanges of all the toes and partial fusion of the proximal and distal phalanges of the great toes. The first metatarsal was greatly shortened and extremely broad. Multiple tarsal coalitions were noted, with calcaneocuboid fu-

sion and synostosis with the first and probably the second cuneiform, bilaterally. Partial synostosis of the lateral cuneiform and third metatarsal was well demonstrated in oblique projections, bilaterally. Partial synostosis of the first metatarsal and the first cuneiform was also demonstrated. Basically, the tarsal bones were grouped into two partially fused masses: the calcaneocuboid and the remaining tarsal and medial metatarsal bones. An accessory center of ossification medial to the base of the first metatarsal was present on both sides.

A probable assimilation of the atlas and occiput was seen on roentgenograms of the cervical spine. No cervical fusions were seen. A congenital block vertebra at the level of the fourth and fifth lumbar and posterior and partial anterior fusion were present in the lumbar spine.

In roentgenograms of the shoulder joint a bilateral hypoplasia of the proximal humeral head and glenoid fossa was seen. The over-all length of the humerus was shortened.

The pelvic shape was triangular and the symphysis pubis was abnormally wide. The femoral necks were short and broad with shallow acetabular fossae and mild coxa valga. A mild hypoplasia and flattening of the humeral condyles, with early degenerative changes at the humeral-ulnar articulation were seen on roentgenograms of the elbows. The radial head was flattened. No significant roentgen abnormalities were detected in the forearms, lower legs, femora, or chest.

Case II. At birth this 10 year old daughter of Case I had malformations of the skull and extremities, similar to those of her mother. In the first 4 months of this child's life, premature fusion of cranial sutures necessitated bicoronal, biparietal, and suboccipital craniotomies. A third surgical procedure was necessary at 8 months of age to correct a mild communicating hydrocephalus associated with an Arnold-Chiari malformation and bilateral tonsillar herniation.

In her first 3 years, developmental landmarks were delayed. Although the girl said her first

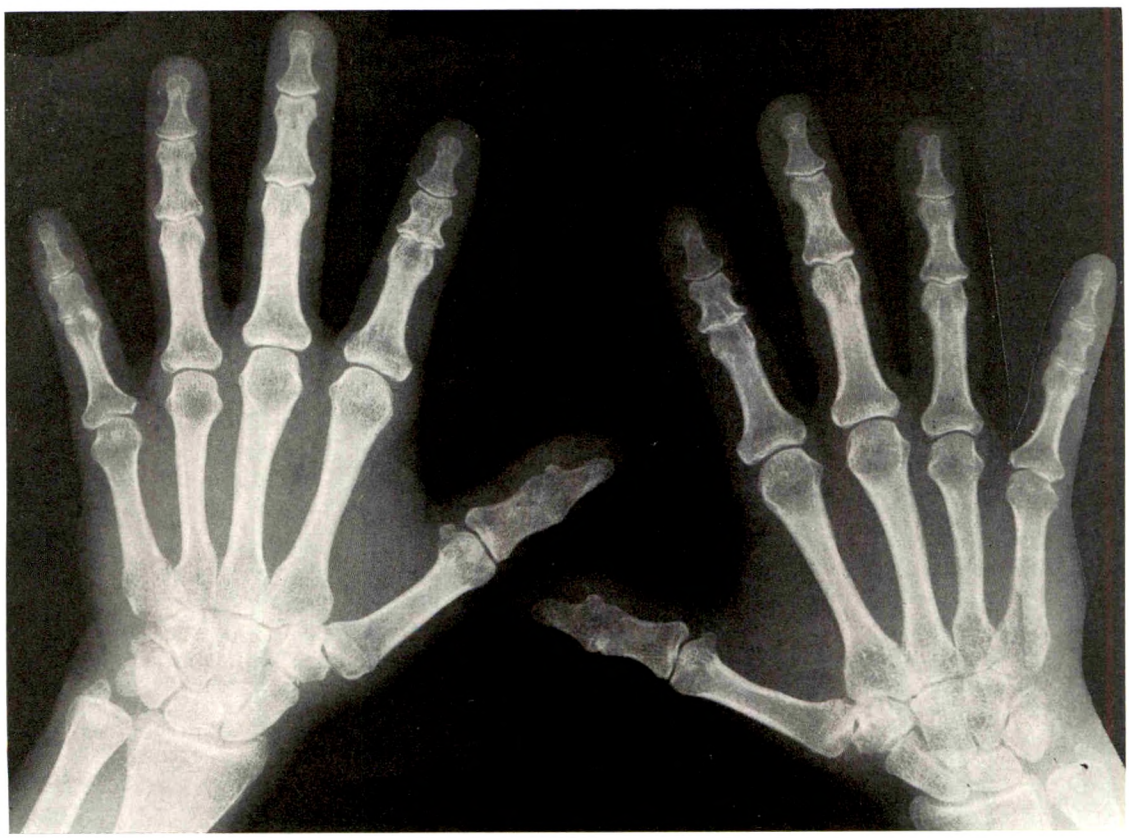


Fig. 3. Case I. Brachymesophalangia of the second and fifth digits and interphalangeal fusion of the thumb are present. The digits are generally shortened and the thumbs broadened minimally.

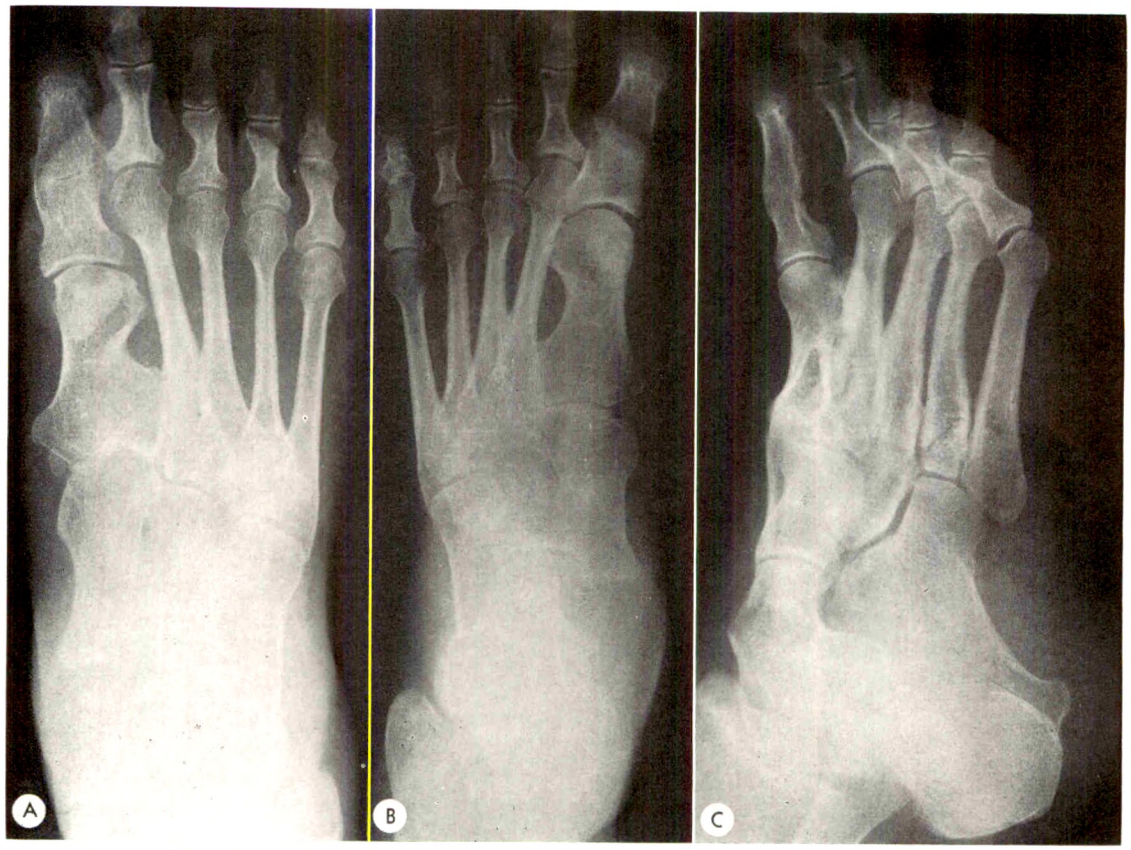


FIG. 4. Case 1. (*A* and *B*) Frontal, and (*C*) oblique projections of the feet. The first metatarsals are markedly broadened and deformed, especially on the right. The interphalangeal joints of the broadened and shortened great toe are fused. The middle phalanges are absent bilaterally, and multiple tarsal bones have fused.

word at 7 months, she did not walk until 2 years of age. At 3 years she required glasses because of severe hyperopia. Thereafter development appeared normal and she progressed well in school. At 9 years of age, petit mal and grand mal seizures began; these were treated with phenobarbital and Gemonil. Also at 9 years, strabismus was corrected surgically.

On physical examination, the patient's resemblance to her mother was striking. The conformation of the skull was similar and the circumference of the head was 52.8 cm. The inner canthal distance was 37 mm. and there was marked antimongoloid slant to the eyes. Pronounced maxillary hypoplasia was noted and the mouth was held open. The alveolar ridges were wide and the palate was narrow but high (Fig. 6). The teeth were crowded and orthodontic procedures had been performed to correct malalignment. The ears were low-set but normal in conformation. External rotation of the elbows was limited markedly. Shortening of all fingers, abnormally wide thumbs, and slight clinodactyly of both fifth fingers were observed. Dermatoglyphics were normal. The great toes were broad and a soft-tissue syndactyly of both second toes extended as far distally as the nails (Fig. 7). The patient's height was 137 cm. Although formal testing was not done, mentation appeared somewhat slow. However, her teacher stated that the child's school work was above average.

Roentgenographic Findings (Fig. 8–13). Skull roentgenograms were obtained at 8 and 10 years of age. Previous craniectomy defects were noted along the coronal and lambdoid sutures bilaterally. Metallic clips about the foramen magnum had been placed during the previous surgical procedures because of premature cranial synostosis and Arnold-Chiari malformation. The frontal bone was increased in height and was flat, and the coronal diameter of the skull was increased. The lesser sphenoid wings slanted upward and were unusually dense. A shallow

anterior fossa slanted upward. The pituitary fossa appeared small in the lateral projection and the interorbital distances were increased. The maxillary bones were markedly hypoplastic, the palate was arched, and the teeth were crowded. The basal view of the skull demonstrated a small mandible and increased width of the skull. The frontal and maxillary sinuses were hypoplastic and the sphenoid sinus was large. In the cervical spine the spinous processes and posterior elements of the fifth and sixth cervical vertebrae were fused completely and the anterior elements of these vertebral bodies were partially fused. The posterior part of the first cervical vertebra was hypoplastic and was assimilated partially into the occiput.

Generalized mild brachydactyly of the hand, shortening of the middle phalanges of the second and fifth digits, and minimal cone-shaped epiphyses on these phalanges were demonstrated. Roentgenograms of the proximal phalanx of the thumb showed a partially assimilated anomalous ossification center in the distal portion. No fusion of the carpal bones was noted or bony fusion of the phalanges. The thumbs were abnormally broad. At the age of 8 years, the roentgenograms of the feet demonstrated hypoplasia of the middle phalanges and absence of the epiphyses of the distal and middle pha-

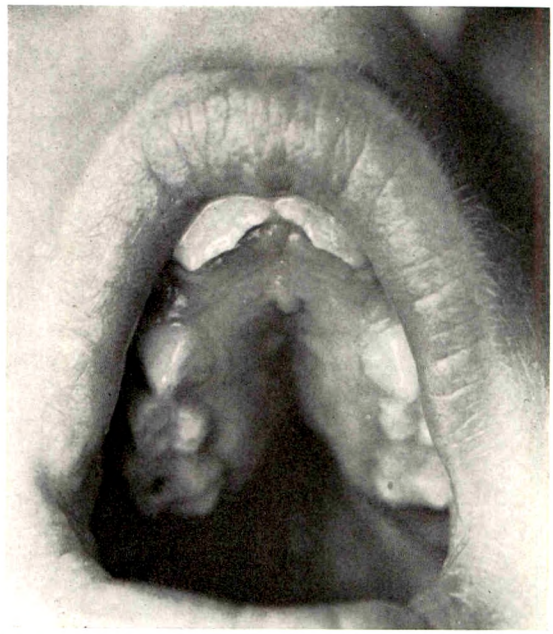


Fig. 6. Case 11. High narrow palate with wide alveolar ridges. The lateral incisors have been removed because of crowding of the teeth.

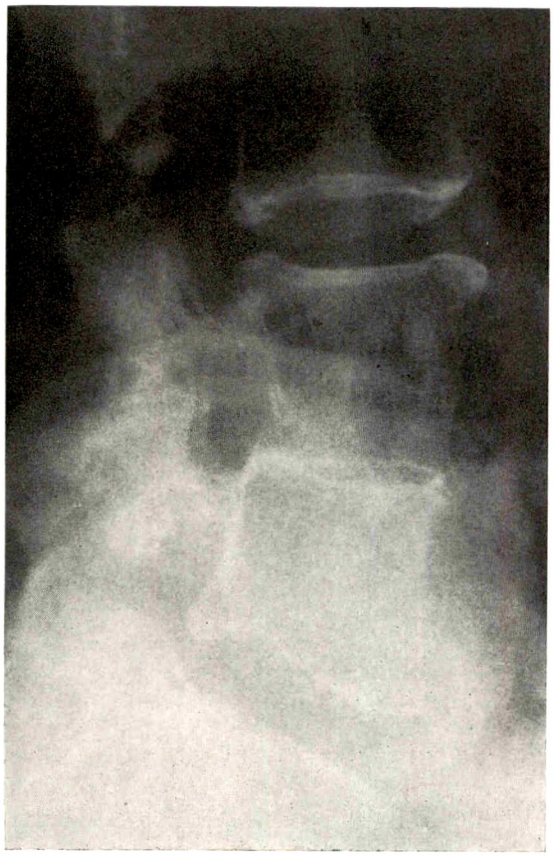


Fig. 5. Case 1. Lateral view of lower lumbar spine. The anterior and posterior elements of the fourth and fifth lumbar vertebrae have fused.



Fig. 7. Case 11. Broad great toes with extensive soft-tissue syndactyly of the second and third toes bilaterally.

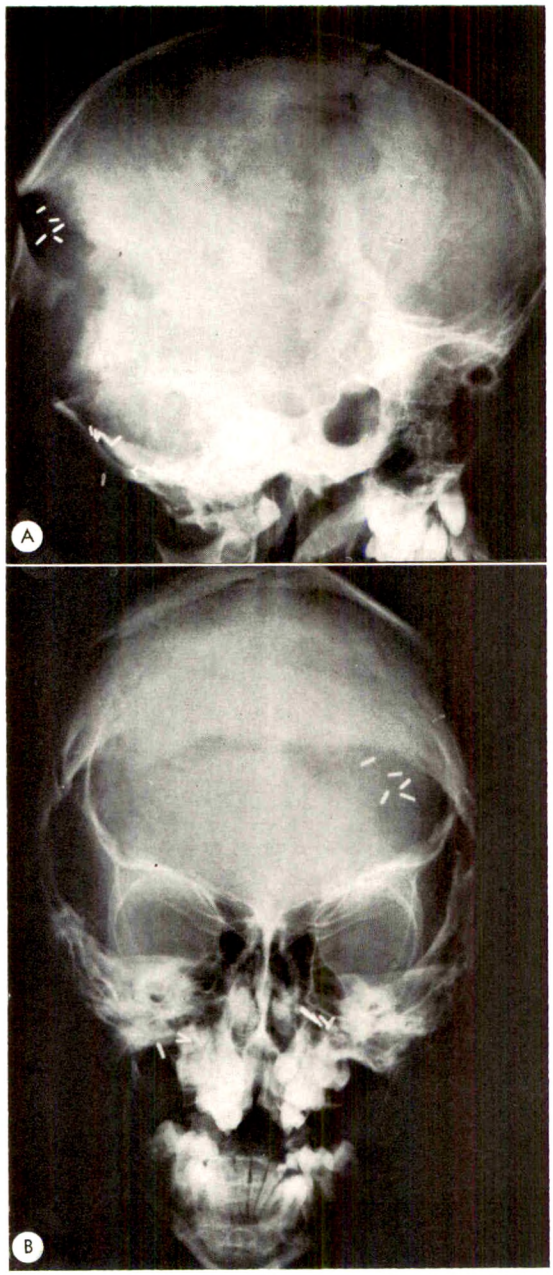


FIG. 8. Case II. (*A*) Lateral and (*B*) frontal projections of the skull. The postoperative defects are the result of craniectomy performed because of Arnold-Chiari malformation and premature sutural synostosis. Severe maxillary hypoplasia, crowding of the teeth, and narrowing of the palate are present.

langes. An anomalous ossification center was present in the proximal phalanx of the great toe which was abnormally wide and considerably shortened. The first metatarsal was also extremely broad and short, especially on the left side. The epiphysis of the left first metatarsal was deformed and medially placed. Early fusion was seen of the deformed medial cuneiform with the first metatarsal and navicular bones. The navicular and middle cuneiform were fused on the right side. Solid calcaneocuboid fusion was present bilaterally.

Roentgenograms at the age of 10 years demonstrated a progressive synostosis of the previously described early fusions with the calcaneocuboid fusion forming one solid mass, and multiple coalitions between the cuneiforms, navicular, and first 3 metatarsals. The middle and distal phalanges had not yet fused.

In the roentgenogram of the elbow joint, considerable hypoplasia of all the bones was seen. The normal anterior angulation of the distal humeral condyles was absent and the radial head was hypoplastic. The epiphysis of the proximal radius was extremely small and flat. In the lumbar spine, the intervertebral disk between the fourth and fifth lumbar vertebrae was narrow and the posterior elements were fused. The right twelfth rib was hypoplastic. Coxa valga, abnormal widening of the symphysis, and hypoplasia of the greater trochanters were seen in roentgenograms of the pelvis.

No significant roentgen abnormalities could be detected in the chest, knees, lower extremities, forearms, humeri, or femora. The acromial process of the scapula appeared unusually large.

CASE III. This patient, the 5 year old daughter of Case I, had malformations similar to those of her sister and mother. During the first year of life, premature fusion of the coronal suture was noted and craniectomies were performed. Early development was slightly delayed and the child did not walk until 17 months. Mental development, however, was considered normal thereafter.

On physical examination, the patient's face resembled that of her mother and sister (Fig. 14, *A* and *B*). The forehead was bulging and a surgical bony defect was noticeable. The head measured 45.4 cm. in circumference and the inner canthal distance was 30 mm. The right ear was slightly low-set. The alveolar ridges were wide and the palate was deep. Only the right upper incisor, which had been capped, remained in the jaw. The lower teeth were placed irregularly. External rotation of the forearm was moderately limited and full extension of the elbow could not be attained. The thumbs were

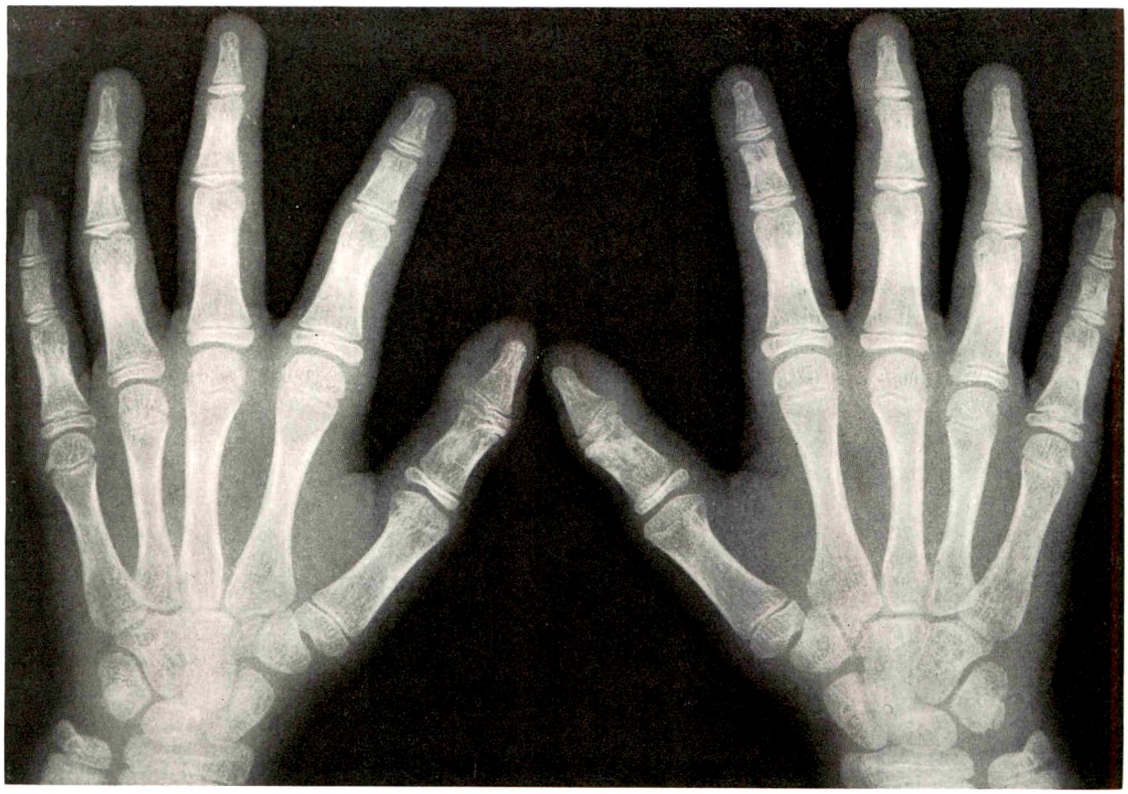


Fig. 9. Case II. Anteroposterior view of the hands. The second and fifth middle phalanges are minimally shortened and the thumbs are broadened. An anomalous ossification center is seen at the distal end of the proximal phalanx of the thumb, which may lead to fusion, as in the mother.

broad, the terminal phalanges were deviated radially, and clinodactyly was seen of both fifth fingers. Brachydactyly was generalized. Dermatoglyphics were normal. The great toes were unusually broad and a soft-tissue syndactyly reached to the nails between the left second and third toes. Shortening of the long bones was generalized and the patient's height was 103 cm. The ratio of the upper to the lower segment was 1.2:1.

Roentgenographic Findings (Fig. 15–19). The deformities of the skull were the same as in the mother and the sister. Early fusion of the posterior element of the third and fourth cervical vertebrae was seen. Pronounced maxillary hypoplasia and crowding of the teeth were noted. A generalized brachyphalangia was observed in the hands. Cone-shaped epiphyses of minimal degree were present on the middle phalanges of the second and fifth fingers which were considerably shortened. The thumbs were minimally broadened. In the feet a complete synostosis of the calcaneus and cuboid was demonstrated. The first metatarsal was broad and short. The middle phalanges were absent in all the toes. The tarsal bones were not yet sufficiently ossified to determine possible fusions.

Hypoplasia of the elbow joint and a slight bowing of the radius were noted. No bony fusions were demonstrated in the lumbar spine. In the pelvis the symphysis pubis was abnormally wide and bilateral coxa valga was present.

DISCUSSION

Although the classification of the various forms of acrocephalosyndactylism has been further refined, the terminology proposed by Blank[4] is the easiest to remember. Blank[4] reviewed the literature and concluded that all cases of acrocephalosyndactyly could be divided into typical and atypical forms. Temtamy[32] in her thesis on hand malformations concurred with this basic division. The typical form is a sporadic, dominant mutant, as described by Apert.[1,8,12,19] Essential for the diagnosis of the Apert syndrome is an interosseous

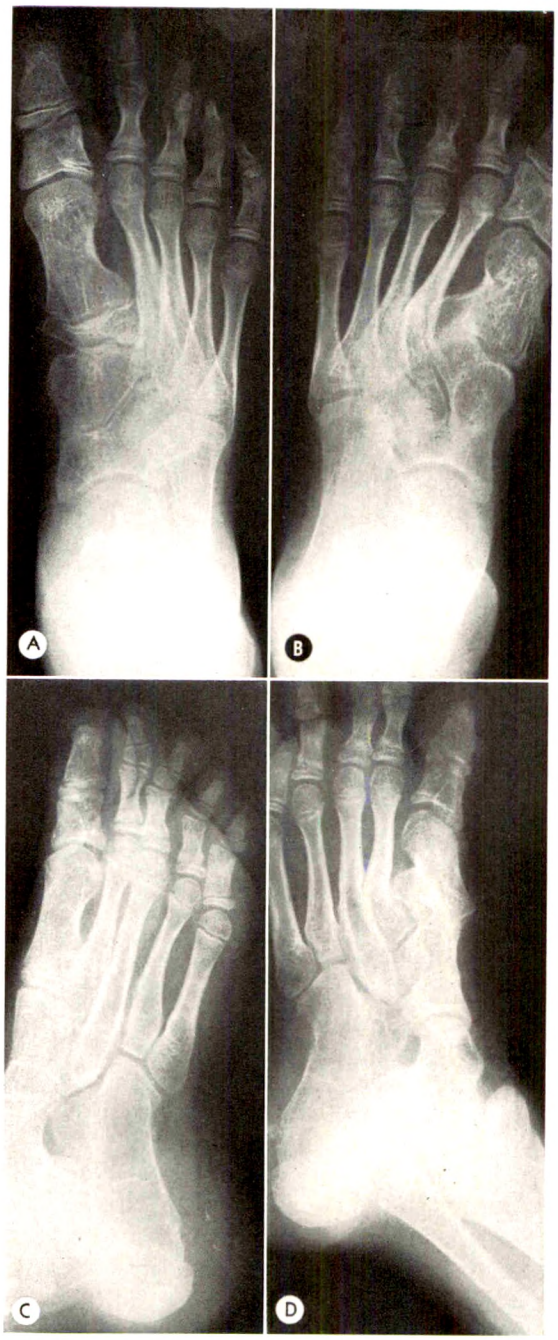


FIG. 10. Case II. (*A* and *B*) Anteroposterior and (*C* and *D*) oblique projections of the feet. The first metatarsals are severely deformed and an unusual epiphysis of the proximal phalanx of the great toe is present. The epiphyses of the distal and middle phalanges are absent, probably prior to their fusion. The calcaneocuboid and multiple other tarsal and metatarsal bones are fused.

fusion of the distal phalanges with complete syndactyly, and usually a single nail (synorchia). Hoover *et al.*[13] have described in detail the malformations in 20 cases of Apert ACS. In these, severe premature synostosis of the cranial sutures resulted in the classic "tower skull." Other associated anomalies, such as mental retardation, maxillary hypoplasia, limited motion of the elbows, and visual difficulties were often present. Other causes of secondary craniosynostosis have been reviewed by Duggan and co-workers.[10]

All of the other variations of acrocephalosyndactyly were called atypical ACS. In none of these variants was the severe interdigital osseous fusion seen that is char-



FIG. 11. Case II. Lateral view of the cervical spine. Hypoplasia of the posterior elements of the first cervical vertebra is noted; patient had Arnold-Chiari malformation. Complete fusion of the posterior elements of the fifth and sixth cervical vertebrae and partial fusion of the anterior intervertebral body are noted.

acteristic of the Apert syndrome. Temtamy[31,32] and others have further subdivided this group of non-Apert ACS according to the pattern of inheritance, absence, or presence of polydactyly, severity of syndactyly, and other associated anomalies.

Since these further subdivisions of atypical ACS are relatively rare and often manifest only minor differences in the pattern of malformation, only 1 of these, the Pfeiffer syndrome, will be discussed further. Details of the other 5 types are in the reports by Temtamy and others.[3,17,22,31,32,34]

The Pfeiffer variant appears the most common type of the group of atypical forms of acrocephalosyndactyly. Several reports[17,22,38] have appeared since the original description by Pfeiffer,[21] in 1964. The diagnostic features include autosomal dominant familial transmission, broad thumbs and broad great toes, normal intelligence, and only mild soft-tissue syndactyly of the toes and fingers. The skull deformities are the same as seen in the typical Apert syndrome, with a severe increase in height of the skull and premature sutural synostosis. Other findings that are

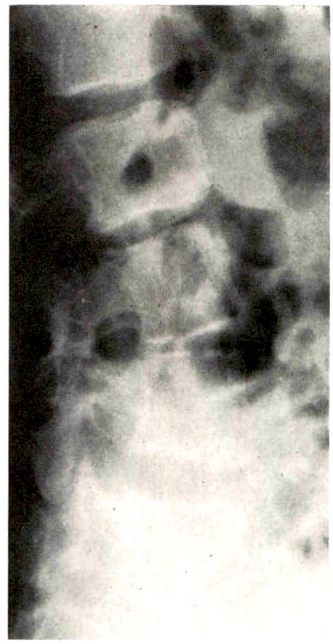


Fig. 12. Case II. Lateral view of the lumbar spine. Partial fusion of the anterior and posterior elements of the fourth and fifth lumbar vertebrae is similar to that seen in the mother.

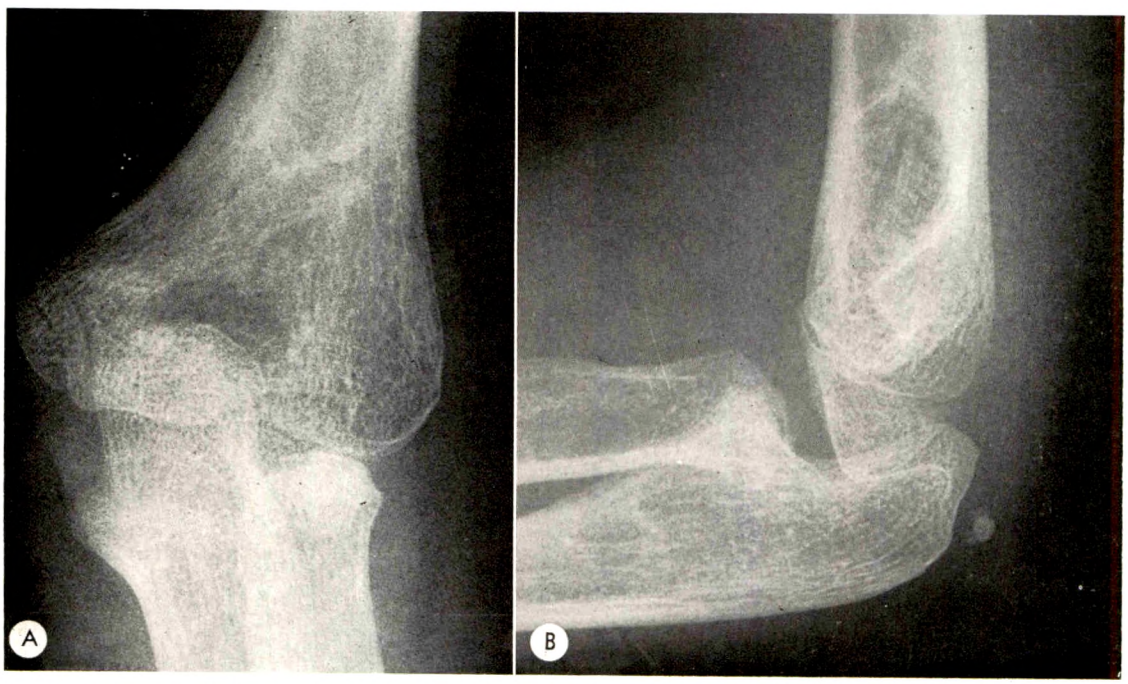


Fig. 13. Case II. (*A*) Anteroposterior and (*B*) lateral projections of the elbow. The normal anterior angulation of the distal humerus has been lost, and all the bones of the elbow joint are hypoplastic.



FIG. 14. Case III. (*A* and *B*) Views of face and head showing maxillary hypoplasia, wide forehead, and increased inner canthal distance.

characteristic of the Pfeiffer syndrome include a flat nasal bridge, hypertelorism, antimongoloid slant of the palpebral fissures, high-arched palate, varus deformities of the great toes, brachymesophalangia, trapezoidal shape of the proximal phalanx of the thumb, and a deformed, wide first metatarsal. The mother and affected siblings of this report had all these features. Additional roentgenographic features found in this family, not reported previously in cases of the Pfeiffer syndrome included: Arnold-Chiari malformation (Case II), congenital fusions of the cervical spine (Cases II and III) and of the lumbar spine (Cases I and II), coxa valga (Cases I, II, and III), and widening of the symphysis pubis



FIG. 15. Case III. A broad thumb, markedly shortened second and fifth middle phalanges, and abnormal distal end of the proximal phalanx of the thumb are seen in the hand.

(Cases I, II, and III). All these features except the Arnold-Chiari malformation have been described previously in the typical Apert syndrome.[6,7,23]

Another prominent feature in the 3 members of this family was the severe coalition of the tarsal bones. Fusions of the tarsal bones were described in previously reported cases of the Pfeiffer syndrome, but not emphasized.[17,38] Schauerte and St-Aubin[25] stressed the progressive nature of the synosteoses in the classic Apert syndrome and presented examples of this condition in the feet, hands, and cervical spine. Points of roentgenographic interest besides the fusion of tarsal bones and vertebrae include hypoplasia of the elbow and shoulder joint, fusion of the interphalangeal joints of the thumbs, and selective shortening of the middle phalanges of the index and little fingers.

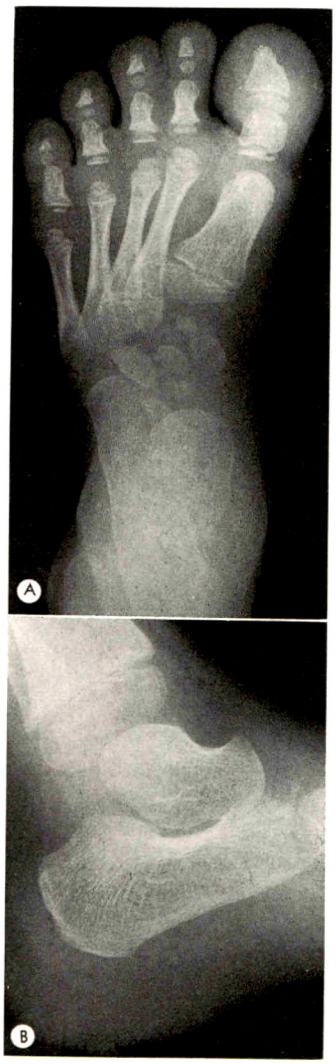


FIG. 16. Case III. (*A*) Anteroposterior and (*B*) lateral views of the foot showing absence of the epiphyses of the distal and middle phalanges before fusion has taken place. The first metatarsal is short and broad, and the shortened great toes are greatly widened. Developmental anomalies of the tarsal bones that lead to fusion are present. The calcaneus and cuboid are fused solidly.



FIG. 17. Case III. Fusion of the posterior elements of the third and fourth cervical vertebrae.

Except for one report,[28] all of the types of ACS are inherited in an autosomal dominant manner.[3,4,20,23,32] When polydactyly accompanies ACS, then the inheritance may be autosomal recessive or dominant (Carpenter's[5] and Noack's[18] syndrome, respectively).[31] Nearly all of the earlier reports of ACS were those of sporadic new mutations. The cases described by Weech,[36] van den Bosch[33] (typical ACS) and Waardenburg *et al.*[34] (atypical ACS) presented familial examples.

In 1964 Pfeiffer[21] presented a familial example of atypical ACS, and further reports by Zippel and Schüler,[38] Martsolf *et al.*,[17] and others, have established the entity as a genetically distinct form of atypical ACS. Recognition of a familial form of ACS in the young infant is of

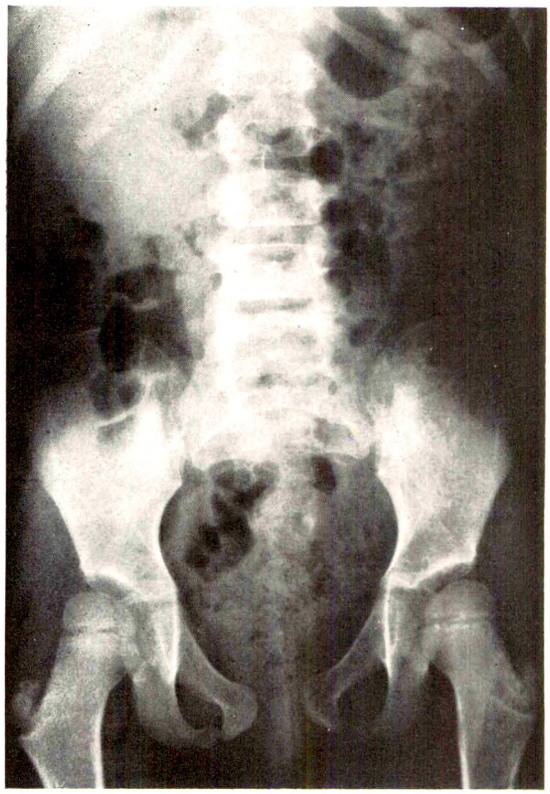


FIG. 18. Case III. Widening of the symphysis pubis and bilateral coxa valga.

obvious value in the prompt, correct genetic counseling of parents. The risk of occurrence in offspring of affected individuals is 50 per cent and there is no evidence for incomplete penetrance (unaffected individuals who carry the gene).

The etiology of acrocephalosyndactyly is not known. Blank[4] and Frerichs[11] implicated an increased paternal age—a known phenomenon in new mutant cases of achondroplasia. Recently, Dodson *et al.*[9] described a typical case of the Apert syndrome which was associated with a Group A chromosomal aberration. In their review of chromosomal analysis in patients with ACS, 3 abnormal chromosomes were discovered among 20 patients examined. All of these were in the typical form of ACS (Apert syndrome). Although all of the aberrations in ACS involved the Group A chromosome, the described abnormalities in each patient were different, and a specific chromosomal abnormality cannot be correlated with the Apert syndrome. In the family with Pfeiffer syndrome herein reported, no chromosome abnormalities were detected. In other instances of this syndrome, Pfeiffer,[21,22] and Zippel and Schüler[38] reported that their analysis also disclosed normal chromosomes. Other causes of the Apert syndrome have been suggested. Pilosebaceous abnormalities of the forearms (acne vulgaris) in 7 postpubertal patients with the Apert syndrome were reported by Solomon *et al.*[27] Since forearm acne is rare, the authors considered abnormal androgen metabolism as a possible etiologic factor in the Apert syndrome.

Other syndromes that frequently manifest broad thumbs and broad great toes are distinguished easily from the Pfeiffer syndrome by the lack of acrocephaly. The malformations of the thumb and great toe in the Rubinstein-Taybi syndrome[24,29,30] may be similar to those of the Pfeiffer syndrome. The frontodigital syndrome,[16] the oto-palato-digital syndrome,[15] Leri's pleonosteosis,[17] and Type B brachydactyly[32] also may include similar digital abnormalities. The acrocephalopolysyndactyly

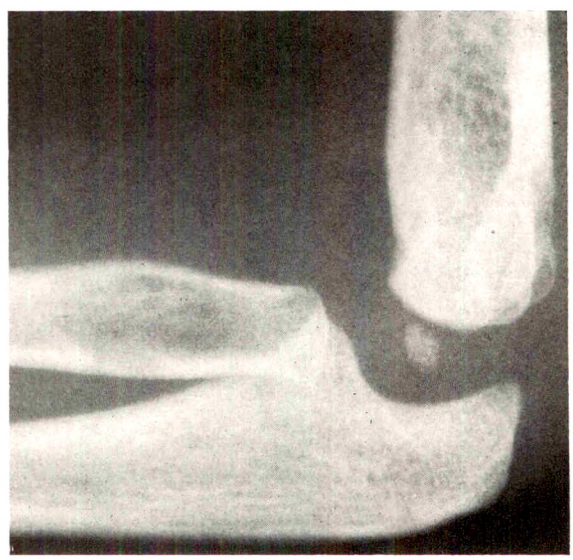


FIG. 19. Case III. Lateral view of the elbow demonstrates generalized hypoplasia of the bones of the elbow.

syndromes described by Carpenter[5] and by Noack[18] included cranial and digital abnormalities, but these syndromes are recognizable by the presence of preaxial polydactyly. The delta phalanx described by Jones[14] is closely related to the deformities in the great toe of our patients.

Although the malformations of acrocephalosyndactyly are predominantly of the head and distal extremities, a generalized skeletal dysplasia is clearly present. Deformities may be present in the shoulder, elbow, pelvis, hips, and spine,[6,23,37] as in this family with the Pfeiffer syndrome. Actually, a generalized mesodermal defect is present in the Apert syndrome, with abnormal viscera, cleft palate, chamber-angle abnormalities, other ocular problems, and bifid uvula.[4,26,32]

The clinical and roentgenographic features of congenital ring constrictions of the digits, secondary to mechanical and amniotic bands, are superficially similar to deformities in the Apert syndrome, but they can and should be differentiated.[2,35]

SUMMARY

A family with acrocephalosyndactyly, which differs from classic Apert's syndrome, has been described.

The autosomal dominant manner of inheritance and the lack of interdigital osseous fusion clearly differentiates it from Apert's syndrome. Other characteristic features include broad thumbs and great toes, normal intelligence, and only mild soft-tissue syndactyly.

Additional roentgen findings are presented and a differential diagnosis is discussed.

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REFERENCES

1. Apert, E. De l'acrocéphalosyndactylie. *Bull. et mém. d. hôp. Paris*, 1906, *23*, 1310–1330.
2. Baker, C. J., and Rudolph, A. J. Congenital ring constrictions and intrauterine amputations. *A.M.A. Am. J. Dis. Child.*, 1971, *121*, 393–400.
3. Bartsocas, C. S., Weber, A. L., and Crawford, J. D. Acrocephalosyndactyly type 3: Chotzen's syndrome. *J. Pediat.*, 1970, *77*, 267–272.
4. Blank, C. E. Apert's syndrome type of acrocephalosyndactyly—observations on British series of thirty-nine cases. *Ann. Human Genet.*, 1960, *24*, 151–164.
5. Carpenter, G. Two sisters showing malformations of skull and other congenital abnormalities. *Rep. Soc. Study Dis. Child*, 1901, *1*, 110–118.
6. Cohn, B. N. True oxycephaly with syndactylism: case report. *Am. J. Surg.*, 1945, *68*, 93–99.
7. Cooper, R. Acrocephalosyndactyly with report of case. *Brit. J. Radiol.*, 1953, *26*, 533–538.
8. Crome, L. Critique of current views on acrocephaly and related conditions. *J. Ment. Sc.*, 1961, *107*, 459–474.
9. Dodson, W. E., Museles, M., Kennedy, J. L., Jr., and Al-Aish, M. Acrocephalosyndactylia associated with chromosomal translocation. *A.M.A. Am. J. Dis. Child.*, 1970, *120*, 360–362.
10. Duggan, C. A., Keener, E. B., and Gay, B. B., Jr. Secondary craniosynostosis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *109*, 277–293.
11. Frerichs, C. T. Apert's syndrome. *Nebraska M. J.*, 1967, *52*, 491–495.
12. Gorlin, R. J., and Sedano, H. Acrocephalosyndactyly (Apert's syndrome). *Mod. Med.*, 1969, *37*, 235–237.
13. Hoover, G. H., Flatt, A. E., and Weiss, M. W. Hand and Apert's syndrome. *J. Bone & Joint Surg.*, 1970, *52-A*, 878–895.
14. Jones, G. B. Delta phalanx. *J. Bone & Joint Surg.*, 1964, *46-B*, 226–228.
15. Langer, L. O., Jr. Roentgenographic features of oto-palato-digital (OPD) syndrome. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *100*, 63–70.
16. Marshall, R. E., and Smith, D. W. Frontodigital syndrome: dominantly inherited disorder with normal intelligence. *J. Pediat.*, 1970, *77*, 129–133.
17. Martsolf, J. T., Cracco, J. B., Carpenter, G. G., and O'Hara, A. E. Pfeiffer syndrome: unusual type of acrocephalosyndactyly with broad thumbs and great toes. *A.M.A. Am. J. Dis. Child.*, 1971, *121*, 257–262.
18. Noack, M. Ein Beitrag zum Krankheitsbild der Akrozephalosyndaktylie (Apert). *Arch. Kinderheilk*, 1959, *160*, 168–171.
19. Palacios, E., and Schimke, R. N. Craniosynostosis-syndactylism. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *106*, 144–155.

20. PARK, E. A., and POWERS, G. F. Acrocephaly and scaphocephaly with symmetrically distributed malformations of extremities: study of so-called acrocephalosyndactylism. *Am. J. Dis. Child.*, 1920, *20*, 235–315.
21. PFEIFFER, R. A. Dominant erbliche Akrozephalosyndaktylie. *Z. Kinderheilk.*, 1964, *90*, 301–320.
22. PFEIFFER, R. A. Associated deformities of head and hands. *Birth Defects Original Article Series*, 1969, *5*, No. 3, pp. 18–34.
23. PILLAY, V. K. Acrocephalosyndactyly in Singapore: study of five Chinese males. *J. Bone & Joint Surg.*, 1964, *46-B*, 94–101.
24. RUBINSTEIN, J. H. Broad thumbs syndrome—progress report 1968. *Birth Defects Original Article Series*, 1969, *5*, No. 2, pp. 25–41.
25. SCHAUERTE, E. W., and ST-AUBIN, P. M. Progressive synosteosis in Apert's syndrome (acrocephalosyndactyly): with description of roentgenographic changes in feet. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *97*, 67–73.
26. SEELENFREUND, M., and GARTNER, S. Acrocephalosyndactyly (Apert's syndrome). *A.M.A. Arch. Ophth.*, 1967, *78*, 8–11.
27. SOLOMON, L. M., FRETZIN, D., and PRUZANSKY, S. Pilosebaceous abnormalities in Apert's syndrome. *A.M.A. Arch. Dermat.*, 1970, *102*, 381–385.
28. SUMMITT, R. L. Recessive acrocephalosyndactyly with normal intelligence. *Birth Defects Original Article Series*, 1969, *5*, No. 3, pp. 35–38.
29. TAUSANOV, M., DUMA, H., and GRIVCEVA, N. Unusual form of acrocephalo-syndactyly resembling syndrome of Rubinstein and Taybi. *God. Zborn Med. Fak. Skopje*, 1968, *14*, 419–425.
30. TAYBI, H., and RUBINSTEIN, J. H. Broad thumbs and toes, and unusual facial features: probable mental retardation syndrome. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1965, *93*, 362–366.
31. TEMTAMY, S. A. Carpenter's syndrome: acrocephalopolysyndactyly: autosomal recessive syndrome. *J. Pediat.*, 1966, *69*, 111–120.
32. TEMTAMY, S. A. Genetic factors in hand malformations. Thesis, Johns Hopkins University, Baltimore, 1966.
33. VAN DEN BOSCH, J. Cited by Zippel and Schüler.[38]
34. WAARDENBURG, P. J., FRANCESCHETTI, A., and KLEIN, D. Genetics and Ophthalmology. Charles C Thomas, Publisher, Springfield, Ill., 1961, pp. 310–321.
35. WALSH, R. J. Acrosyndactyly: study of twenty-seven patients. *Clin. Orthop.*, 1970, *71*, 99–111.
36. WEECH, A. A. Combined acrocephaly and syndactylism occurring in mother and daughter: case report. *Bull. Johns Hopkins Hosp.*, 1927, *40*, 73–76.
37. WIGERT, V. Die Akrozephalosyndaktylie; Weiteres über die allgemeinen Skelettveränderungen. *Acta psychiat. neurol.*, 1932, *7*, 701–718.
38. ZIPPEL, H., and SCHÜLER, K. H. Dominant vererbte Akrozephalosyndaktylie ACS. *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, 1969, *110*, 234–245.

COCKAYNE'S SYNDROME*

ROENTGEN FINDINGS

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COCKAYNE'S syndrome is an uncommon form of inherited truncal dwarfism characterized by growth failure as well as neurologic retardation which usually begins in the second year of life.

Clinical description of this syndrome was first reported in 1936 by Cockayne,[3] and subsequently about 35 cases have been described, predominantly in the pediatric literature.[4–6,9,17,20,23,26,29] Normal at birth, the children show progressive mental retardation, muscle weakness and dermal photosensitivity. They have a characteristic senile facies due to closely spaced, sunken eyes in a narrow face with decreased subcutaneous tissue. Frequently there is deafness and progressive blindness from retinal degeneration and cataracts. Early severe dental caries has been an almost invariable feature.

The exact etiology of Cockayne's syndrome, apparently of autosomal recessive inheritance, has eluded investigators. Chromosomal and metabolic evaluation of reported cases has not as yet disclosed a consistent abnormality.

Roentgen findings were mentioned in part by MacDonald *et al.*,[15] but Land and Nogrady[13] in 1969 were the first to fully evaluate this condition from the roentgenographic standpoint. Alton *et al.*[1] have recently reported 3 cases in the radiologic literature.

Our purpose is to report 5 more cases of Cockayne's syndrome, stressing the specificity of roentgen findings in the well developed cases.

REPORT OF CASES

Case 1. S.W. is probably the most severely affected child that has been reported. This Caucasian male was first seen at age $5\frac{1}{2}$ months with bilateral cataracts, decreased muscle mass, spasticity, and sunken eyes. He was hospitalized at age $4\frac{1}{2}$ years and at age $6\frac{1}{2}$ years for multiple dental caries. On these 2 occasions he was extremely small in stature and demonstrated marked mental and motor retardation (Fig. 1, *A–C*).

Roentgenographic Findings. At age $5\frac{1}{2}$ months skull roentgenograms showed slight microcephaly and hypotelorism (Fig. 2, *A* and *B*). The chest was normal except for minimal posterior wedging of the mid thoracic vertebral bodies (Fig. 4*A*).

At age $6\frac{1}{2}$ years the roentgen findings have become marked. Since $5\frac{1}{2}$ months of age the skull showed essentially no growth (Fig. 2, *C* and *D*). The microcephaly was now severe, with a thick, coarse calvarium and a small sella. Many teeth have been extracted. The earlier hypotelorism now appeared more obvious.

The chest was quite abnormal at age $4\frac{1}{2}$ years with long, thin ribs and clavicles, marked increase in the anteroposterior diameter, a pectus carinatum deformity of the sternum, and severe soft tissue wasting. The slender clavicles had an unusual "handle bar" configuration, with a concentric curve to the acromion processes and distal clavicles. At age $6\frac{1}{2}$ years all of these chest findings were even more striking (Fig. 3, *A* and *B*).

The spine now showed severe posterior wedging of vertebral bodies with thoracic kyphosis and cervical lordosis (Fig. 3*B*). The vascular channels of the now flattened vertebral bodies were prominent.

The pelvis at age 6 years showed relatively small iliac wings with increased iliac angle ("antimongoloid" configuration or "infolding" of iliac wings). A bilateral coxa valga deformity was also present (Fig. 5*A*).

The long bones demonstrated slender, long diaphyses with dense cortices, while the metaphyses were widened and demineralized. The epiphyseal centers were large, thereby producing relatively large joints. The ossification cen-

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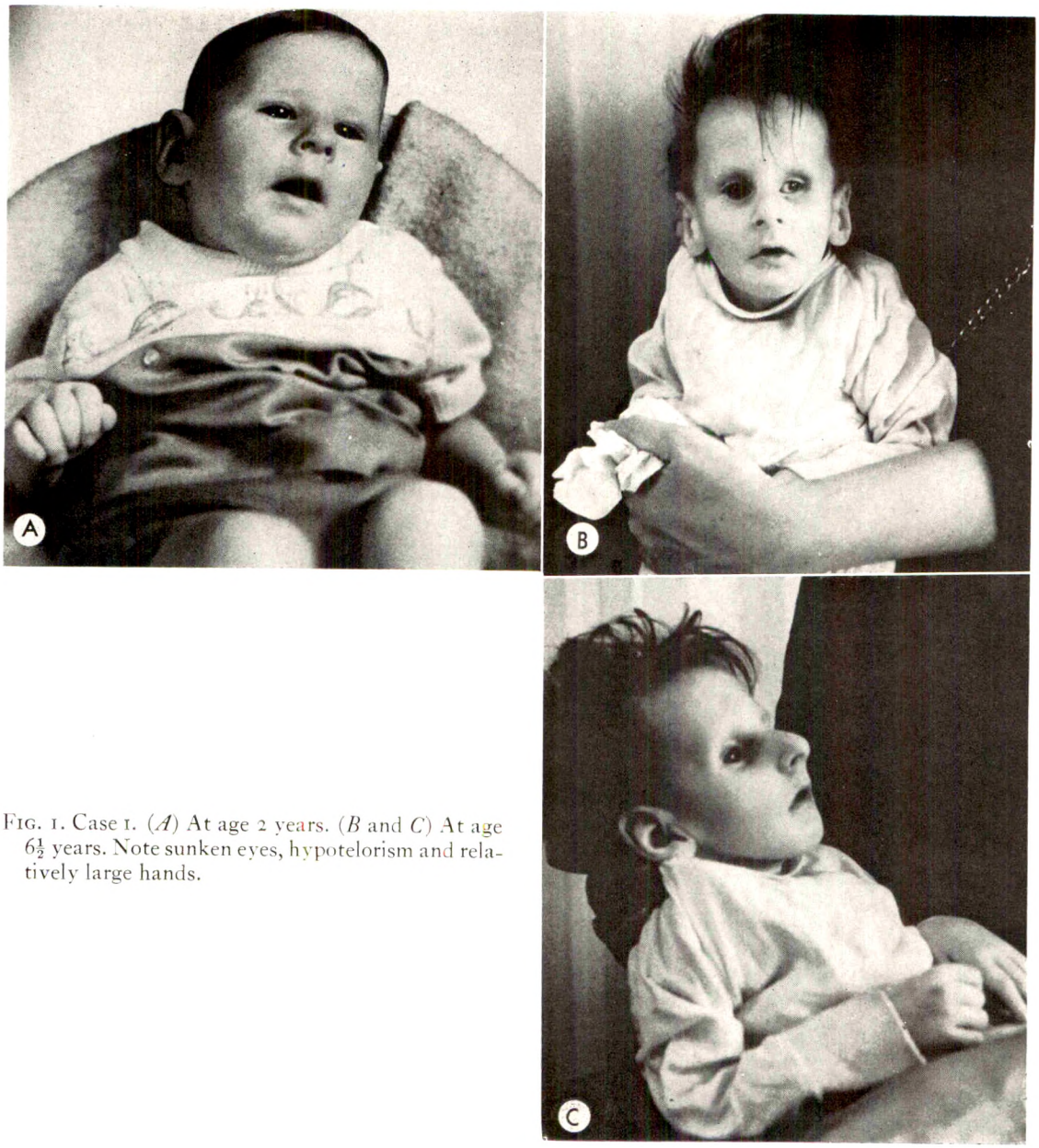


FIG. 1. Case 1. (*A*) At age 2 years. (*B* and *C*) At age 6½ years. Note sunken eyes, hypotelorism and relatively large hands.

ter for each greater trochanter was sclerotic at 6½ years (Fig. 5*A*). These changes were more dramatic in the large joints, where they were associated with flexion contractures and generalized osteoporosis (Fig. 8*A*).

The hands at 6½ years had demineralized carpal bones, but the epiphyseal centers for the distal phalanges (2 through 5) were punctate and sclerotic (Fig. 6*A*).

The tarsal bones were enlarged and there was sclerosis of the distal epiphysis of the second metatarsal (Fig. 6*B*). The second toe was shortened—a finding which has been noted in other cases of Cockayne's syndrome.[4,26]

Autopsy Findings. At age 7 years the child was received dead on arrival at the hospital. The major postmortem findings included panlobular pulmonary emphysema, acute and chronic bronchitis, undescended testes with atrophy, involution of thymus and fibrous intimal thickening of the aorta.

Microscopic renal findings were identical to those described in cases reported by Ohno and Hirooka.[22] There was arteriolonephrosclerosis

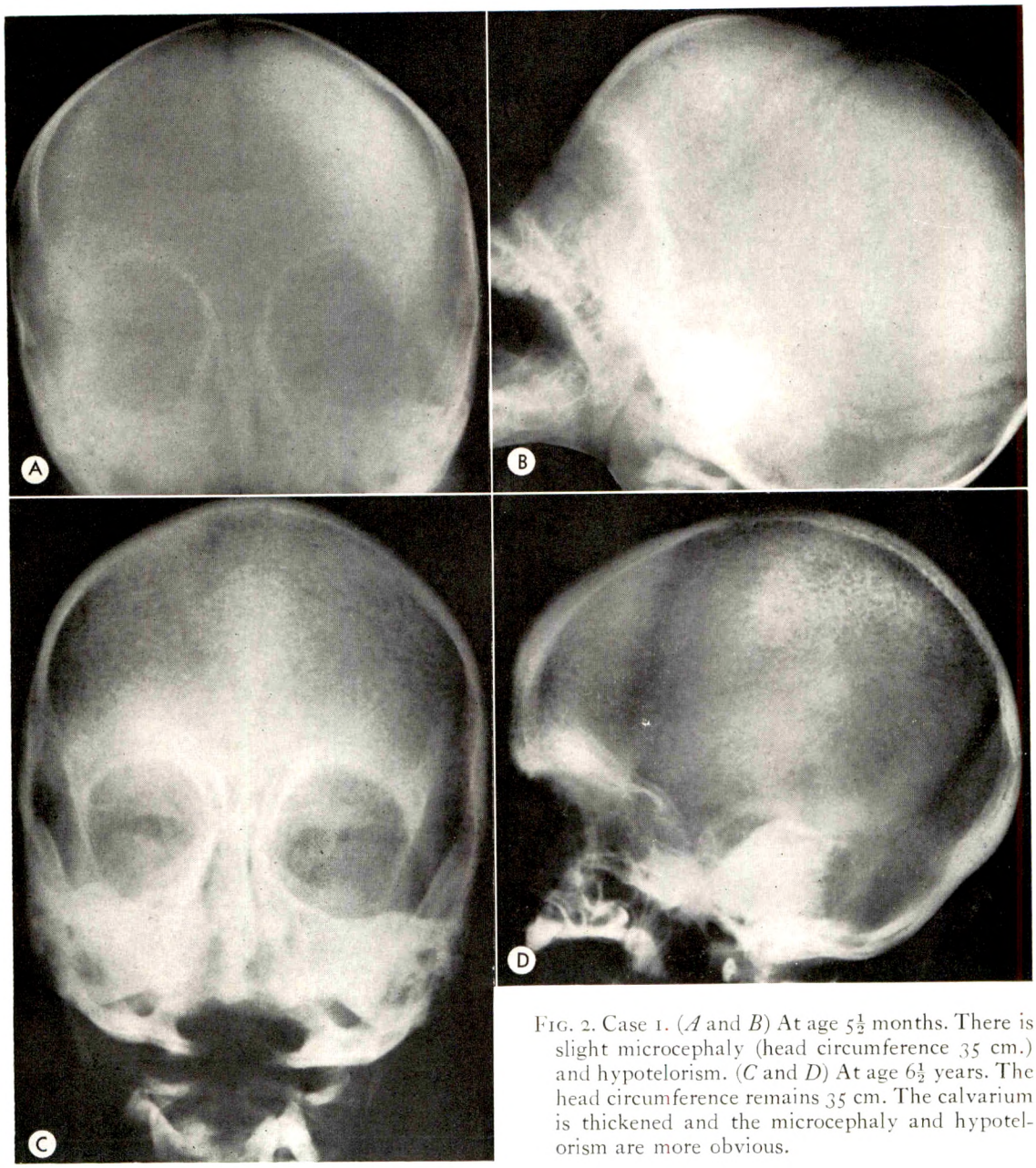


Fig. 2. Case 1. (*A* and *B*) At age 5½ months. There is slight microcephaly (head circumference 35 cm.) and hypotelorism. (*C* and *D*) At age 6½ years. The head circumference remains 35 cm. The calvarium is thickened and the microcephaly and hypotelorism are more obvious.

with capillary wall thickening, interstitial fibrosis, focal tubular atrophy, and hyalinized glomeruli.

Permission to study the brain was not granted.

Case II. G.C. is a Caucasian male who was normal at birth but was thought to have cerebral palsy at age 21 months because of severe mental and motor retardation. He was evaluated again at age 6½ years because of failure to thrive at which time he weighed only 14 lb., 7 oz. Clinically he had dental caries, decreased muscle mass, flexion contractures of the elbows, a cataract in the right eye, chorioretinitis, nystagmus, and microcephaly. A screening test showed aminoaciduria, but a 24 hour urine sample was normal.

Roentgenographic studies at age 3 years and at age 6½ years again showed the progression of

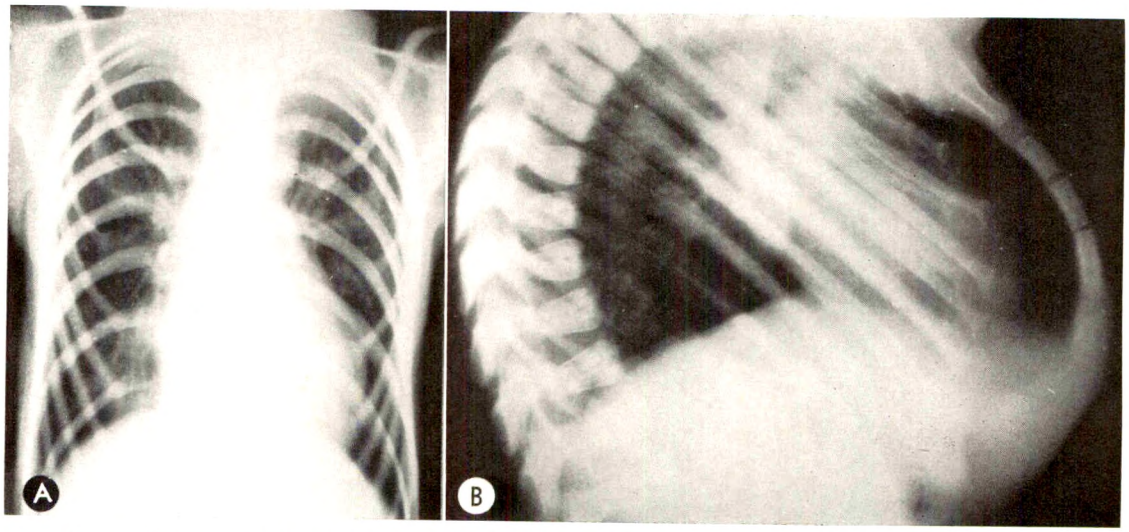


FIG. 3. Case I. (*A*) At age $6\frac{1}{2}$ years showing thin ribs and clavicles. (*B*) Note marked thoracic kyphosis and posterior tapering of vertebral bodies.

the typical findings seen in Case I. The long bone abnormalities were well demonstrated in this patient (Fig. 7). The diaphyses were slender with thickened cortices, widened metaphyses and enlarged epiphyses producing very large joints. The tibial epiphyses and capitellum appeared unusually large. In addition, the skull of this patient at 6 years demonstrated mottled calcification in the area of the paraventricular nuclei which was not present at 3 years. There were punctate calcifications in an enlarged spleen. Barium studies of the upper gastrointestinal tract and colon were normal.

CASE III. E.C. is the younger brother of the previous patient. He had congenital cataracts, but was considered otherwise normal at 9 months of age when his brother was initially diagnosed. However, when he was again seen at 28 months, the patient was still unable to walk or talk.

Roentgenograms at 9 months were obtained

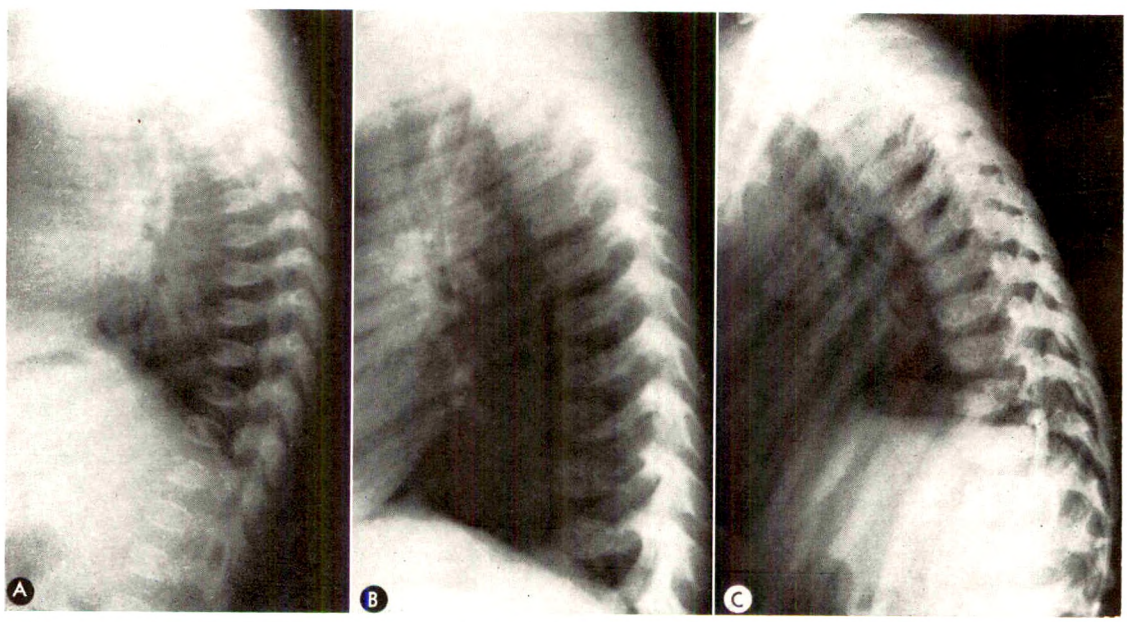


FIG. 4. (*A–C*) Three cases showing posterior wedging of vertebral bodies, prominent anterior vertebral vascular notches, and coarse demineralization. (*A*) Case I; (*B*) Case IV; and (*C*) Case V.

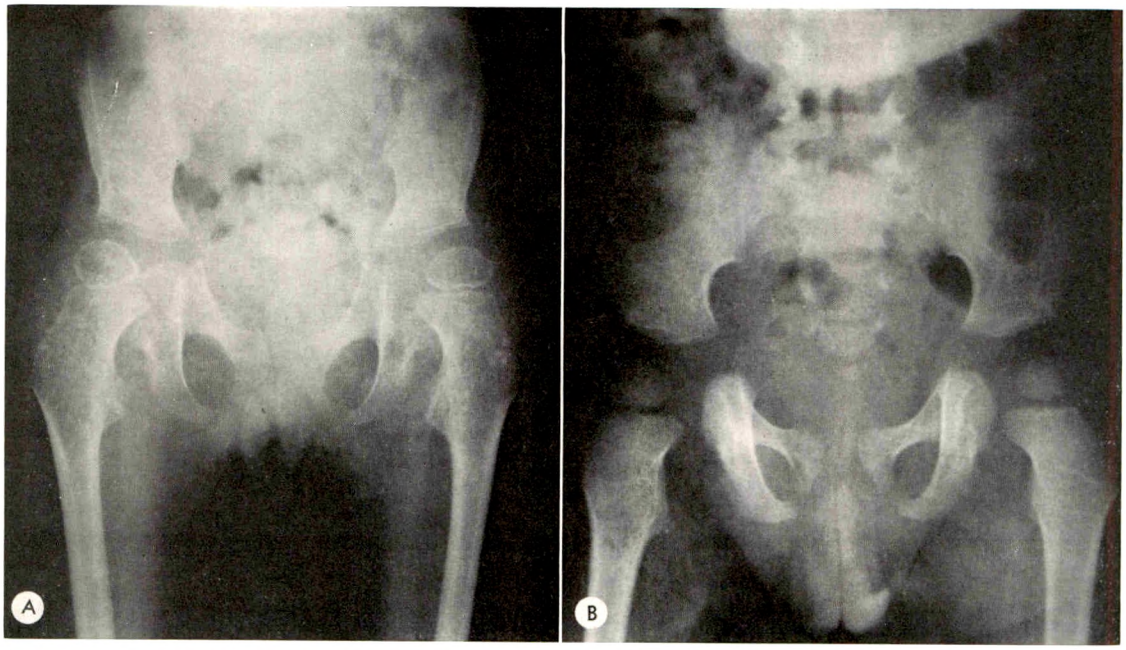


Fig. 5. (*A*) Case I at age 6 years showing large hip joints relative to size of small, almost vertical, iliac bones. Note sclerotic, punctate appearance of developing ossification centers for greater trochanters. Note coxa valga. (*B*) Case III at age 28 months. Note small iliac crests with increased iliac angle.

only of the joints for bone age, and no abnormality was seen. At 28 months, however, there was enough roentgen evidence of Cockayne's syndrome to establish the diagnosis. These changes probably represent the mildest and earliest roentgen findings. The skull and chest were normal except for minimal posterior wedging of the T7 through T9 vertebral bodies. There was now definite evidence of steepening of the iliac angle with small iliac crests (Fig. 5*B*). There was slight coxa valga. The diaphyses of the long bones were normal, but the metaphyses of the proximal humeri and distal femora were widened. There was slight sclerosis of the epiphyseal centers of the fourth metacarpals. There again appeared to be slight splenomegaly.

Case IV. K.W. is a Caucasian female admitted at age 2 years for dental surgery for severe caries. At this time she weighed 20 lb. and was mildly retarded with decreased muscle mass, poor coordination, spastic diplegia, dermal photosensitivity, nystagmus, and hyperpigmented retina. Urine amino acids were normal.

Roentgen findings at age 2 years were minimal with skull size at lower limits of normal, normal chest, posterior wedging of the T6 through T10 vertebral bodies, steep iliac angle, and slight sclerosis of the ossification center of the greater trochanters. There was slight angulation of the radial head, which was due to the enlarged elbow joint as well as contracture (Fig. 8*B*). Epiphyseal centers of the hands and feet were normal at this time.

At age 4 years there was progression of these findings into the fully developed syndrome as in the first 2 cases. Most striking was the complete lack of growth of the skull. There was also slight splenomegaly. Barium studies of the upper gastrointestinal tract and colon were normal. Intravenous urography showed slight enlargement of the left kidney.

Case V. This is a 9 year old white female who is the oldest patient reported in this series. She had normal early development except for a retarded ability to walk. She stopped growing at age 3 years and presented at age 9 years weighing approximately 20 lb. She also had bilateral cataracts, sunken eyes, nystagmus, dental caries, flexion contracture of joints, and tremor of hands and feet. A detailed clinical description of the patient has been previously published.[9]

Roentgen findings of the skull, chest and skeletal system were typical for Cockayne's syndrome. Most outstanding were the dense

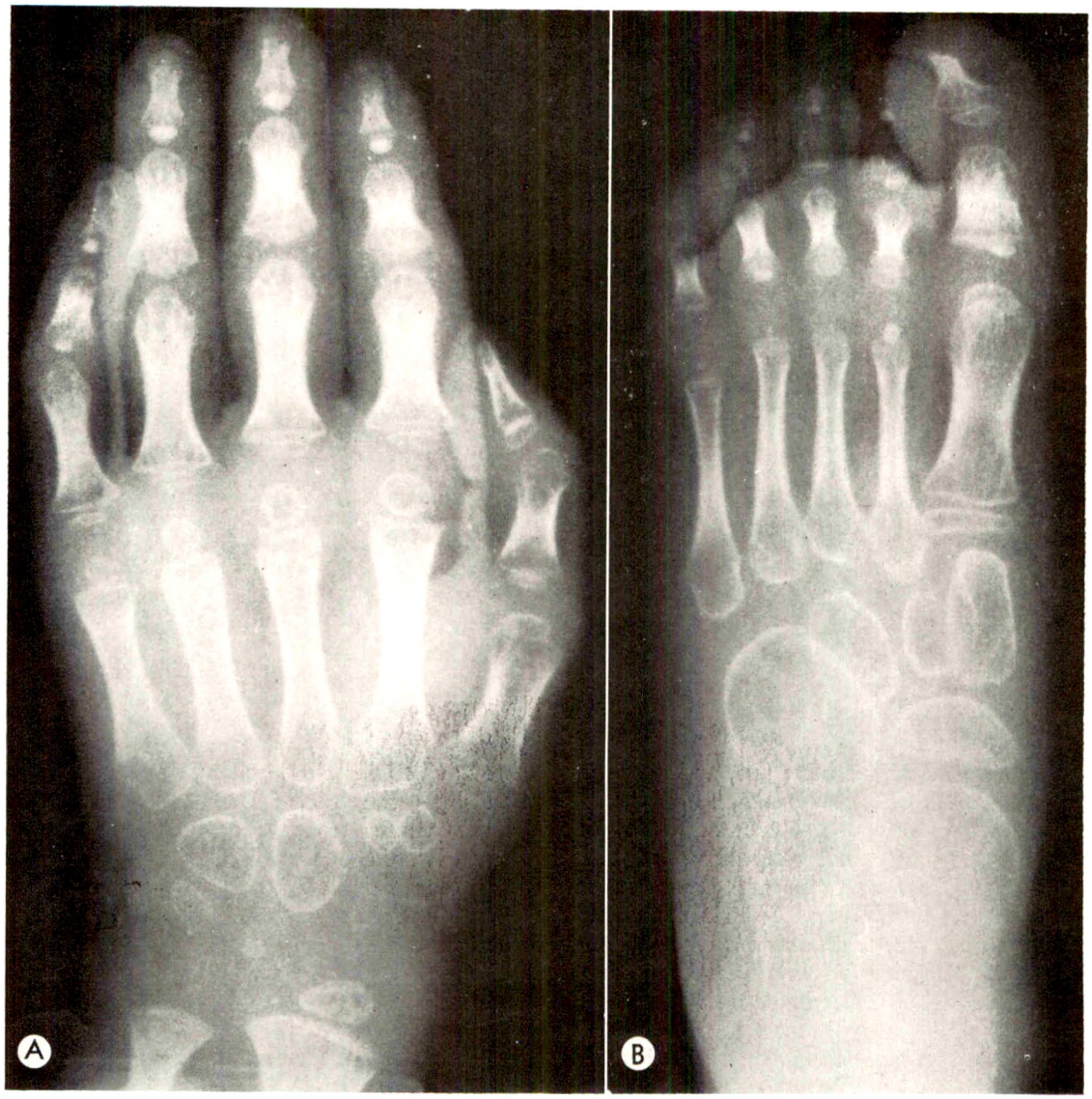


FIG. 6. Case 1. At age $6\frac{1}{2}$ years showing (*A*) sclerotic distal phalangeal epiphyseal centers, (*B*) enlarged tarsal bones and sclerosis of epiphyseal center of second metatarsal. Note that the second toe is short.

intracranial calcifications in the area of the paraventricular nuclei and frontal lobes (Fig. 9, *A* and *B*). This patient had anterior as well as posterior tapering of the vertebral bodies, creating a "biconvex" configuration (Fig. 4*C*).

BONE AGE DETERMINATIONS

Skeletal maturation has varied in the previously reported cases, ranging from normal[4,5,15,20] to advanced.[13,22,26] In our cases the bone ages, based mainly on the Greulich-Pyle atlas,[10] showed slight retardation in 3 cases, and there was normal bone age in 1.

Our Case V at 9 years of age showed normal maturation of carpal centers, but premature fusion of phalangeal centers (Fig. 10, *A* and *B*). Inspection of published hand roentgenograms suggests that this has occurred in other cases at this age.[1,22]

We consider many cases of Cockayne's syndrome to have a normal bone age during infancy and early childhood and to have

an advanced bone age in later childhood, reflecting the premature fusion of phalangeal centers.

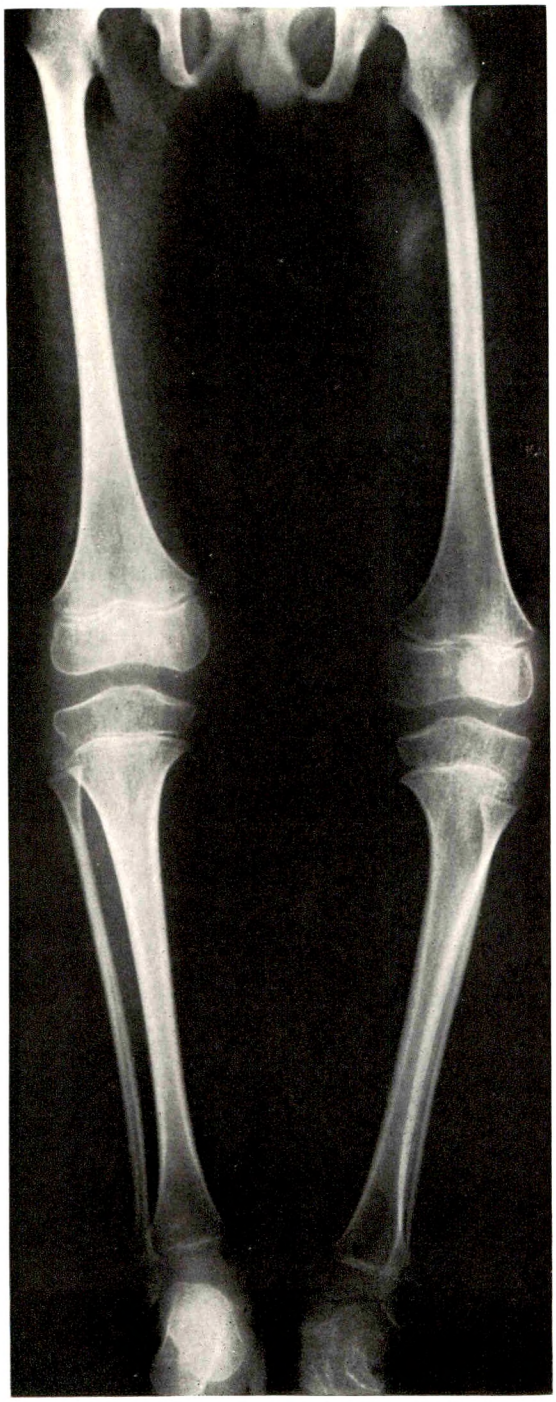


Fig. 7. Case II at age 6 years. Note slender diaphyses with relatively large epiphyseal centers.

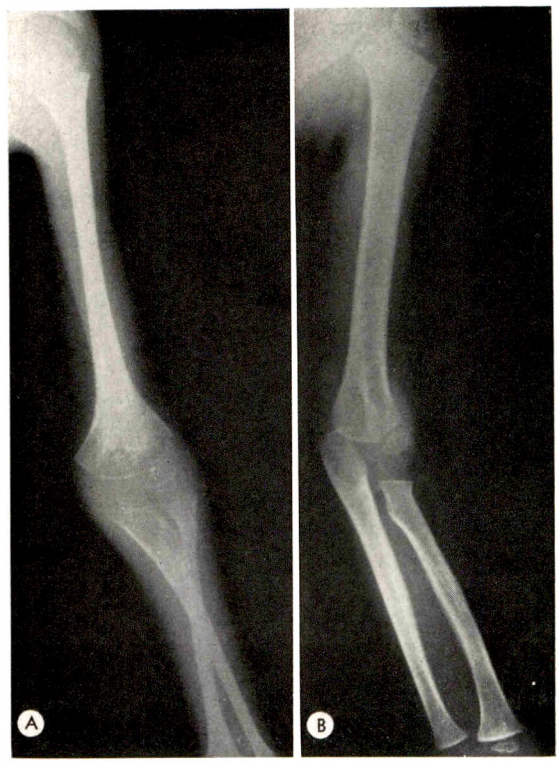


Fig. 8. (*A*) Case I at age $6\frac{1}{2}$ years showing enlarged elbow joint. (*B*) Case IV at age 2 years showing slight increase in slant of radial head.

DISCUSSION

Much investigation has failed to uncover the basic pathophysiology of this syndrome. Cotton *et al.*[5] recently reported a case having hyperglycemia and elevated plasma insulin. The authors speculate that increased growth hormone elaboration may occur. Fujimoto *et al.*[9] reported a case with hyperlipoproteinemia, hyperinsulinemia, renal disease, and normal growth hormone response.

We have considered some of the roentgen findings as clues to the paths along which research should be directed. Cerebral calcifications (particularly in basal ganglia) have been a frequent feature in reported cases and were present in 2 of our 5 cases. Basal ganglia calcifications have been related to decreased serum calcium.[12] Two of our cases had unexplained upper abdominal calcification, perhaps within the spleen. Alton *et al.*[1] reported a case with interver-

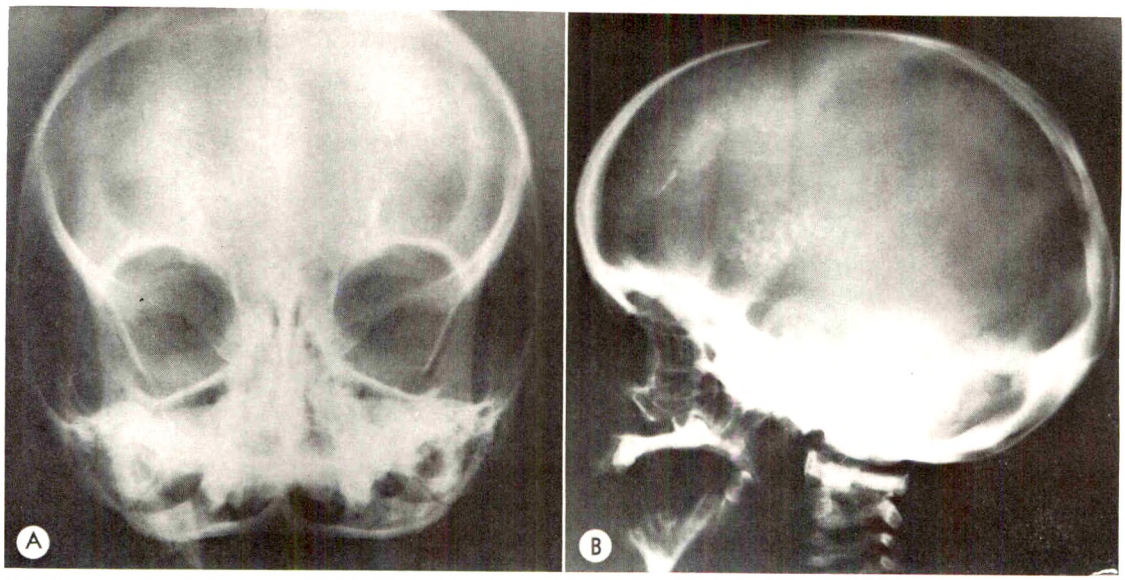


FIG. 9. (*A* and *B*) Case v at age 9 years showing calcifications in basal ganglia and frontal lobes. Note the absence of many teeth from severe caries.

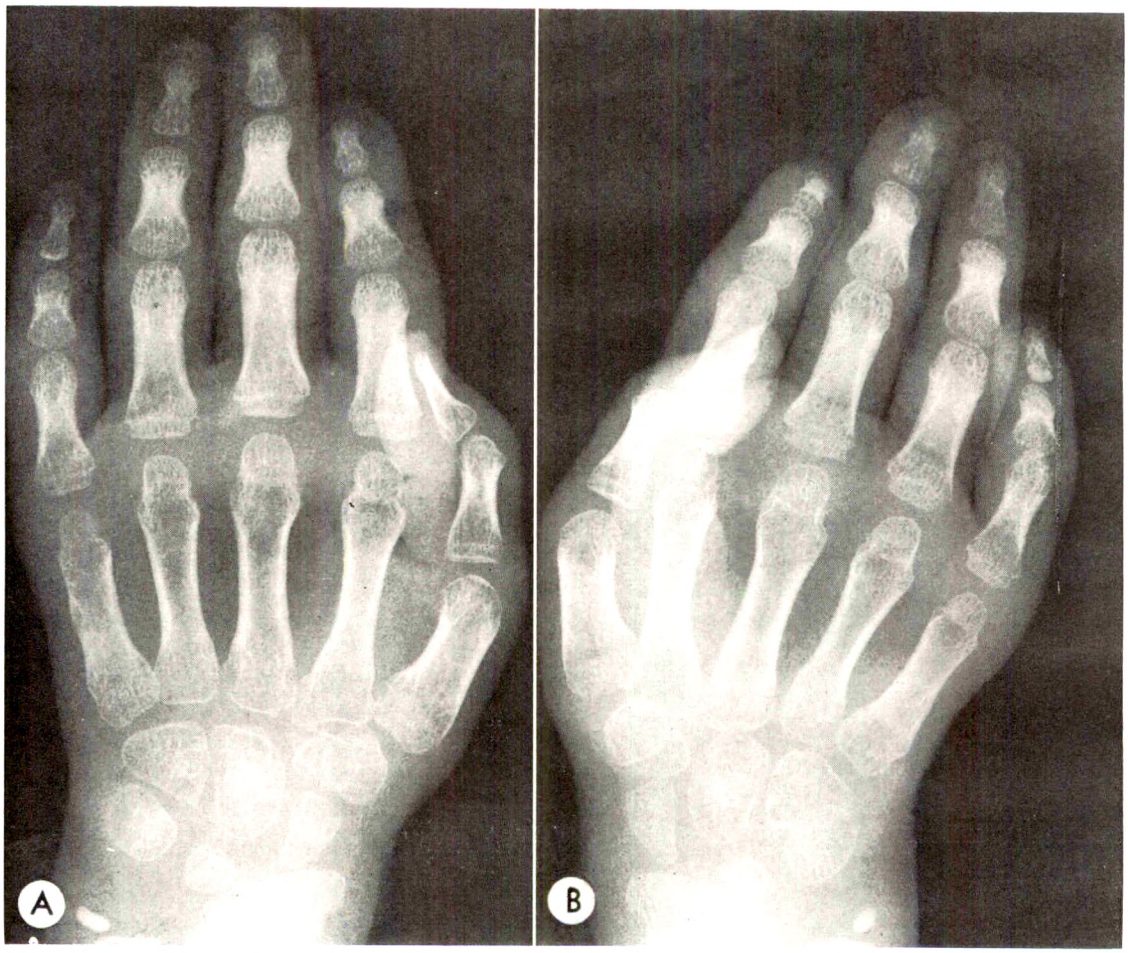


FIG. 10. (*A* and *B*) Case v at age 9 years showing premature fusion of phalangeal epiphyses. Note sclerosis of distal ulnar epiphyses.

TABLE I

COMPARATIVE SUMMARY OF COCKAYNE'S SYNDROME, HOMOCYSTINURIA, PROGERIA AND BIRD-HEADED DWARFS

	Cockayne's Syndrome	Homocystinuria	Progeria	Bird-Headed Dwarf, Seckel's Syndrome
Clinical onset	During second year	After 1 year	At 1 year	Prenatal—very low birth weight
Dwarfism	Truncal	None	Truncal	Proportionate
Dolichostenomelia (long, thin extremities)	Present	Present	Present	Present
Joint abnormalities	Large epiphyses in *tarsal* centers and in *knees*, *elbows*, and *ankles* with flaring of the associated metaphyses producing joint contractures	Large epiphyses in distal *carpal* centers and in *knees*, and *ankles* with flaring of metaphyses producing joint contractures	Capitellum sometimes large, periarticular fibrosis causing stiff joints	Dislocated radial head, congenital hip dislocation, absent patella
Osteoporosis	Present	Present	Variable	Present
Scoliosis	Present	Present	Variable	Present
Eye changes	Cataracts, retinal degeneration	Dislocation of lens, cataracts, retinal degeneration and detachment	Normal	Hypertelorism with ptosis of lids
Thoracic deformity	Pectus carinatum	Pectus excavatum and pectus carinatum	None	Defect in sternum
Skin	Photosensitivity, fine, fair hair	Malar flush, fine, fair hair	Thin skin, alopecia Brittle nails	Sparse hair
Mental retardation	Always	Frequent, often with psychosis	Absent	Frequent
Inheritance	Autosomal recessive	Autosomal recessive	Unknown	Autosomal recessive
Facies	Senile	Normal	Senile	Bird-headed–prominent nose
Unusual manifestations	Intracranial calcifications	Thromboemboli	Premature arteriosclerosis, thin calvarium and open fontanelles	Absence of epiphyses of phalanges and metacarpals
Teeth	Carious	Crowded, small	Delay in eruption of deciduous and permanent teeth	Hypoplasia of enamel, partial anodontia

tebral disk calcifications. Previous laboratory evaluation of calcium metabolism in reported cases has disclosed no abnormality, however.

There are a number of features that would suggest the possibility of Cockayne's syndrome being an inborn error of metabolism, such as a disturbance of amino acid metabolism. Patients with *alkaptonuria* have intervertebral disk calcifications and renal stones. Patients with *homocystinuria*[16,19] have elongated extremities, vertebral body abnormalities, frequent microcephaly, enlarged carpal bones and osteoporosis. These patients are normal during infancy and, like in Cockayne's syndrome, homocystinuria is inherited as a recessive trait. Both of these disorders are associated with cataracts and retinal degeneration. *Phenylketonuria* is characterized by progressive mental and physical degeneration in an infant who is normal at birth.[7] These infants also have fine, fair hair and often have eczema.

Cockayne's syndrome also can be compared to several other syndromes which

are characterized by abnormal growth patterns. Patients with *progeria*[20,25] are also dwarfs with thin long bones, coxa valga and ovoid vertebral bodies. Findings of premature aging are progressive, beginning after age 1 year. The normal intelligence and presence of a relatively large skull with multiple wormian bones tend to differentiate this disease from Cockayne's syndrome. Progeria cases have also shown no definite hereditary pattern.

Fitch *et al.*[8] have recently described a case of *bird-headed dwarfism* with features of premature senility. Bird-headed dwarfs differ from Cockayne's syndrome in that they have joint dislocations, a proportionate dwarfism, hypertelorism and a low birth weight indicating a prenatal abnormality.

A comparative summary of the clinical and roentgen findings of Cockayne's syndrome, homocystinuria, progeria, and bird-headed dwarfs is found in Table 1. Most of the entities involved in the differential diagnosis of Cockayne's syndrome are of an abiotrophic nature (the late onset of degenerative changes in a previously normal individual—McKusick[16]).

CONCLUSION AND SUMMARY

Cockayne's syndrome is a specific roentgenologic entity as well as a specific clinical entity, the radiologist being able to first suggest the diagnosis.

The earliest roentgen findings, usually becoming noticeable during the 2nd and 3rd years of life, are microcephaly, posterior tapering of thoracic vertebral bodies, and steepening of the iliac angle in a small pelvis. Later, these children often show intracranial calcifications, unusually slender clavicles and ribs, osteoporosis, relatively long extremities with large tarsal and carpal bones, and sclerosis of developing ossification centers in the extremities.

Diagnosis is important to initiate early genetic counseling with parents concerning future pregnancies. As radiologists are able to participate in the diagnosis, this disorder may be found to be more frequent than previously thought; hence, research will be stimulated to find the cause and treatment.

The roentgenographic and clinical similarities between Cockayne's syndrome and the inborn errors of metabolism such as the aminoacidurias are mentioned.

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We wish to thank Dr. John Buchignani at NIH for obtaining the roentgenograms of Case V and Dr. Tim Hayes for providing the autopsy findings in Case I.

REFERENCES

1. Alton, D. J., McDonald, P., and Reilly, B. J. Cockayne's syndrome: report of three cases. *Radiology*, 1972, *102*, 403–406.
2. Caffey, J. Pediatric X-ray Diagnosis: a Textbook for Students, and Practitioners of Pediatrics, Surgery and Radiology. Fifth edition. Year Book Publishers, Inc., Chicago, 1967.
3. Cockayne, E. A. Dwarfism with retinal atrophy and deafness. *Arch. Dis. Childhood*, 1936, *11*, 1–8.
4. Cockayne, E. A. Dwarfism with retinal atrophy and deafness. *Arch. Dis. Childhood*, 1946, *21*, 52–54.
5. Cotton, R. B., Keats, T. E., and McCoy, E. E. Abnormal blood glucose regulation in Cockayne's syndrome. *Pediatrics*, 1970, *46*, 54–60.
6. Cullen, M., and Mora, H. Sindrome de Cockayne. *Arch. argent. pediat.*, 1961, *55*, 16–21.
7. Feinberg, S. B., and Fisch, R. O. Roentgenographic findings in growing long bones in phenylketonuria. *Radiology*, 1962, *78*, 394–398.
8. Fitch, N., Pinsky, L., and Lachance, R. C. Form of bird-headed dwarfism with features of premature senility. *A.M.A. Am. J. Dis. Child.*, 1970, *120*, 260–264.
9. Fujimoto, W. Y., Greene, M. L., and Seegmiller, J. E. Cockayne's syndrome: report of case with hyperlipoproteinemia, hyperinsulinemia, renal disease, and normal growth hormone. *J. Pediat.*, 1969, *75*, 881–884.
10. Greulich, W. W., and Pyle, S. I. Radiographic Atlas of Skeletal Development of the Hand and Wrist. Second edition. Stanford University Press, Stanford, 1959.
11. Harper, R. G., Orti, E., and Baker, R. K. Bird-headed dwarfs, Seckel's syndrome. *J. Pediat.*, 1967, *70*, 799–804.

12. Hastings-James, R. Lenticulodentate calcifications. *Radiology*, 1970, *97*, 571–576.
13. Land, V. J., and Nogrady, M. B. Cockayne's syndrome. *J. Canad. A. Radiologists*, 1969, *20*, 194–203.
14. Lieberman, W. J., Schimek, R. A., and Snyder, C. H. Cockayne's disease. *Am. J. Ophth.*, 1961, *52*, 116–118.
15. MacDonald, W. B., Fitch, K. D., and Lewis, I. C. Cockayne's syndrome: heredofamilial disorder of growth and development. *Pediatrics*, 1960, *25*, 997–1007.
16. McKusick, V. A. Heritable Disorders of Connective Tissue. Third edition. C. V. Mosby Company, St. Louis, 1966.
17. Marie, J., Veveque, B., Hesse, J. C., and Buri, J. Nanism avec retinite pigmentaire et surdité: syndrome de Cockayne. *Semaine hôp. Paris*, 1958, *34*, 2808–2812.
18. Moossy, J. Neuropathology of Cockayne's syndrome. *J. Neuropath. & Exper. Neurol.*, 1967, *26*, 654–660.
19. Morreels, C. L., Jr., Fletcher, B. D., Weilbaecher, R. G., and Dorst, J. P. Roentgenographic features of homocystinuria. *Radiology*, 1968, *90*, 1150–1158.
20. Neill, C. A., and Dingwall, M. M. Syndrome resembling progeria: review of two cases. *Arch. Dis. Childhood*, 1950, *25*, 213–221.
21. Norman, R. M., and Tingey, A. H. Syndrome of microencephaly: striocerebellar calcifications, and leucodystrophy. *J. Neurol., Neurosurg. & Psychiat.*, 1966, *29*, 157–163.
22. Ohno, T., and Hirooka, M. Renal lesions in Cockayne's syndrome. *Tohoku J. Exper. Med.*, 1966, *89*, 151–166.
23. Paddison, R. M., Moossy, J., Derbes, V. J., and Kloepfer, W. Cockayne's syndrome: report of five new cases with biochemical, chromosomal, dermatologic, genetic, and neuro-pathologic observations. *Derm. Trop.*, 1963, *2*, 195–203.
24. Schimke, R. N., McKusick, V. A., Huang, T., and Pollack, A. D. Homocystinuria: studies of 20 families with 38 affected members. *J.A.M.A.*, 1965, *193*, 87–96.
25. Smith, D. W. Recognizable Patterns of Human Malformation. Volume VII. W. B. Saunders Company, Philadelphia, 1970.
26. Spark, H. Cachetic dwarfism resembling Cockayne's-Neill type. *J. Pediat.*, 1965, *66*, 41–47.
27. Uesugi, M., Nakagawa, O., and Uesugi, T. Case regarded as Cockayne's syndrome. *Rinsho Ganka*, 1960, *14*, 1777–1785.
28. Wilkins, L., Blizzard, R. M., and Migeon, C. J. The Diagnosis and Treatment of Endocrine Disorders in Childhood and Adolescence. Third edition. Charles C Thomas, Publisher, Springfield, Ill., 1965.
29. Windmiller, J., and Fink, C. W. Cockayne's syndrome with chromosomal analysis. *A.M.A. Am. J. Dis. Child.*, 1963, *105*, 204–207.

A CARDIO-FACIAL-SKELETAL SYNDROME*

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GOLDENHAR[6] in 1952 described a boy who had a dermoid tumor of the left eye and a small and imperfectly developed left auricle with 3 appendages and a sinus in front of the upper part of the helix. Three years earlier Cotterman and Falls[2] had reported similar abnormalities in a girl who also had abnormalities of several ribs and vertebrae; her sister had a seemingly unrelated defect—partial absence of a pectoralis major muscle.

Holt and Oram[9] in 1960 reported the case of a girl age 13 years who had triphalangeal thumbs, wandering pacemaker, and clinical evidence of an atrial septal defect. Her mother and her mother's sister had clinical signs of atrial septal defect; the former had abnormal thumbs, the latter had as the only skeletal abnormality a slight defect in the left clavicle. Her mother's brother, who also had abnormalities of both hands, was found at necropsy to have an atrial septal defect.

Cayler[1] in 1969 described the cardiofacial syndrome, which he found in 14 infants who had various congenital defects of the heart and unilateral facial palsy.

This paper has two purposes: (1) to report the case of a girl in whom features of these several syndromes are present; and (2) to show from cases in the literature that the Goldenhar, Holt-Oram, and cardiofacial syndromes may be part of a single syndrome, which occasionally includes congenital abnormality of the gastrointestinal tract

REPORT OF A CASE

T.K. (42-42-15), a girl born 27 hours after spontaneous rupture of membranes and 1 month prematurely, weighed 4 pounds, 6 ounces. Her mother had had vaginal bleeding, which had lasted for about 1 hour, several times during the second and third months of the pregnancy; during the fourth month she had had abdominal pain, anorexia, nausea, and fever for several days. She had taken various contraceptive pills until 2 months before her last menstrual period. She and the baby's father were 22 years of age and well. Two normal sons, ages 3 and 5 years, were the products of her only previous pregnancies. No familial disease was known.

The shape and position of the patient's ears were abnormal. When she cried, her mouth was pulled to the right side and only her right eye closed (Fig. 1, *A* and *B*). Preauricular tags of skin were present on both sides; a small tuft of hair grew from one on the right. The right side of the face and the right eye were smaller than the left. A dermoid tumor protruded from the sclera of each eye. The left lower lid was notched laterally. Her right thumb was abnormal (Fig. 2).

She had esophageal atresia with tracheoesophageal fistula; the fistula was divided, and the esophageal segments were anastomosed. Several ribs were asymmetrical; the left second rib was bifid (Fig. 3).

Several months later a loud systolic murmur was heard over the precordium; no diastolic murmur was present and the first and second sounds were normal. Peripheral pulses were normal. She was not cyanotic or tachypneic, and her liver was not enlarged.

At the age of 7 months she weighed 12 pounds. Development was slow, and her mother observed that she kept her head turned to the right and could not bring it to the midline. Roentgenographic examination showed bony fusion of the posterior parts of C2 and C3 (Fig. 4).

When she was 11 months of age, catheterization established the presence of a ventricular septal defect and infundibular pulmonic stenosis. Hematologic and biochemical values were normal.

Other abnormal findings were a bifid uvula, tonsils of unequal size, and partial soft tissue fusion of the second and third toes of both feet.

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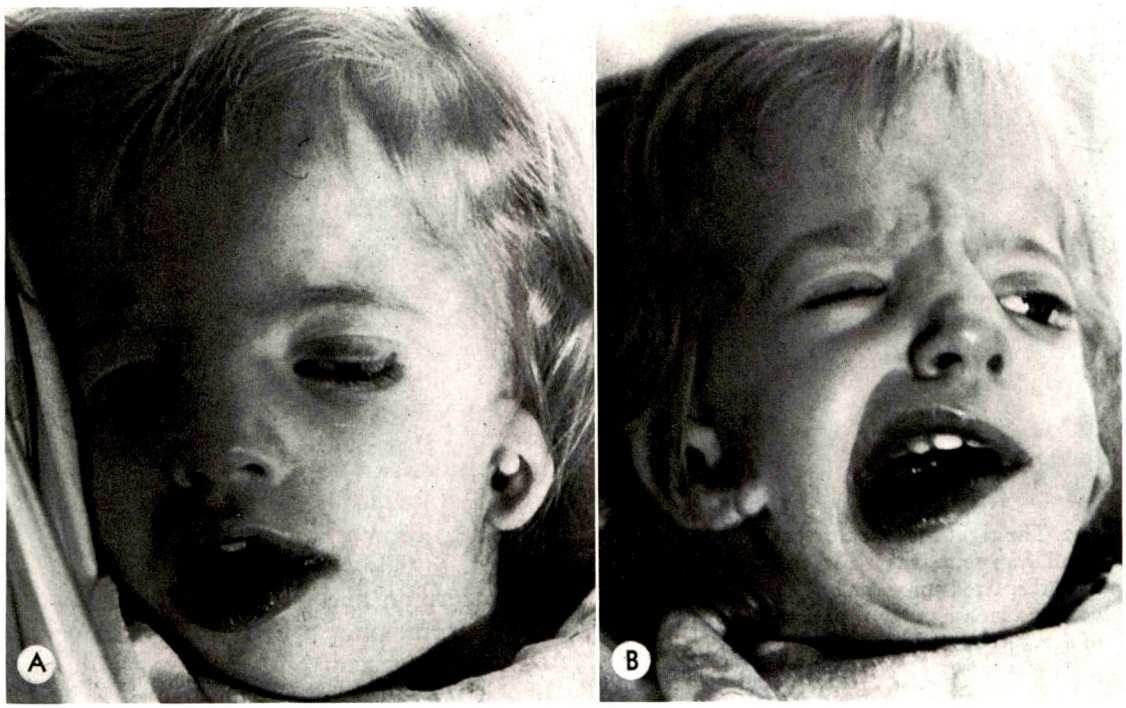


FIG. 1. (*A* and *B*) The mouth is pulled to the right when the patient cries and only the right eye is closed. The pinnas are abnormal; the left is smaller than the right. On the right side is a large preauricular appendage. At the time of this photograph the child was 1 year of age.

A recent angiocardiogram revealed that her pulmonary artery is hyperplastic and tapers to an end about 2 cm. from the hilus.

No other members of the mother's or father's families had similar external abnormalities. Her brother at the age of $6\frac{1}{2}$ years manifested diabetic ketoacidosis and now requires 4 units of regular insulin and 16 units of NPH insulin daily.

DISCUSSION

The features of the Goldenhar syndrome in our patient are epibulbar dermoids, abnormal ears, preauricular skin tags, and abnormal vertebrae.

Since the first description of the Holt-Oram syndrome of atrial septal defect and abnormal thumbs (but also in 1 of the 4 members of the family an abnormal left clavicle, and in another a short forearm with 4 digits), transmitted as an autosomal dominant, the syndrome has been extended by observations in 2 large families.[5,12,16] Several different congenital defects of the heart and skeleton exist separately and together in these families. The features of the Holt-Oram syndrome in our patient are ventricular septal defect with infundibular pulmonic stenosis and hypoplasia of a thumb.

The unilateral palsy of the muscles around the mouth and the cardiac defects that she has are features of the cardiofacial syndrome. In addition, our patient was born with esophageal atresia and tracheoesophageal fistula.

Three other patients who are being followed in our hospital may illuminate parts of a multi-faceted cardio-facial-skeletal syndrome. Diabetes mellitus is present in the families of 2 of the 3 mothers, and the third mother is diabetic and required insulin during the pregnancy which produced her affected son. In none of the 3 families have other abnormal children been born, and in each is another son, 2 of them older and 1 of them younger than their affected sibling.

The younger son born to the diabetic mother had imperforate anus with fistula.

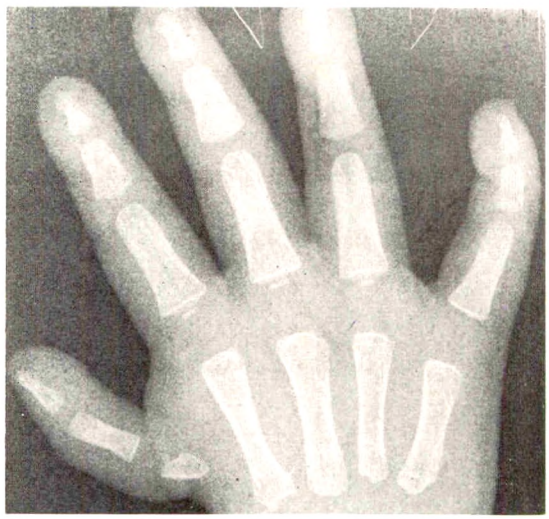


Fig. 2. The right first metacarpal bone consists of a small fragment; the right thumb is hypoplastic and displaced proximally. This roentgenogram was made when the patient was 13 months of age.

He has a hypoplastic lumbar vertebra and scoliosis, tetrad of Fallot, skin tags in front of the left ear, and left thumb smaller than the right. Another child, a boy whose maternal grandmother, maternal aunt, and maternal uncle have diabetes, has several hemivertebrae, a deformed left ear, a preauricular skin tag on each side of the face, left thumb smaller than the right, and atrial and ventricular septal defects. The third child is a girl whose maternal grandparents, maternal aunt, maternal uncle and maternal cousin have diabetes. She has a deformed left pinna with 1 preauricular tag of skin on the left side and 3 on the right side, left side of the mandible smaller than the right, right aortic arch, only a left kidney on excretory urographic examination and no right ureteral orifice on cystoscopic examination, and abnormal hands (Fig. 5, *A* and *B*). She also had imperforate anus with fistula to the vagina, and bilateral inguinal hernia.

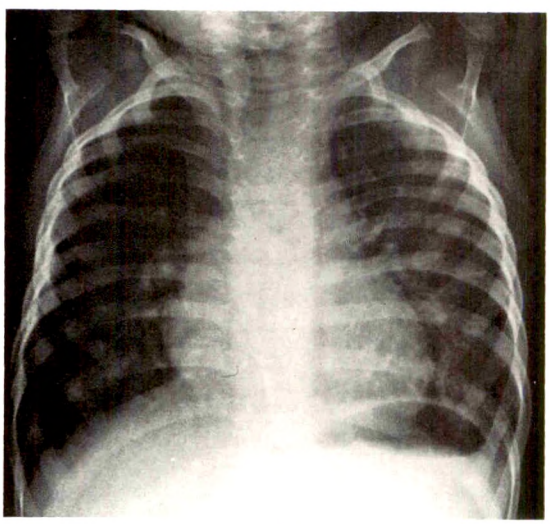


Fig. 3. Abnormalities of the ribs have become more apparent since the patient was first treated for esophageal atresia with tracheoesophageal fistula. Roentgenographic examination when she is 17 months of age shows asymmetry of several of the costal interspaces; the left second rib is bifid. The asymmetry suggests also vertebral abnormality, which is not apparent on the examination.

Cotterman and Falls[2] in 1949 described two sisters: one, age 9 years, had oculoauriculovertebral dysplasia and lesions characterized by absence of pigment in the form of unpigmented hairs among normally pigmented hairs in the frontal region of her scalp, and 2 halo nevi on her body; the other, age 7 years, had a halo nevus and aplasia of a large part of the right pectoralis major with a smaller right nipple. A boy in

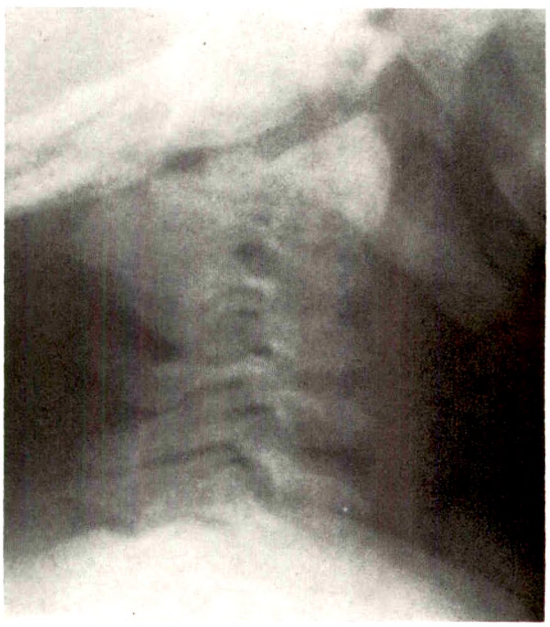


Fig. 4. C2 and C3 are abnormal; their posterior parts are fused.

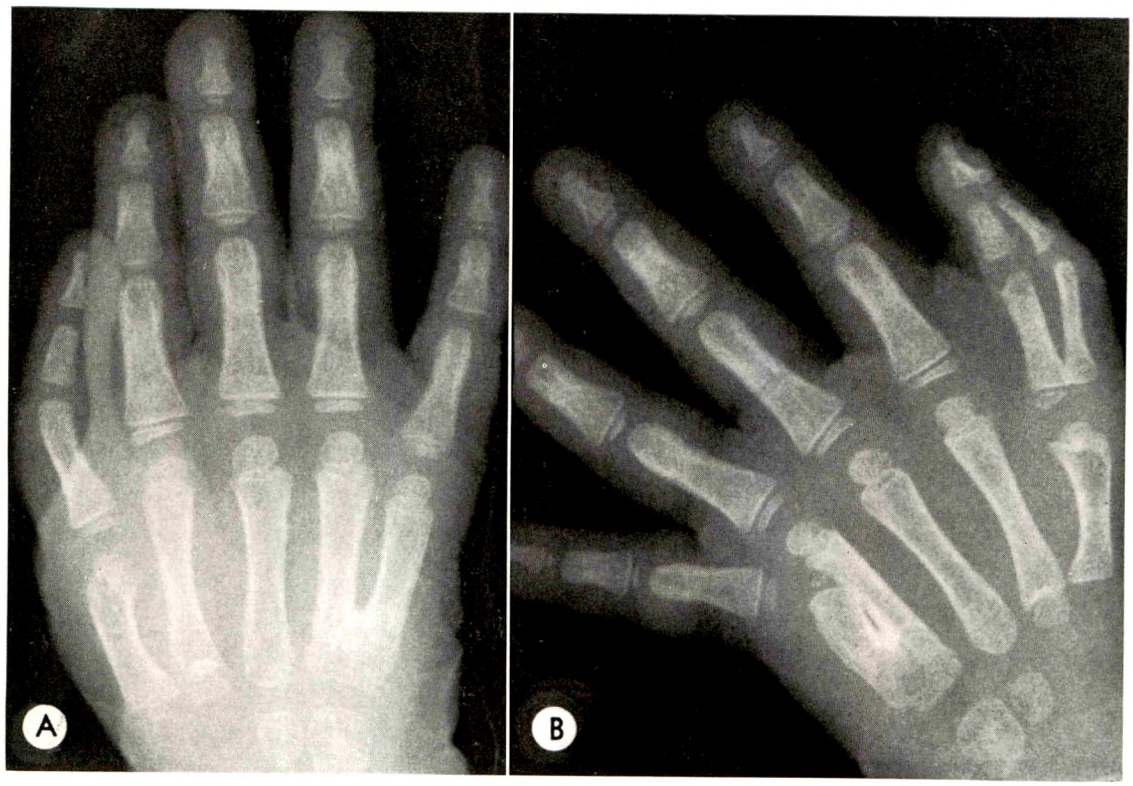


FIG. 5. (*A* and *B*) The right thumb has 3 phalanges; the left thumb is accompanied by a supernumerary thumb, and both have 3 phalanges. The fourth and fifth metacarpal bones are fused proximally.

the family with Holt-Oram syndrome which has been described by Lewis and colleagues,[12] had a normal heart, no thumbs, and hypoplasia of the pectoralis major muscles. Perhaps the 2 patients, 1 in each family, with absence of pectoral muscle, show a link between oculoauriculovertebral dysplasia in one and the Holt-Oram syndrome in the other.

Heikel[8] in an article on aplasia and hypoplasia of the radius refers to a description by Birch-Jensen of a man 18 years of age who lacked a radius, the right pinna and external auditory canal, and who had congenital heart disease. His cousin, a boy 4 years of age, had the same abnormalities.

Summitt[18] described a boy with deformed pinnas, preauricular appendages, epibulbar dermoid, malar and mandibular hypoplasia, and cleft palate. His sister had abnormal pinnas and a preauricular appendage. The father had abnormal pinnas, a preauricular appendage, and micrognathia. Six other members of the family, 2 male and 4 female, in 2 generations, are mildly affected. In this family with male-to-male transmission of the Goldenhar syndrome, the inheritance is autosomal dominant, as it is in the Holt-Oram syndrome.

Our patient with features of 3 syndromes is similar to others who have been described. Gorlin and colleagues[7] described 2 patients with Goldenhar's syndrome. One of them, a girl 9 years of age, also had left lower facial palsy, ventricular septal defect, agenesis of the left lung, and limp left thumb. She had 46 chromosomes, and the buccal smear was chromatin positive. Sugar[17] in a report of 3 cases of Goldenhar's syndrome described a girl 5 weeks of age with right bundle-branch block and weakness of the forehead and the left side of the lower part of the face. Opitz and Faith[14] described a premature girl with Goldenhar's syndrome and coarctation of the aorta; asymmetry of her face was thought to be due to a cleft of

the left side of the mouth and perhaps also to a left lower facial palsy.

In a case report of a boy with abnormalities of both pinnas, preauricular appendages, right facial palsy, vertebral abnormality, and clinical signs of atrial septal defect, Nair and Omana[13] cite the observation of Potter[15] that absence of large portions of the external ear is usually associated with defects of the middle ear, impaired or absent hearing, and aplasia of all or part of the nucleus of the facial nerve. Ford[4] has observed an association between weakness of the facial muscles and malformation of the external ear, along with club feet, defects of the pectoral muscles, syndactyly, defects of digits, and mental deficiency in some cases.

Of Cayler's 14 patients with congenital defects of the heart and unilateral lower facial palsy, 2 boys had aplasia of a radius; 1 of them also had imperforate anus; the other had facial asymmetry, a right "branchial cleft anomaly," and absence of the right kidney. A girl with tetrad of Fallot and patent ductus arteriosus had an abnormal right pinna.[1]

Other abnormalities may be associated. Five patients with agenesis of a lung were described by Ferguson and Neuhauser.[3] One had a malformed ear; 1 lacked a hand; and 1 had vertebral and rib abnormalities. Of 6 patients with the DiGeorge syndrome of thymic aplasia and hypoparathyroidism,[10,11] 1 had asymmetric and malformed ears, bifid uvula, right aortic arch, and aberrant left subclavian artery; 1 had malformed ears and tetrad of Fallot; 1 had right aortic arch, micrognathia, and abnormalities of the hands and feet; and 1 had esophageal atresia.

Although it may be misleading to draw conclusions based on resemblances, in the absence of knowledge of specific causes, the following hypothesis seems reasonable: in the fully afflicted individual the heart is abnormal; the hand, arm, clavicle, and/or spine are abnormal; the eye, the ear, maxilla, and mandible are abnormal; and part of the nucleus of the facial nerve is absent. Occasionally, gastrointestinal abnormality, esophageal atresia or imperforate anus with fistula is present. A lung or kidney may be absent. Further speculation, again based only on resemblances, leads to the conclusion that thymic and parathyroid deficiency may occur.

SUMMARY

Features of the syndromes of oculoauriculovertebral dysplasia (Goldenhar), congenital heart disease and abnormality of the upper limb (Holt-Oram), and congenital heart disease and facial palsy (Cayler) have jointly been present in a girl.

The association of these syndromes in one patient and the common features that are present in various case reports in the literature suggest that these are not separate syndromes, but parts of one syndrome.

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REFERENCES

1. Cayler, G. G. Cardiofacial syndrome. *Arch. Dis. Childhood*, 1969, *44*, 69–75.
2. Cotterman, C. W., and Falls, H. F. Unilateral developmental anomalies in sisters: status Bonnevie-Ullrich. *Am. J. Human Genet.*, 1949, *1*, 203–213.
3. Ferguson, C. F., and Neuhauser, E. B. Congenital absence of lung (agenesis) and other abnormalities of tracheobronchial tree. Am. J. Roentgenol. & Rad. Therapy, 1944, *52*, 459–471.
4. Ford, F. R. Diseases of the Nervous System in Infancy, Childhood, and Adolescence. Charles C Thomas, Publisher, Springfield, Ill., 1966, pp. 56.
5. Gall, J. C., Jr., Stern, A. M., Cohen, M. M., Adams, M. S., and Davidson, R. T. Holt-Oram syndrome: clinical and genetic study of large family. *Am. J. Human Genet.*, 1966, *18*, 187–200.
6. Goldenhar, M. Associations malformatives de l'oeil et de l'oreille, en particulier le syndrome dermoide epibulbaire—appendices auriculaires—fistula auris congenita et ses relations avec la dysostose mandibulo-faciale. *J. de Genet. Hum.*, 1952, *1*, 243–282.
7. Gorlin, R. J., Jue, K. L., Jacobsen, U., and Goldschmidt, E. Oculoauriculovertebral dysplasia. *J. Pediat.*, 1963, *63*, 991–999.
8. Heikel, H. V. Aplasia and hypoplasia of radius. *Acta orthop.*, 1959, Suppl. 39, 1–155.

9. Holt, M., and Oram, S. Familial heart disease with skeletal malformations. *Brit. Heart J.*, 1960, *22*, 236–242.
10. Kirkpatrick, J. A., and DiGeorge, A. M. Congenital absence of thymus. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *103*, 32–37.
11. Kretschmer, R., Say, B., Brown, D., and Rosen, F. S. Congenital aplasia of thymus gland (DiGeorge's syndrome). *New England J. Med.*, 1968, *279*, 1295–1301.
12. Lewis, K. B., Bruce, R. S., Baum, D., and Motulsky, A. Upper limb cardiovascular syndrome: an autosomal dominant genetic effect on embryogenesis. *J.A.M.A.*, 1965, *193*, 1080–1086.
13. Nair, N. S., and Omana, M. Rare triad of congenital malformations: bilateral microtia, spinal anomalies and heart affection. *Indian J. Pediat.*, 1963, *30*, 359–361.
14. Opitz, J. M., and Faith, G. C. Visceral anomalies in infant with Goldenhar syndrome. *Birth Defects* (Orig. Art. Ser.), 1969, *5*, 104–105.
15. Potter, E. L. Pathology of the Fetus and the Newborn. Year Book Publishers, Inc., Chicago, 1957, pp. 476.
16. Poznanski, A. K., Gall, J. C., Jr., and Stern, A. M. Skeletal manifestations of Holt-Oram syndrome. *Radiology*, 1970, *94*, 45–53.
17. Sugar, H. S. Oculoauriculovertebral dysplasia syndrome of Goldenhar. *Am. J. Ophth.*, 1966, *62*, 678–682.
18. Summitt, R. L. Familial Goldenhar syndrome *Birth Defects* (Orig. Art. Ser.), 1969, *5*, 106–109

OSSEOUS CHANGES OF OSTEOPATHIA STRIATA AND PYLE'S DISEASE OCCURRING IN A PATIENT WITH AN 11 YEAR FOLLOW-UP*

A CASE REPORT

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IN 1924, Voorhoeve[22] described the bone changes of osteopathia striata exhibited by 3 patients from 1 family. He suggested that the osseous defect was related to dyschondroplasia, osteopetrosis and osteopoikilosis. The disease is rare and there have been only isolated case reports.[1,3–6,11,13]

In 1931, Pyle[17] described a case of unusual bone development in a boy of 5 years. The features were splaying of the ends of the long bones, and skull changes of osteopetrosis. Since the initial description, cases have been reported with some variability in findings.[2,7–10,12,14–16,18,19–21] However, the general features are metaphyseal changes in long bones, skull changes of osteopetrosis with ocular hypertelorism, and definite familial tendency. Hence, the descriptive name of the disease is familial craniometaphyseal dysplasia.

Numerous papers have discussed the 2 diseases with case reports and presentations of theories for the osseous dysplasias.

This is the report of a case with features of both diseases.

REPORT OF A CASE

An 11 year old white girl was referred to the dental clinic because of maxillary and mandibular deformities. She had been seen at intervals since birth at another hospital where a diagnosis of craniometaphyseal dysplasia had been made. She was the youngest of 3 children. The parents and other children were normal and there was no family history of bone disease. At birth the skull was brachycephalic with ocular hypertelorism. Since birth, the patient has had nasal obstruction and hearing deficit. Both are due to osseous defects. Extensive and repeated blood chemistry studies have been normal.

Roentgenographic Findings (Fig. 1–5). A skeletal survey of 1971 was compared with roentgenograms from 1960 and 1968. Findings were much the same except for bone growth and changes in the epiphyses. The long bones showed some metaphyseal splaying which was more pronounced on the earlier roentgenograms. The most marked splaying was in the clavicles, medially. Vertical striations were mainly metaphyseal but extended into the epiphyses and the diaphyses. There were marked radial striations in the iliac bones. The skull showed marked thickening. There were no frontal sinuses and only rudimentary ethmoid air cells and maxillary antra. The sella turcica was small. The palate was high arched and the orbits were widely separated. The vertebrae were normal.

DISCUSSION

The reported case showed osseous changes of both osteopathia striata and Pyle's disease. There are metaphyseal splayings, particularly of the clavicles, plus the cranial changes described in Pyle's disease. The long bone striations and fan-like striations in the pelvis are typical of osteopathia striata.

Previously reported cases of osteopathia striata have exhibited associated changes of osteopetrosis. Hurt's case[11] was a man of 51 years who had typical metaphyseal striations. Rib biopsy showed the histology of osteopetrosis. Bloor[1] reported a case of osteopathia striata in a 3 year old girl with skull changes of osteopetrosis and ocular hypertelorism. Jones and Mulcahy[13] de-

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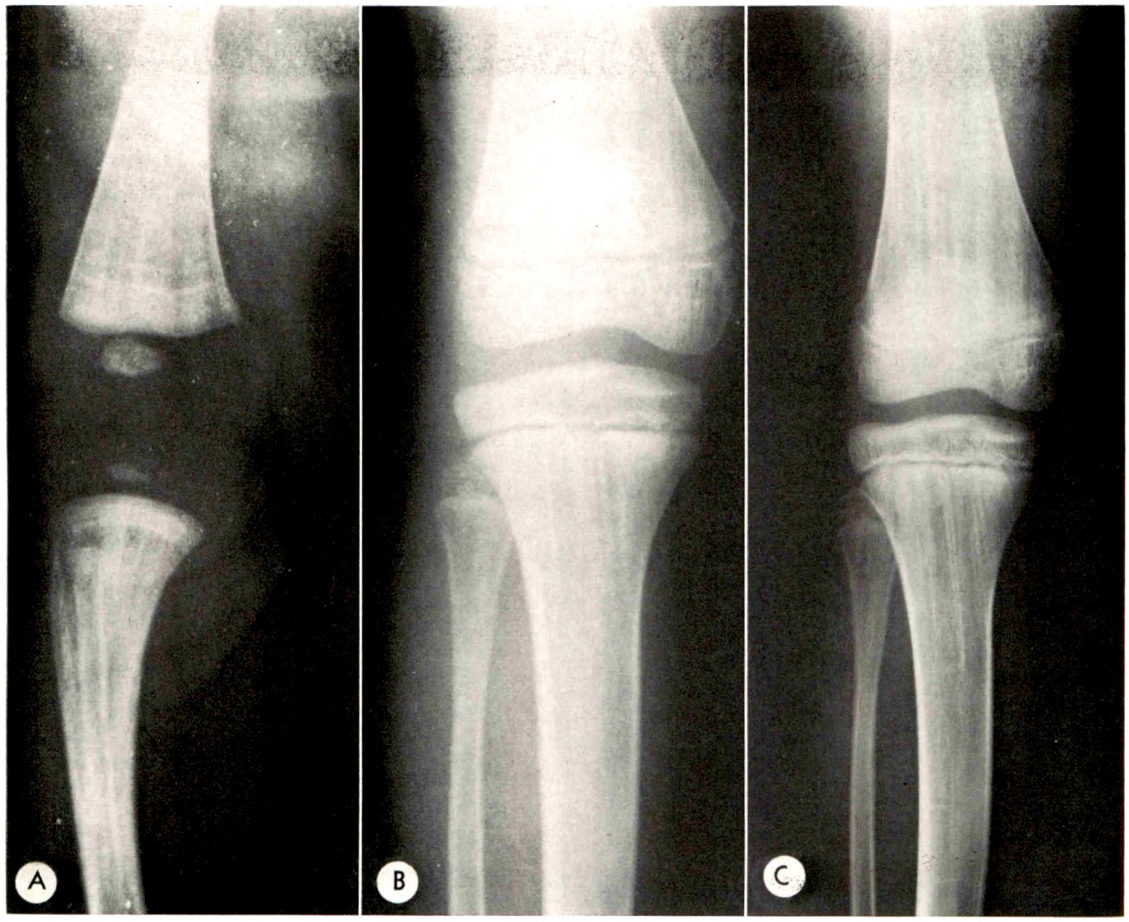


Fig. 1. (*A*) Left leg at $3\frac{1}{2}$ months. (*B*) Left leg at 8 years. (*C*) Left leg at 11 years.

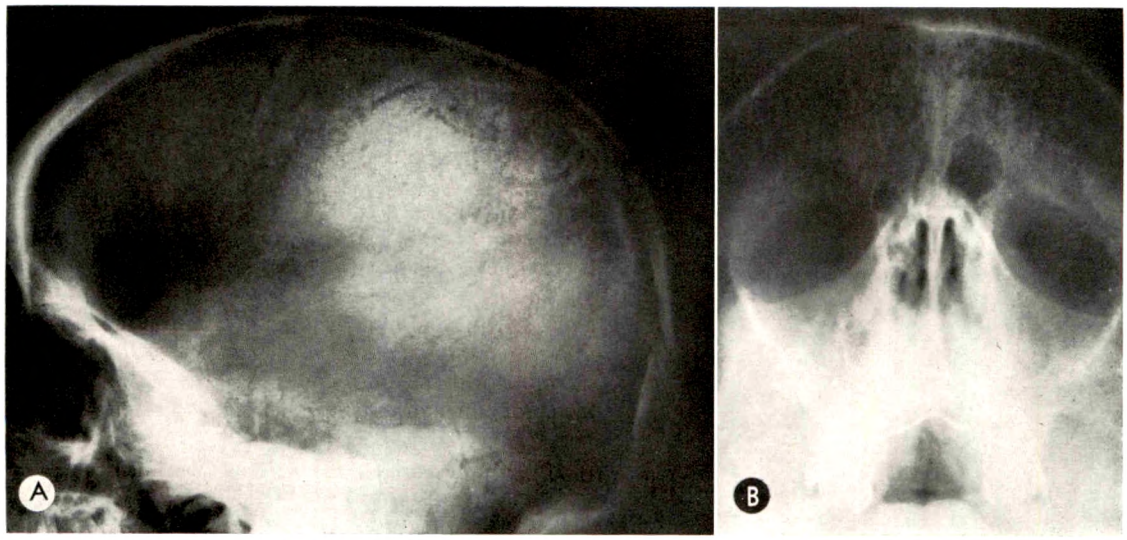


Fig. 2. (*A*) Lateral skull at 11 years. (*B*) Water's view of sinuses at 11 years.

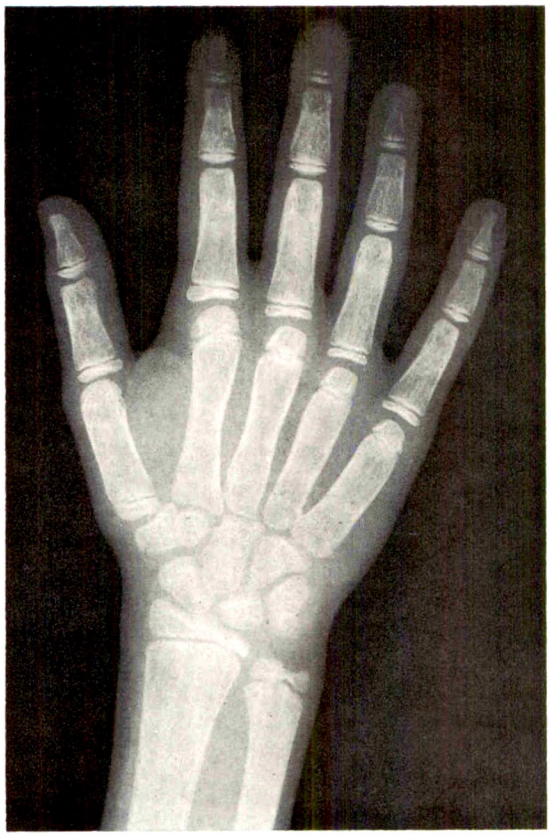


FIG. 3. Right hand at 11 years.

scribed the case of a 26 year old female who was seen because of hearing loss since birth. Her skull roentgenograms showed osteopetrosis and her long bones were typical of osteopathia striata. The case reported here



FIG. 4. Pelvis at 11 years.

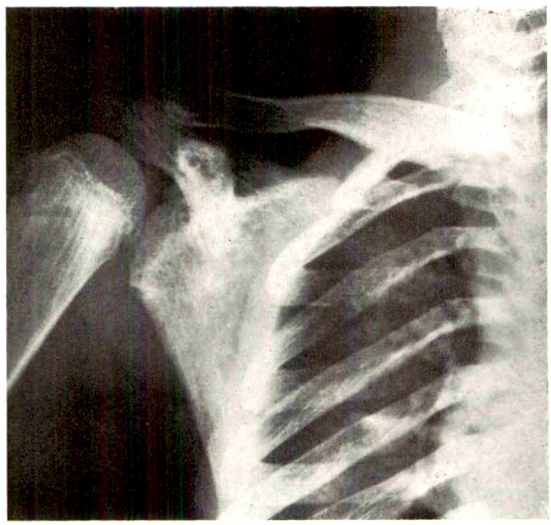


FIG. 5. Right shoulder girdle at 11 years.

is the first to show metaphyseal splaying in addition to striations.

SUMMARY

It would seem that osteopathia striata, osteopetrosis and osteopoikilosis are very closely related, as originally suggested by Voorhoeve. This case indicates that possibly Pyle's disease should be included in the group.

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REFERENCES

1. BLOOR, D. U. Case of osteopathia striata. *J. Bone & Joint Surg.*, 1954, *36-B*, 261–265.
2. BRONS, R., and KOUDSTAAL, J. Craniometaphyseal dysplasia. *Oral Path.*, 1969, *28*, 202–215.
3. D'ETTORE, A., and BORGHI, A. L'osteopathia striata. *Endocrinol Sc. Cost.*, 1967, *29*, 165–178.
4. FAIRBANK, H. A. Case of unilateral affection of skeleton of unknown origin. *Brit. J. Surg.*, 1925, *12*, 594–599.
5. FAIRBANK, H. A. T. Generalized diseases of skeleton. *Proc. Roy. Soc. Med.*, 1935, *28*, 1611–1619.
6. FAIRBANK, H. A. Osteopathia striata. *J. Bone & Joint Surg.*, 1950, *32-B*, 117–125.
7. FELD, H., SWITZER, R. A., DEXTER, M. W., and LANGER, E. M. Familial metaphyseal dysplasia. *Radiology*, 1955, *65*, 206–212.

8. Girdwood, T. G., Gibson, W. J., and Mackintosh, T. F. Craniometaphyseal dysplasia congenita: Pyle's disease in young child. *Brit. J. Radiol.*, 1969, *42*, 299–303.
9. Halliday, J. Rare case of bone dystrophy. *Brit. J. Surg.*, 1949, *37*, 52–63.
10. Hermel, M. B., Gershon-Cohen, J., and Jones, D. T. Familial metaphyseal dysplasia. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1953, *70*, 413–421.
11. Hurt, R. L. Osteopathia striata-Voorhoeve's disease: report of case presenting features of osteopathia striata and osteopetrosis. *J. Bone & Joint Surg.*, 1953, *35-B*, 89–96.
12. Jackson, W. P., Albright, F., Drewry, G., Hanelin, J., and Rubin, M. I. Metaphyseal dysplasia, epiphyseal dysplasia, diaphyseal dysplasia and related conditions. 1. Familial metaphyseal dysplasia and craniometaphyseal dysplasia, their relation to leontiasis ossea and osteopetrosis; disorders of bone remodeling. *A.M.A. Arch. Int. Med.*, 1954, *94*, 871–885.
13. Jones, M. D., and Mulcahy, N. D. Osteopathia striata, osteopetrosis and impaired hearing: case report. *A.M.A. Arch. Otolaryng.*, 1968, *87*, 116–118.
14. Kietzer, G., and Paparella, M. M. Otolaryngological disorders in craniometaphyseal dysplasia. *Laryngoscope*, 1969, *79*, 921–941.
15. Komins, C. Familial metaphyseal dysplasia: Pyle's disease. *Brit. J. Radiol.*, 1954, *27*, 670–675.
16. Mori, P. A., and Holt, J. F. Cranial manifestations of familial metaphyseal dysplasia. *Radiology*, 1956, *66*, 335–343.
17. Pyle, E. Case of unusual bone development. *J. Bone & Joint Surg.*, 1931, *13*, 874–876.
18. Ross, M. W., and Altman, D. H. Familial metaphyseal dysplasia: review of clinical and radiologic features of Pyle's disease. *Clin. Pediat.*, 1967, *6*, 143–149.
19. Rubin, P. Dynamic Classification of Bone Dysplasia. Year Book Publishers, Inc., Chicago, 1964, pp. 280–296.
20. Schwarz, E. Craniometaphyseal dysplasia. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1960, *84*, 461–466.
21. Small, P. O., and Wallis, J. J. Pyle's disease or craniometaphyseal dysplasia tarda. *Brit. J. Radiol.*, 1970, *43*, 811–813.
22. Voorhoeve, N. L'image radiologique non encore décrite d'une anomalie du squelette. *Acta radiol.*, 1924, *3*, 407–427.

ANEURYSMAL BONE CYSTS IN CHILDREN*

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IN an article on unicameral bone cysts in 1942, Jaffe and Lichtenstein[9] described 2 cases of a "peculiar blood-containing cyst of large size," which they referred to as an "aneurysmal cyst." This term refers to the roentgenographic appearance which typically shows ballooning of the bone with preservation of a thin rim of cortical bone in even the most extreme example.

A recent review by Dabska and Buraczewski[4] cited 193 cases of this entity. Following that review, several new series have appeared,[3,13] and even though some of them consisted in part of previously described cases[1,14] there are now well over 300 reported aneurysmal bone cysts. This growing body of published material shows that the aneurysmal bone cyst is no longer considered a rare entity and should be given increased consideration in the differential diagnosis of lytic lesions of bone.

Since 1957, the pathologic diagnosis of aneurysmal bone cyst has been made by the Pathology Department of Children's Hospital Medical Center on 28 occasions. Two of these later proved to be malignant tumors: one was made on a consultative basis, and since roentgenograms were available for review this patient is included in the present study.

In the 26 cases of verified aneurysmal bone cyst, the oldest patient was 17 years and the youngest 3 years. The mean age was 10.5 years. Eighteen aneurysmal bone cysts appeared in females and 8 in males. This distribution has been noted previously.[1,14] Ten cysts were found in long bones, 8 in the vertebra including the sacrum, 2 in the flat bones, 5 in the small tubular bones and 1 in the facial bones. This material is presented in Table I.

In the patients whose records were available for review, 11 children had some history of trauma. This is probably a chance occurrence, as is noted in other bone tumors.[14] Eight patients presented with fractures; 1 patient was reported to have had 5 fractures of the humerus over a 3 year period. Fourteen patients had palpable masses including 1 which was palpated or rectal examination. Six of the 8 spinal lesions presented with neurologic findings, and 7 required laminectomy. Only 2 cases

TABLE I

DISTRIBUTION OF ANEURYSMAL BONE CYSTS

Vertebral Column		8
Cervical	1	
Dorsal	2	
Lumbar	4	
Sacral	1	
Flat Bones		2
Pelvis	2	
Long Bones		10
Femur	3	
Tibia	4	
Fibula	2	
Humerus	1	
Small Tubular Bones		5
Calcaneus	2	
Metacarpal	2	
Phalanx	1	
Facial Bones		1
Zygoma	1	
Total		26

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required curettage after the initial treatment. One girl required evacuation of postoperative hematomas 3 times after the initial surgery, but she is now well. However, the fact that 4 of our 8 patients with spinal lesions retained or increased their neurologic deficits after surgery emphasizes the respect that these tumors must be given. All the cysts outside of the spine were treated with curettage and bone graft. The case involving the zygoma recurred, requiring a subtemporal decompression. Radiation therapy was used in only 1 patient, and then only postoperatively.

PATHOLOGY

The gross appearance of the aneurysmal bone cyst is a characteristic spongy mass made up of blood-filled spaces in a meshwork of connective tissue.

Microscopically, anastomosing cavernous channels are seen, most of which contain fluid blood. These channels are lined by prominent cuboidal cells resembling endothelium and by occasional multinucleated giant cells (Fig. 1). Similar giant cells and some foam cells and hemosiderophages are seen in the fibrous stroma. The connective tissue trabeculae often contain a small amount of new bone. The stromal cells appear thin and show no abnormal activity.

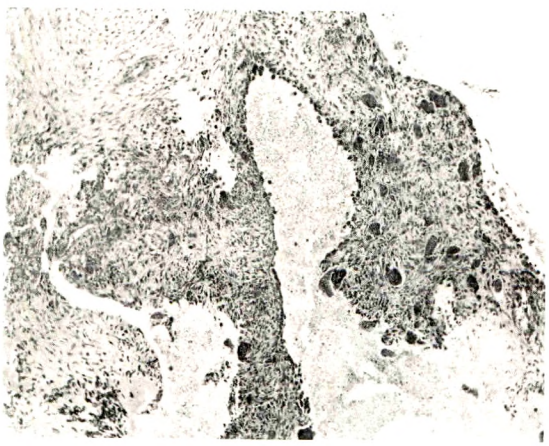


FIG. 1. The large cavernous channels containing red blood cells are shown lined by swollen cuboidal cells and occasional giant cells. (Hematoxylin and Eosin ×110.)

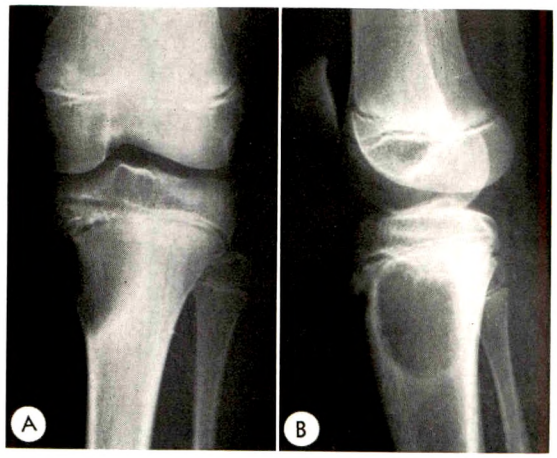


FIG. 2. (*A*) Anteroposterior roentgenogram of the left knee showing an eccentric lytic metaphyseal lesion with a thin but intact cortical rim. (*B*) Lateral projection of the same patient showing the well demarcated border with a thin sclerotic rim.

Pathologic diagnoses of aneurysmal bone cysts were offered initially in 2 cases later proven to be malignant. In both, the roentgenologic features were atypical for the diagnosis of aneurysmal bone cyst. A 6 year old girl with a lesion of the distal femur was initially diagnosed as telangiectatic osteosarcoma, a diagnosis which was overruled by 3 consultant opinions in favor of aneurysmal bone cyst. A second biopsy performed during the rapid progression of the lesion confirmed the original diagnosis of malignancy; this was probably an example of what has been termed malignant aneurysmal bone cyst. The second case included a highly vascular lesion of the ilium, proven on second biopsy to be an Ewing's tumor. It should be noted that areas resembling aneurysmal bone cyst are relatively common in the margins of a wide variety of other benign and malignant diseases of bone, but in this report the diagnosis of aneurysmal bone cyst is unequivocal.

ROENTGENOGRAPHIC FINDINGS

Long Bones. Roentgenograms of all 10 cysts showed smooth lytic lesions with well

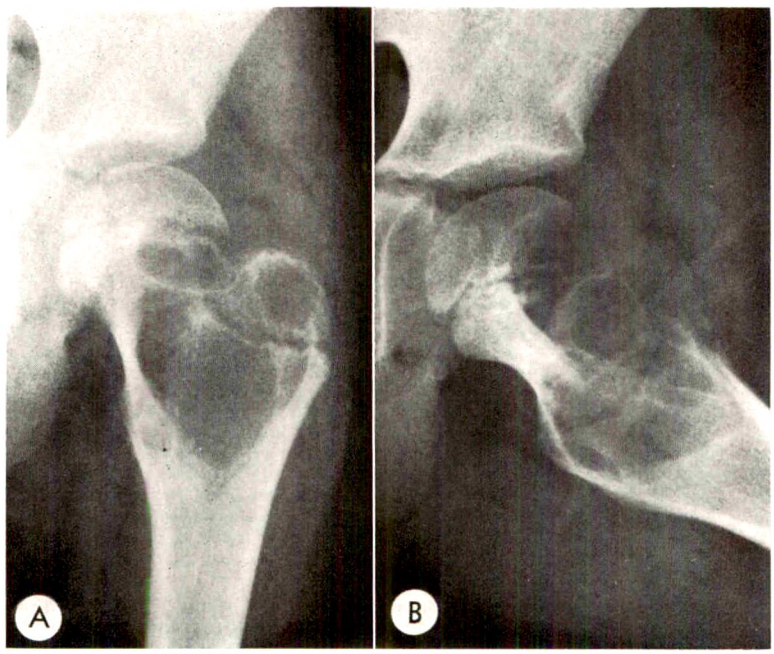


FIG. 3. (*A*) Anteroposterior roentgenogram of the left hip demonstrating a large lytic intertrochanteric lesion that extends along the neck and into both the capital femoral and greater trochanteric epiphyses. (*B*) Lateral view of the same patient showing fairly well demarcated borders of the cyst. The cortical rim is not readily identifiable.

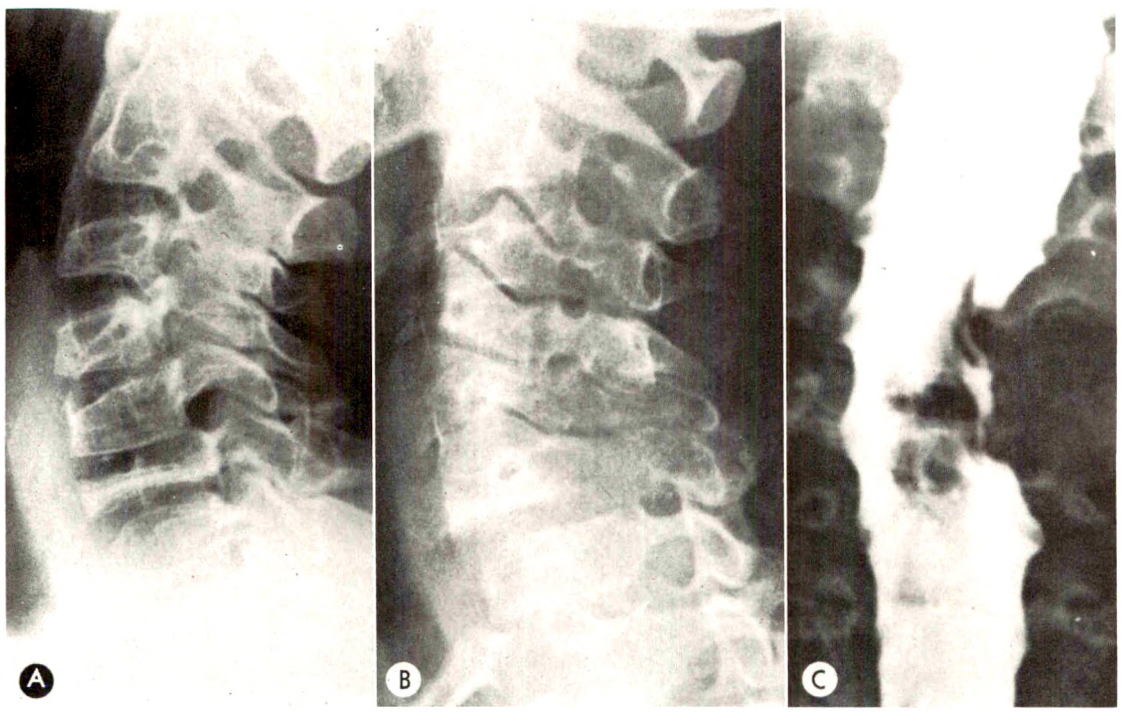


FIG. 4. (*A*) Lateral view of the cervical spine showing collapse of C6. (*B*) An oblique view showing an expansile lesion of the posterior spinal arch. (*C*) Cervical myelogram of the same patient illustrates a lobulated extradural defect which displaces the cord to the right.

circumscribed borders. In most instances, a faint sclerotic rim of bone was seen, suggesting the benignancy of the tumor. Only 4 cysts were eccentric in position. One cyst was adjacent to and extended across 2 epiphyseal plates. Epiphyseal extension is rather unusual. No para-osteal lesions were seen.[12] The fine honeycomb or soap-bubble appearance[5,12] was noted clearly in only 3 instances. Figure 2, *A* and *B*, illustrates a typical case, while Figure 3, *A* and *B*, shows a cyst with extension across the epiphyseal plate.

Spine. The posterior elements are the usual location of the aneurysmal bone cyst.[5,12] Our 8 patients were no exception. One cyst also extended anteriorly with involvement of the vertebral body (Fig. 4, *A* and *B*). Involvement of multiple vertebrae was noted in 7 of 15 cases in another recent series.[14] In our patients the cysts were most frequently expansile with a thin rim, although the expansion was more difficult to discern in the dorsal spine where loss of a pedicle was the most obvious finding. Three of our cases showed a complete extradural defect with displacement of the sac to the opposite side. The cervical lesion was by far the most impressive, with a lobulated irregular defect that displaced the cord to the opposite side (Fig. 4*C*). In the instances with complete extradural block, there was actual dural invasion by the tumor.[6] In 2 cysts of the lumbar spine, the ureter showed moderate lateral displacement without compression.

Flat Bones. The 2 patients with flat bone involvement had pubic lesions. One presented with a 6 cm. mass palpated on rectal examination. Roentgenograms showed a large lytic lesion which extended into the acetabulum, but a thin sclerotic rim was visible on the plain roentgenograms and laminagram (Fig. 5*A*). The bladder was displaced to the opposite side (Fig. 5*B*). Another case has been reported in which the pelvic tumor presented as a low abdominal mass.[8]

Small Tubular Bones. These 5 cases were noted in a phalanx, 2 metacarpals and 2 os calci, all rather uncommon sites, particularly the latter.[1,4] In these small tubular bones, the lesions displayed the typical location and symmetric and central expansion. Roentgenograms of these cysts showed the coarse trabeculation with honeycomb pattern more clearly than did those of other sites. The os calcis lesions were both strikingly similar (Fig. 6).

Facial Bones. Although involvement of the calvarium, mandible[4] and maxilla[2,16] have been reported, our 3 year old girl is, to our knowledge, the first case of an aneurysmal bone cyst in the zygoma. This cyst was incompletely excised, and re-

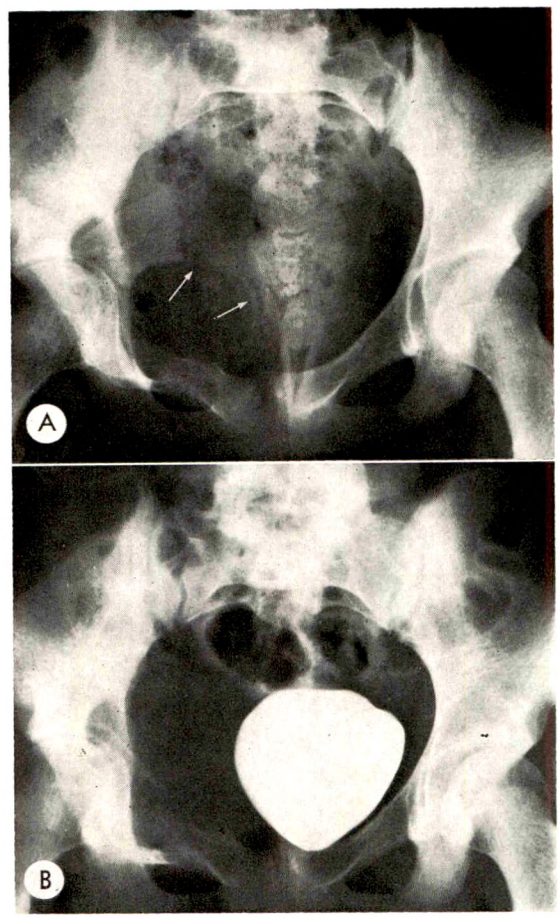


Fig. 5. (*A*) Anteroposterior roentgenogram of the pelvis showing a lytic lesion of the right superior pubis which extends into the acetabulum. Arrows mark the thin rim which was further delineated on laminagraphy. (*B*) Cystogram of the same patient showing displacement of the bladder to the left.

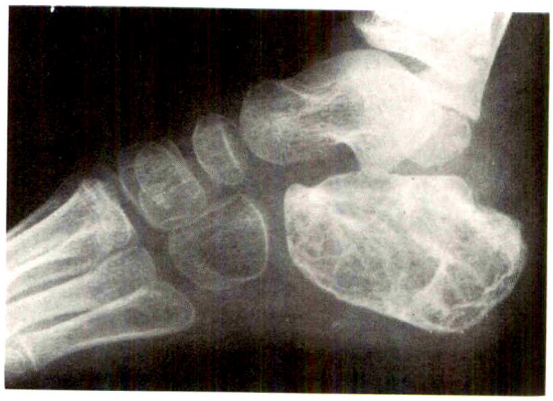


FIG. 6. Lateral view of the left os calcis with a uniform cystic expansion and a diffuse "basket-weave" pattern.

curred, destroying the entire right zygoma as well as the floor of the middle fossa. A right carotid arteriogram showed elevation of the middle cerebral artery, indicating a subtemporal mass (Fig. 7).

DISCUSSION

Although the etiology of aneurysmal bone cysts is yet to be determined, several theories have been proposed. Lichtenstein[11] has favored a "local circulatory disturbance" causing an increased venous pressure and engorged vascular bed. This would be either a venous thrombosis or arteriovenous aneurysm. Jaffe and Lichtenstein[9] preferred to consider hemorrhage engrafted on a pre-existing lesion, and cited 2 cases related to nonossifying fibroma and fibrous dysplasia. Biesecker *et al.*[1] combined the 2 theories. In their series of 66 cases, 21 showed another primary bone lesion in or adjacent to the cyst, the commonest being nonossifying fibroma, chondroblastoma and giant cell tumor. In addition, they found 3 of 6 aneurysmal bone cysts to have pressures verging on the arteriolar level, which would verify the reports of copious bleeding noted on surgery. They suggest that the pre-existing tumor causes an arteriovenous fistula, the outgrowth of which is the aneurysmal bone cyst. Other than 1 humeral lesion, which could have been a simple bone cyst, we have no evidence for a pre-existing lesion antedating any of our aneurysmal bone cysts. Slowick and associates[13] believe that there is a disparity between arterial inflow and venous outflow in these tumors.

Edling[7] preferred to group the aneurysmal bone cyst, simple bone cyst, giant cell tumor, and fibrous dysplasia under the category of dysfibroplasia. He believes that they all are related to a blocking of normal ossification accompanied by laying down of fibrous tissue, and that their occurrence at different areas gives them their characteristic appearance. He prefers the terminology subperiosteal dysfibroplasia for aneurysmal bone cysts, epiphyseal dysfibroplasia for giant cell tumors, metaphyseal for simple bone cysts and diaphyseal for fibrous dysplasia. It is difficult to see how a giant cell tumor can be grouped with the aneurysmal bone cyst.[3]

Tumors of the bone, such as vascular



FIG. 7. Anteroposterior view of a right carotid arteriogram showing elevation of the middle cerebral artery, which in this patient indicated a subtemporal mass.

giant cell tumor, hemangioma with trauma and healing, and telangiectatic osteogenic sarcomas enter the pathologic differential diagnosis of aneurysmal bone cysts. The roentgenogram, clinical picture and histology should all be considered together carefully for an adequate diagnosis.

The lining cells of the aneurysmal spaces appear to be endothelial. Endothelial cells are considered the precursors of osteoblasts and osteocytes by Trueta.[15] He has demonstrated by light microscopy and ultrastructure studies the endothelial derivation of osteoblasts and osteocytes. The giant cells in aneurysmal bone cysts are similar to those of giant cell tumors of bone and those in hyperparathyroidism. These are considered to be endothelial in origin by Johnson,[10] who quotes numerous other authors who share this view. If, in fact, endothelial cells are the source of osteoblasts, osteocytes, giant cells and the vascular spaces, it is possible to understand how dysplasia is an etiologic factor in the evolution of this lesion.

While the plain roentgen examination will always be the prime diagnostic method, tomography is frequently of value in eliciting the residual thin cortical rim and suggesting the benignancy of this lesion. Myelography is of great value in evaluating spinal involvement and should be used more routinely to help detect the potentially more serious lesion. Since these cysts can be rapidly progressive, the presence of an extradural defect should encourage prompt treatment.

Using surgical treatment alone, only 4 cases of short term recurrence were noted. Only 1 patient received radiation therapy in conjunction with surgery. As sarcomas have been reported arising in irradiated sites, radiation therapy should be used judiciously in treating these benign lesions.[14] While some successful responses have been reported,[12] we believe that irradiation should be reserved for surgical failures, or for children who might be inoperable due to location of the cyst.

SUMMARY

Twenty-six cases of aneurysmal bone cyst in children are reported with clinical, pathologic and roentgenographic findings.

Positive myelographic findings were present in 5 cases.

A zygomatic tumor and 2 tumors of the os calcis are included.

The necessity for correlation of the pathologic findings with the clinical and roentgen findings to achieve accurate diagnosis is emphasized.

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REFERENCES

1. Biesecker, J. L., Marcove, R. C., Huvos, A. G., and Mike, V. Aneurysmal bone cysts: clinical study of sixty-six cases. *Cancer*, 1970, *26*, 615–625.
2. Byrd, D. L., Allen, J. W., Kindrick, R. D., and DeWitt, J. D. Aneurysmal bone cyst of maxilla. *J. Oral Surg.*, 1969, *27*, 296–300.
3. Clough, J. R., and Price, C. H. Aneurysmal bone cysts: review of twelve cases. *J. Bone & Joint Surg.*, 1968, *50-B*, 116–127.
4. Dabska, M., and Buraczewski, J. Aneurysmal bone cyst: pathology, clinical course and radiologic appearances. *Cancer*, 1969, *23*, 371–389.
5. Dahlin, D. C., Besse, B. E., Jr., Pugh, D. G., and Ghormley, R. K. Aneurysmal bone cysts. *Radiology*, 1955, *64*, 56–65.
6. Donaldson, W. F., Jr. Aneurysmal bone cyst. *J. Bone & Joint Surg.*, 1962, *44-A*, 25–39.
7. Edling, N. P. Is aneurysmal bone cyst true pathologic entity? *Cancer*, 1965, *18*, 1127–1130.
8. Garnjobst, W., and Hopkins, R. Aneurysmal bone cyst of pubis: report of case presenting as abdominal mass. *J. Bone & Joint Surg.*, 1967, *49-A*, 971–975.
9. Jaffee, H. L., and Lichtenstein, L. Solitary unicameral bone cyst with emphasis on roentgen picture, pathologic appearance and pathogenesis. *Arch. Surg.*, 1942, *44*, 1004–1025.

10. JOHNSON, W. W. Giant cells of benign giant cell tumors of bone. *Arch. Path.*, 1930, *10*, 197–205.
11. LICHTENSTEIN, L. Aneurysmal bone cyst: further observations. *Cancer*, 1953, *6*, 1228–1237.
12. SHERMAN, R. S., and SOONG, K. Y. Aneurysmal bone cyst: its roentgen diagnosis. *Radiology*, 1957, *68*, 54–64.
13. SLOWICK, F. A., JR., CAMPBELL, C. J., and KETTELKAMP, D. B. Aneurysmal bone cyst: analysis of thirteen cases. *J. Bone & Joint Surg.*, 1968, *50-A*, 1142–1151.
14. TILMAN, B. P., DAHLIN, D. C., LIPSCOMB, P. R., and STEWART, J. R. Aneurysmal bone cyst: analysis of ninety-five cases. *Proc. Staff Meet. Mayo Clin.*, 1968, *43*, 478–495.
15. TRUETA, J. Osteogenesis: studies since Mac Ewen's time. *Scottish M. J.*, 1966, *11*, 33–45.
16. WANG, S. Y. Aneurysmal bone cyst in maxilla. *Plast. & Reconstruct. Surg.*, 1960, *25*, 62–72.

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THE VALUE OF ROENTGENOGRAPHY IN DIFFERENTIATING APLASTIC ANEMIA FROM LEUKEMIA MASQUERADING AS APLASTIC ANEMIA*

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IN a review of the cases of aplastic anemia seen at the St. Louis Children's Hospital, 3 patients originally diagnosed as having aplastic anemia subsequently were discovered to have leukemia. The phenomenon of acute lymphoblastic leukemia masquerading as aplastic anemia is well known. Our patients had skeletal lesions compatible with leukemia when aplastic anemia was diagnosed. Fifteen patients with true aplastic anemia had normal skeletal series.

The importance of these roentgenographic findings is the subject of the present paper.

REPORT OF CASES

CASE I. G.E. was a 5 year old boy referred to St. Louis Children's Hospital for anemia. He complained of generalized bone pain, anorexia and fever. When examined he was pale and had hepatosplenomegaly. The hemoglobin was 7.5 gm. per cent. The white blood cell count was 1,250: no platelets were seen. While in the hospital he developed left flank pain and hematuria. An intravenous urogram showed bilaterally enlarged kidneys with some calyceal distortion (Fig. 1). The most probable diagnosis seemed to be either leukemia or multiple intrarenal abscesses. Skeletal roentgenograms showed metaphyseal demineralization (Fig. 2). A bone marrow examination revealed a hypocellular marrow with the myeloid and erythroid series markedly decreased. No megakaryocytes or blast cells were seen. A diagnosis of aplastic anemia was made. The patient was transfused and placed on cortisone for 40 days. He responded well, and the hemoglobin, white blood cell count and bone marrow examinations returned to normal. Six weeks after the steroids were stopped, the patient became weak and pale. A bone marrow examination showed acute lymphoblastic leukemia. The patient died 2 months later with leukemia and sepsis.

CASE II. K.M. was a 6 year old girl who, several months prior to hospital admission in 1955, started to have pain in her arms and legs, fatigue and fever. She was diagnosed as having rheumatic fever. She responded promptly to aspirin and cortisone. The steroids were stopped. Two months later the leg pain recurred with fever. She was pale, appeared chronically ill and had a hemoglobin concentration of 7 gm. per cent. The white blood cell count was 926 with 266,000 platelets. The reticulocyte count was 31 per cent. Bone marrow examination showed marked hypoplasia of all elements, especially the granulocytic series. Bone marrow examinations from 3 sites were all the same. A diagnosis of aplastic anemia was made. Roentgenograms of the hands, wrists and knees were obtained because of the initial clinical impression of rheumatoid arthritis. They showed radiolucencies of the metaphyseal region sus-

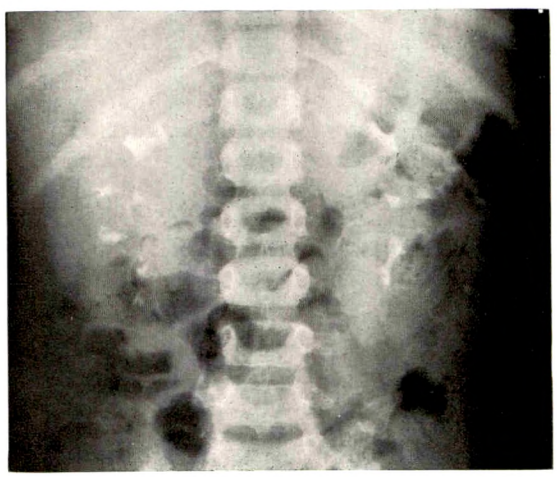


FIG. 1. Case I. Intravenous urogram demonstrating enlarged and infiltrated kidneys.

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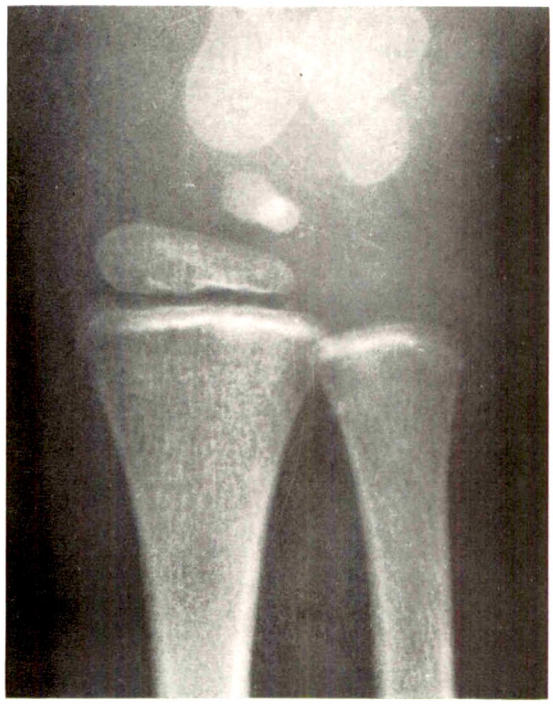


FIG. 2. Case I. Roentgenogram of the wrist. There is metaphyseal demineralization.

picious of leukemia (Fig. 3). The patient was again placed on steroids and responded well clinically and hematologically. Nine weeks after therapy was discontinued, the white blood cell count rose and a repeat bone marrow examination showed complete replacement by lymphoblastic forms. The patient died about 1 year after the correct diagnosis of leukemia was established.

CASE III. A 27 month old girl entered the hospital with a 3 week history of unresponsive pneumonia. The physical examination was normal except for pallor. The hemoglobin was 7.1 gm. per cent and the white blood cell count was 1,280. Roentgenograms showed metaphyseal rarefaction. The bone marrow examination revealed complete absence of the myeloid element with adequate red blood cell precursors and megakaryocytes. *Escherichia coli* was cultured from the blood. The diagnostic impression was aplastic anemia secondary to chloromycetin therapy or to sepsis. The patient was treated with blood transfusions, antibiotics and testosterone. Within 1 month, the hemoglobin, white blood cell count and bone marrow examinations returned to normal. She did well for an additional 3 months, when she developed fever and cough. She was anemic and the bone marrow was packed with blast cells. After the diagnosis of acute lymphoblastic leukemia was made, the patient was placed on 6 mercaptopurine and prednisone. She responded initially, but died in relapse 7 months later.

DISCUSSION

In general, the diagnosis of acute lymphoblastic leukemia is not a difficult one. The clinical picture and the bone marrow examination are characteristic.[16] However, on occasion, leukemia presents with a depressed bone marrow that leads to a diagnosis of aplastic anemia or pancytopenia. Since it is known that idiopathic acquired aplastic anemia may progress into acute leukemia, a prognostic uncertainty is omnipresent when it is diagnosed.[2,6] Preparation and examination of the buffy coat may differentiate leukemia from aplastic anemia.[12] In 1970 Melhorn *et al.*[8] suggested that the dramatic response to steroids with apparent hematologic and clinical recovery was useful in distinguishing true aplastic anemia from leukemia masquerading as aplastic anemia. In the cases presented here the roentgen findings of leukemia suggest that the correct diagnosis was not true aplastic anemia.

The incidence of skeletal lesions in childhood acute leukemia varies between 47–100

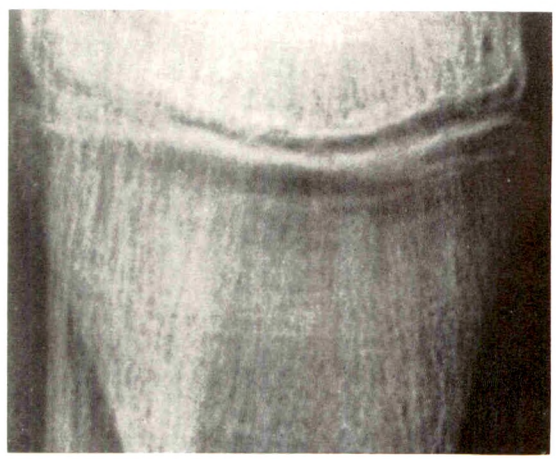


FIG. 3. Case II. Lateral roentgenogram of the knee. The tibia shows a metaphyseal radiolucent band.

per cent with an average of approximately 70–75 per cent.[10,11] Such lesions are most often found in terminal patients.

The skeletal changes of leukemia are usually classified into several categories (one or more manifestations may be present simultaneously): (1) generalized rarefaction; (2) a transverse metaphyseal radiolucent line adjacent to the zone of provisional calcification at the ends of the long bones and beneath the cortex in the flat bones; (3) osteolytic lesions; (4) subperiosteal new bone formation with or without destructive cortical lesions; and (5) osteosclerosis.

A generalized rarefaction is common, and was seen in 100 per cent of the children in a series reported by Thomas *et al.*[13] It is difficult to separate the effects on the bone by leukemic infiltrations of the bone marrow, steroid administration, chemotherapeutic agents and inactivity of the patient.

The juxta-epiphyseal radiolucent bands are usually 1–6 mm. in width, although they may reach 15 mm.[13,15] They are often bilateral and usually extend from one cortex to the other. The band results chiefly from a disturbance of endochondral bone formation, resulting in abnormally small trabeculae during a period of longitudinal bone growth.[13] It may be seen in a variety of other conditions.[1] The changes are found in rapid growth areas—wrists, knees and ankles, although any part of the skeleton may be involved.

The osteolytic lesions may be localized or diffuse. Usually they are less than 5 mm. in diameter[13] and attack the spongiosa of the long bones first, although they may involve any bone and extend throughout it.

Subperiosteal new bone formation can result from leukemic masses interspersed between the cortex and the periosteum and can involve either a short or long segment of bone.[13] Underlying destruction of cortical bone may not be appreciated on the roentgenograms. Occasionally, multiple layering or even radiating spicules may be present.[4] Osteosclerosis is rare.[9] Bone infarcts have been described.[4]

The skeletal lesions of leukemia may heal with remission. Progression of these changes has been reported in cases with hematologic remission.[1,5]

Leukemic infiltration may cause the kidneys to enlarge with stretching of the calyceal structures.[14] The latter may also be grossly distorted. The renal involvement is usually bilateral, but can be unilateral. The excretion of the contrast material is usually good. Enlargement of both kidneys with calyceal distortion was found in 1 of our patients. Infiltration in one or multiple other organ systems (thymus,[3] gastrointestinal, lymph nodes, spleen, liver, pericardium, etc.) can occur. Increased intracranial pressure can be seen from central nervous system involvement.

As several authors note,[9,15] the roentgenographic findings are often important in establishing the correct diagnosis from such similarly manifested conditions as rheumatic fever and rheumatoid arthritis. Although the individual skeletal lesions of leukemia are not pathognomonic, they are suggestive and can indicate an optimal site for biopsy.

The distinction between aplastic anemia and the aplastic phase of leukemia is important in prognosis and therapy. Aplastic anemia is currently being treated with prolonged administration of androgens alone or combined with steroids. Short term steroids are generally used in leukemia therapy to induce remission. They are not recommended for maintenance therapy.

SUMMARY

Three cases of leukemia originally diagnosed by peripheral blood and bone marrow studies as aplastic anemia are reported.

They had roentgenographic skeletal lesions, nonspecific of, but compatible with leukemia when initially seen. One patient also had enlarged kidneys with distorted calyceal anatomy from leukemia infiltrates. Fifteen true aplastic anemia patients had normal skeletal series.

A roentgen survey of the skeleton and kidneys may be of value in differentiating

true aplastic anemia from the aplastic phase of leukemia.

The optimal site for skeletal biopsy can also be determined.

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REFERENCES

1. BRUNNER, S., GUDJBERG, C. E., and INVERSEN, T. Skeletal lesions in leukemia in children. *Acta radiol.*, 1958, *49*, 419–424.
2. CLEMENT, D. H. Aplastic anemia. *Pediat. Clin. North America*, 1962, *9*, 703–725.
3. DAVIS, L. A., and MCCREADIE, S. R. Enlarged thymus gland in leukemia in childhood. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1962, *88*, 924–927.
4. GRIFFITHS, D. L. Bone changes and blood disease. In: Modern Trends in Blood Diseases. Edited by J. F. Wilkinson. Butterworth & Co., Lt., London, 1955, pp. 1–21.
5. KARPINSKI, F. E., JR., and MARTIN, J. F. Skeletal lesions of leukemic children treated with aminopterin. *J. Pediat.*, 1950, *37*, 208–223.
6. LEWIS, S. M. Course and prognosis in aplastic anaemia. *Brit. M. J.*, 1965, *1*, 1027–1031.
7. LIGHTWOOD, R., BARRIE, H., and BUTLER, N. Observations on 100 cases of leukemia in childhood. *Brit. M. J.*, 1960, *1*, 747–752.
8. MELHORN, D. K., GROSS, S., and NEWMAN, A. J. Acute childhood leukemia presenting as aplastic anemia: response to corticosteroids. *J. Pediat.*, 1970, *77*, 647–652.
9. MOSELEY, J. E. Patterns of bone change in leukemias and myelosclerosis. *J. Mt. Sinai Hosp. New York*, 1961, *18*, 1–31.
10. SILVERMAN, F. N. Skeletal lesions in leukemia: clinical and roentgenographic observations in 103 infants and children, with review of literature. AM. J. ROENTGENOL. & RAD. THERAPY, 1948, *59*, 819–844.
11. SIMMONS, C. R., HARLE, T. S., and SINGLETON, E. B. Osseous manifestations of leukemia in children. *Radiol. Clin. North America*, 1968, *6*, 115–130.
12. STURGEON, P. Idiopathic aplastic anemia in children: its early differentiation from aleukemic leukemia by bone marrow aspiration. *Pediatrics*, 1951, *8*, 216.
13. THOMAS, L. B., FORKNER, C. E., JR., FREI, E., III., BESSE, B. E., JR., and STABENAU, J. R. Skeletal lesions of acute leukemia. *Cancer*, 1961, *14*, 608–621.
14. TUCKER, A. S., NEWMAN, A. J., and PERSKY, L. Kidneys in childhood leukemia. *Radiology*, 1962, *78*, 407–412.
15. WILLSON, J. K. Bone lesions of childhood leukemia: survey of 410 cases. *Radiology*, 1959, *72*, 672–681.
16. WINTROBE, M. M. Clinical Hematology. Fourth edition. Lea & Febiger, Philadelphia, 1956, pp. 906–1007.

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SYNOVIAL CYSTS IN JUVENILE RHEUMATOID ARTHRITIS*

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ALTHOUGH development of synovial cysts in adult patients with rheumatoid arthritis is a well recognized phenomenon,[6–8,10–12] synovial cyst development in juvenile rheumatoid arthritis has gained little attention. This may be explained on the basis of the relative rarity of these cysts in children *versus* relative frequency in the middle-aged adult. However, since synovial cysts do occur in juvenile rheumatoid arthritis and can be a problem in diagnosis, documentation of cases is important.

Two children with rheumatoid arthritis admitted to Strong Memorial Hospital with mid-limb cystic masses are reported. In 1 child the cystic mass extended from a swollen knee into the calf, and in the other from a swollen shoulder into the mid-upper arm. Radiodense contrast material was used to outline the cysts and to prove their synovial origin. In 1 case this was accomplished by arthrography and in the other by direct cyst puncture (synovial cysto-arthrography).

REPORT OF CASES

CASE I. J.G. (SMH No. 72-13-74). This $10\frac{1}{2}$ year old boy slipped and fell on ice and sustained an injury to his right lower leg. A painful soft tissue mass developed over the popliteal fossa of the right knee and extended into the calf.

Past history indicated 2 previous hospital admissions in the 2 year period prior to this accident. The diagnosis of juvenile rheumatoid arthritis had been established during that period when he presented with migratory joint pain associated with the soft tissue swelling and tenderness, limitation of motion in various joints, spikes of uncontrolled fever, and characteristic rheumatoid maculopapular rash over the body.

Major laboratory findings during the 2 admissions were leukocytosis and elevated erythrocyte sedimentation rate (as high as 50 mm./hr. [Westergren]). Latex fixation test and lupus erythematosus cell preparation were negative. Electrocardiograms were normal on both occasions.

The patient had been treated over the previous 2 years alternatively with aspirin, prednisone and indocin. Five months prior to the accident, a trial of gold therapy was instituted.

An arthritic survey obtained 6 months prior to the accident showed periarticular demineralization, particularly about the metatarsophalangeal joints, the proximal interphalangeal joints and the hips. In addition, there was a juxta-articular erosion of the right ulnar metaphysis and synovitis of both ankle joints without bone erosion.

The soft tissue swelling in the right popliteal area was thought to represent a ruptured synovial cyst. To check this possibility an arthrogram of the right knee was obtained. Seven cubic centimeters of blood-tinged fluid was aspirated prior to instillation of air and conray 60. The double contrast examination showed a cystic extension approximately 16 cm. long, 5 cm. wide and 2 cm. deep from the posterior medial aspect of the right knee involving the semimembranosus bursa and extending into the calf (Fig. 1, *A* and *B*). The appearance was that of a narrow neck connecting the described cyst with the gastrocnemius bursa via the semimembranosus bursa (Fig. 2, *A* and *B*). Numerous small filling defects within the cysts were noted, probably representing fibrino-lipoid bodies. No point of rupture was demonstrated. During the next few weeks the tenderness disappeared, and on physical examination the soft tissue mass had become smaller.

CASE II. S.G. (SMH No. 66-98-29). This 6 year old boy entered Strong Memorial Hospital

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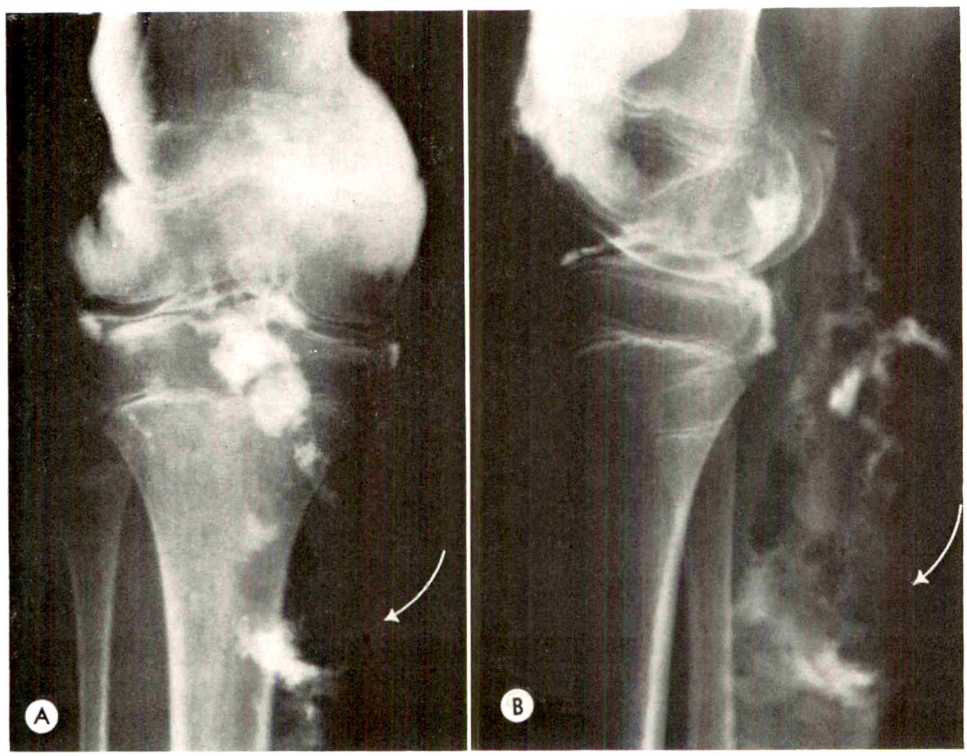


FIG. 1. Case 1. (*A*) Anteroposterior and (*B*) lateral roentgenograms. Conray 60 and air contrast study shows a cystic extension into the calf (arrows) from the posterior medial aspect of the semimembranosus bursa of the right knee.

for the second time (previous admission 2 months before had established the diagnosis of juvenile rheumatoid arthritis) with a history of daily fever spikes for the preceding 2 months up to 104° F., and attacks of joint pain and swelling most pronounced in the metacarpophalangeal and proximal interphalangeal joints, elbows, knees and ankles. Posterior neck pain and stiffness were initially present. Fatigue and anorexia were prominent symptoms, and anterior chest rash was noted on the first admission. Soft tissue swelling of the medial anterior aspect of the left upper arm was noted 1 week prior to the admission. He had been receiving aspirin (25 mg. q.i.d.) for the previous 2 months until 2 weeks prior to admission when prednisone (30 mg.) was started.

On physical examination a non-tender, fluctuating, soft tissue mass anterior to the left humerus was noted. Non-tender swelling was also present over radial heads bilaterally, and limitation of flexion was present in both wrist joints. No lymph nodes were palpable and there was no abdominal organomegaly. There was no skin rash. Heart and lungs were normal.

Laboratory results were as follows: white blood cell count 51,900; segmented neutrophils 96 per cent; lymphocytes 3 per cent; monocytes 1 per cent; erythrocyte sedimentation rate 67 mm./hr. (Westergren); gamma globulin 1,740 mg. per cent; gamma A 37 mg. per cent; gamma M 38 mg. per cent. Latex fixation test, anti-nuclear factor and Coombs tests were negative. Electrocardiogram was normal.

Roentgenologic findings. An arthritic survey of the hands and pelvis showed mild soft tissue swelling around both wrists. A soft tissue mass was noted along the anteromedial aspect of the left humeral shaft. There was no periosteal reaction or other bone abnormality.

Because fluid had been previously aspirated from the mass, a radiopaque contrast examination was requested. Cystography was performed following aseptic preparation by placement of a No. 18 gauge needle into the cystic mass along the medial aspect of the upper mid-arm under fluoroscopic control. Approximately 25 cc. of a yellow-white mucinous fluid was aspirated. This was followed by the instillation of about 20 cc. of a 25 per cent solution of hypaque. A

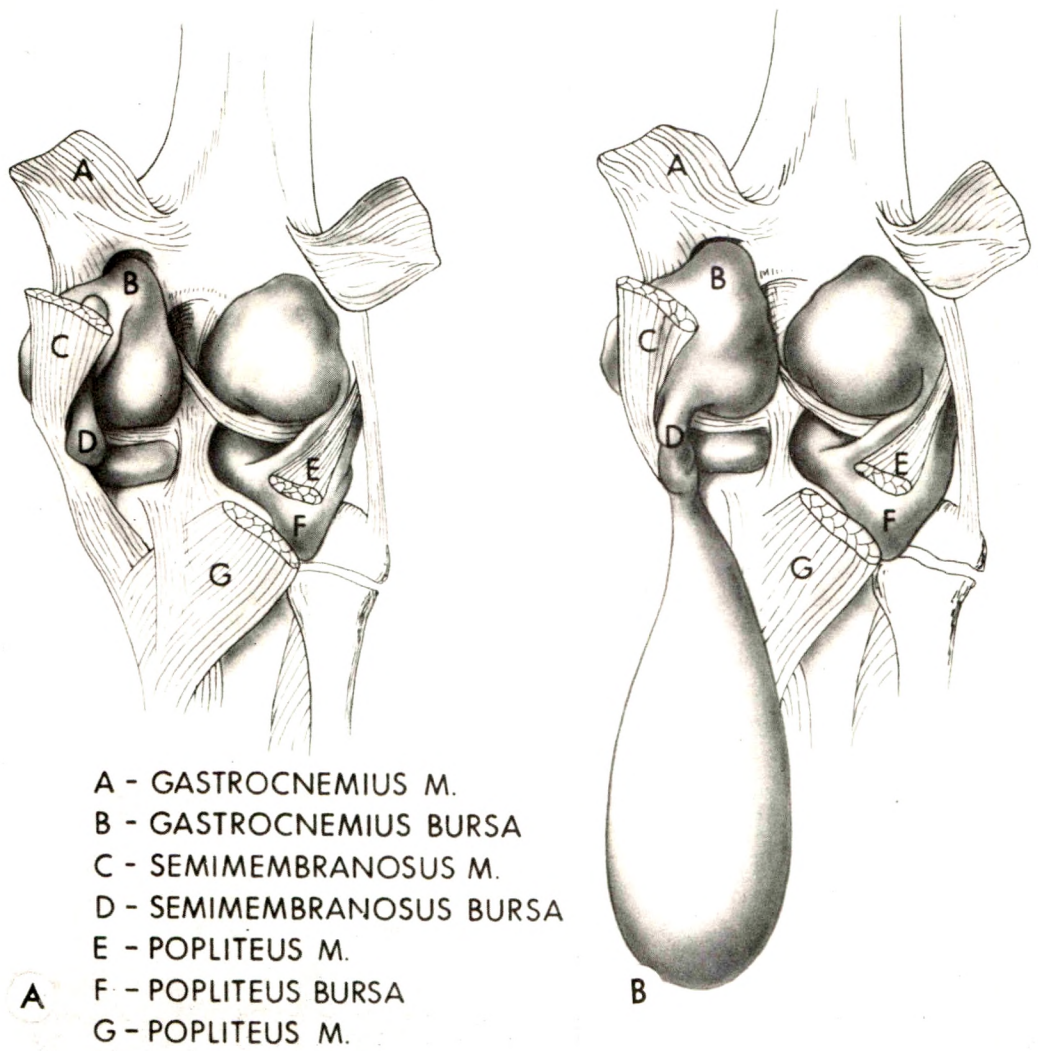


FIG. 2. Case 1. Diagrammatic presentation. (*A*) Posteroanterior view of normal knee bursae. (*B*) Posteroanterior view of bursae in rheumatoid arthritis with cystic extension via the narrow neck of the semimembranosus bursa (D).

pear-shaped, cystic cavity was demonstrated with a communication between the cyst and the bursa, surrounding the long head of the biceps and communicating with the shoulder joint cavity and the bursa under the subscapularis muscle (Fig. 3, *A* and *B*). This is diagrammatically demonstrated in Figure 4*B* which includes mild distention of the subacromial and subcoracoid bursae (not demonstrated in the synovial cystogram). Many white blood cells, the majority polymorphonuclear leukocytes, were observed by microscopy of the aspirated fluid. No crystals were seen under polarized light.

In a follow-up examination, the synovial cyst was unchanged in size in the course of the 2 years. A similar cyst, although somewhat smaller, developed in the opposite arm several months later.

DISCUSSION

It was clearly shown by Caughey and Bywaters[2] in 1963 that intra-articular pressure significantly increases during physical

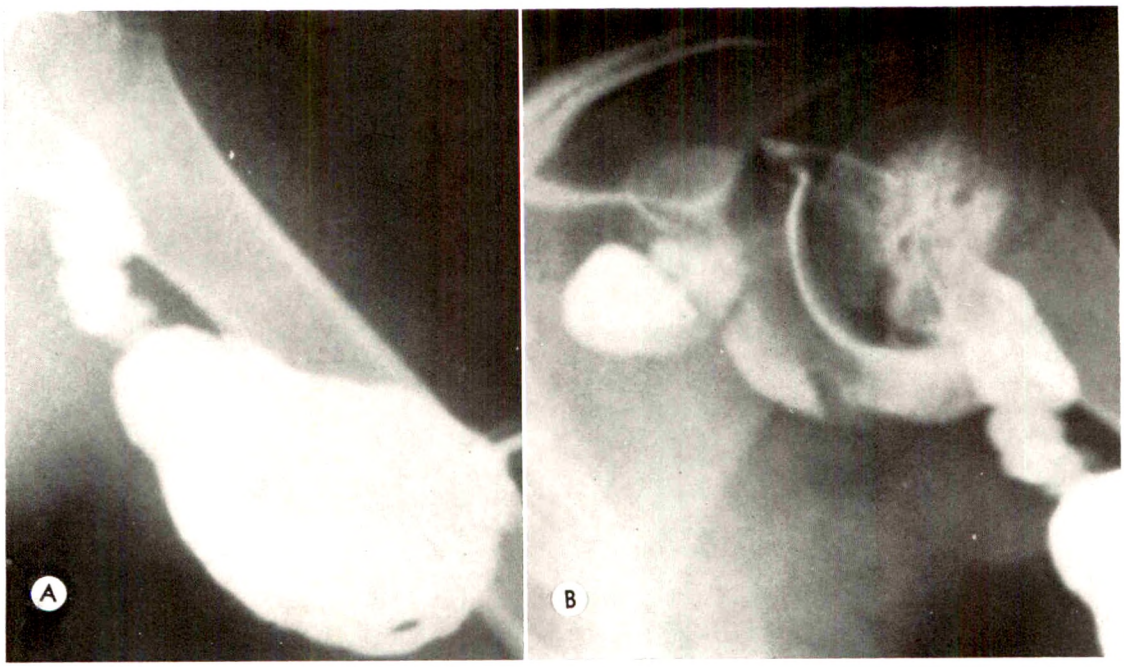


FIG. 3. Case II. (*A*) A pear-shaped cyst is present extending from the synovial sheath of the biceps and (*B*) communicating with shoulder joint cavity and the subscapularis bursa.

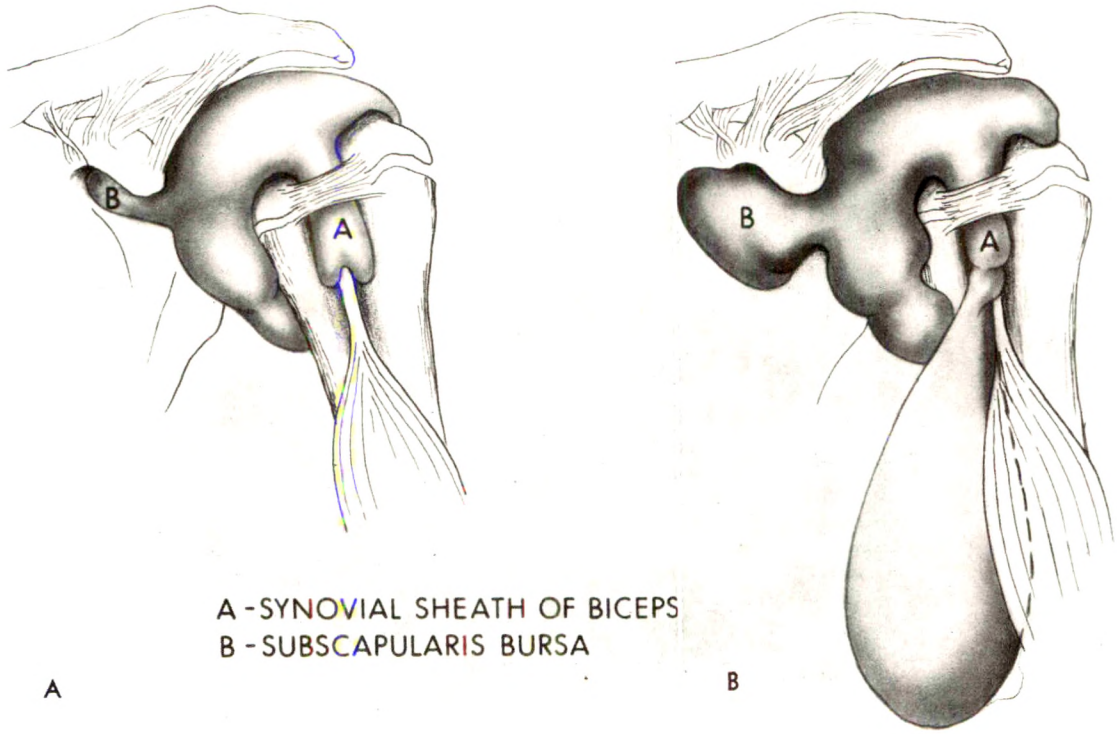


FIG. 4. Case II. Diagrammatic presentation. (*A*) Normal shoulder joint bursae. (*B*) Distended shoulder joint bursae and synovial cyst of upper arm. Note that the subcoracoid, subacromial and subdeltoid bursae are not included.

activity and in the presence of joint effusion. Interestingly, intra-articular readings, particularly in patients with rheumatoid arthritis associated with effusion, were statistically significantly higher as compared with controls presenting with effusion but without rheumatoid arthritis.[9] These findings offer a logical explanation as to how the cysts are formed, particularly in rheumatoid arthritis. Alteration in the connective tissues of a joint capsule may also play a significant role. Steroid therapy may well be the additional factor in altering the capsular connective tissue,[7] as a significant number of patients reported, including our 2 cases, have been on prednisone. The mechanical aspect of the formation of popliteal synovial cysts has been previously described.[3,6,7,10]

In adults the cysts most commonly arise secondarily as the result of effusion or fluid from a traumatic or inflammatory process within the joint. In children a primary process is rarely identified. Not a single case of primary joint disease was found in an evaluation of 23 children by Gristina and Wilson in 1964.[4] They postulated that the subclinical external trauma and compression to the popliteal space by the classroom seat is the major causative factor, and could explain the peak incidence of popliteal synovial cysts in children in the 6th and 7th years of the first decade. Rubbing of the normal bursa between the musculotendinous portions of the gastrocnemius and the semimembranosus muscles is thought to be another cause in this age group.[4] The higher incidence in boys (2:1) is related to their being more physically active than girls.

In our 2 cases the mechanism of cyst development is probably similar, if not identical, to that seen in the adult form of rheumatoid arthritis. Although in our first case trauma to the knee joint precipitated the clinical findings, the roentgenologic picture is identical to the popliteal cyst of rheumatoid arthritis. These cysts are usually large and painful, extend deep into the calf, have a narrow neck and develop rapidly. This is in contrast to the classical Baker's cyst, representing an enlargement of the gastrocnemius semimembranosus bursa, which is small, noninflammatory and specific in its anatomic location.[10]

The synovial cyst in Case II is rather unique considering its uncommon anatomic location at the shoulder and upper arm. The pear-shaped cyst has a neck arising anatomically from the synovial sheath of the long head of the biceps, which lies in the bicipital groove and is covered anteriorly by the transverse humeral ligament. The cyst communicates with the subscapularis bursa and the joint cavity, but not with the subdeltoid, subcoracoid or subacromial bursae. Only in a few cases have large synovial cysts been described in association with joints other than the knees.[5,12] Several adult cases of rheumatoid arthritis have been described with effusion and cystic distention of the subacromial and subdeltoid bursae. In Case II the subacromial and subdeltoid bursae are not opacified. This indicates that the musculotendinous cuff is intact.

Fibrino-lipoid bodies dispersed within the viscous fluid are usually found in the synovial cysts of rheumatoid arthritis. Pathologically, cysts may be classified as fibrinous, synovial, inflammatory and transitional.[1] Most cysts in patients with rheumatoid arthritis are of the synovial type. These cysts may occur in degenerative osteoarthritis, in traumatic arthritis, in villonodular synovitis, in chondromalacia of patella, in the phenomenon of loose intra-articular bodies, in club-feet and in poliomyelitis.[4]

Major complications are acute rupture and infection. After surgical excision, cysts may recur if the primary process is still active, and if the primary causative factors for the cyst formation are not eradicated.

Contrast material can be introduced either by the direct puncture of the cyst (synovial cystography), probably the more preferable way in children, or by direct instillation into the joint space (arthrography). For arthrography we use conray 60 and for cystography renografin 30 is the preferable water soluble contrast material,

because it is the least painful if injected outside of the cyst into the soft tissue. If communication between joint space and cyst is present or if rupture is present, the former should be demonstrated easily and the latter determined clearly by radiopaque contrast examination. The demonstration of the site of origin can be of surgical importance.

SUMMARY

Synovial cysts may occur in association with juvenile rheumatoid arthritis. Two such patients are reported.

The mechanism of formation of such cysts is probably similar, if not identical, to the synovial cyst formation in adult rheumatoid arthritis. Steroid therapy probably has some role in the development of these cysts.

The etiology of popliteal cysts in healthy children is briefly reviewed.

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REFERENCES

1. BURLESON, R. J., BICKEL, W. H., and DAHLIN, D. C. Popliteal cyst: clinico-pathologic survey. *J. Bone & Joint Surg.*, 1956, *37-A*, 1265–1274.
2. CAUGHEY, D. E., and BYWATERS, E. G. Joint fluid pressure in chronic knee effusions. *Ann. Rheumat. Dis.*, 1963, *22*, 106–109.
3. DOPPMAN, J. L. Baker's cyst and normal gastrocnemio-semimembranosus bursa. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1965, *94*, 646–652.
4. GRISTINA, A. G., and WILSON, P. D. Popliteal cysts in adults and children: review of 90 cases. *A.M.A. Arch. Surg.*, 1964, *88*, 357–363.
5. HALL, A. P., and HEALEY, L. A. Infected synovial cysts arising as complication of septic arthritis in patient with rheumatoid arthritis. *Arth. & Rheumat.*, 1968, *11*, 579–584.
6. HALL, A. P., and SCOTT, J. T. Synovial cysts and rupture of knee joint in rheumatoid arthritis. *Ann. Rheumat. Dis.*, 1966, *25*, 32–41.
7. HARVEY, J. P., JR., and CORCOS, J. Large cysts in lower leg originating in knee occurring in patients with rheumatoid arthritis. *Arth. & Rheumat.*, 1960, *3*, 218–228.
8. HOLLINGSWORTH, J. W. Local and Systemic Complications of Rheumatoid Arthritis. Chapter 7. W. B. Saunders Company, Philadelphia, 1968.
9. JAYSON, M. I., and DIXON, A. ST.J. Quadriceps contraction and intra-articular pressure. *Clin. Sc.*, 1969, *37*, 874.
10. PERRI, J. A., RODNAN, G. P., and MANKIN, H. J. Giant synovial cysts of calf in patients with rheumatoid arthritis. *J. Bone & Joint Surg.*, 1968, *50-A*, 709–719.
11. SCOTT, E. G., and DAVIS, J. S., IV. Giant synovial dissecting cyst: uncommon feature of rheumatoid arthritis. *Virginia M. Month.*, 1969, *96*, 449–452.
12. WATSON, J. D., and OCHSNER, S. F. Compression of bladder due to "rheumatoid" cysts of hip joint. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1967, *99*, 695–696.

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RENAL PAPILLARY NECROSIS IN THE NEWBORN*

A CASE REPORT WITH ROENTGENOLOGIC DOCUMENTATION OF LATE SEQUELAE

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RENAL papillary necrosis in the newborn was once considered a fatal disease. There was no roentgenologically documented case reported in the literature prior to 1969.

We report the case history and the roentgenologic findings in a 6 year old child who had severe asphyxia, renal papillary necrosis and bilateral adrenal hemorrhage in the newborn period.

REPORT OF A CASE

B. Baby Boy (M.C.H. #290373), was delivered as a compound breech presentation after 38 weeks of gestation. After birth, the baby was flaccid and cyanosed with no audible heart sounds. External cardiac massage and intubation were required for resuscitation.

On admission to The Montreal Children's Hospital, he was found to be cyanotic, with slow, shallow respirations and a weak pulse. There was marked central nervous system depression with absent Moro, grasp and suck reflexes.

Laboratory data shortly after admission indicated extreme acidosis which was treated with intravenous sodium bicarbonate. Intramuscular chloramphenicol was given for suspected pneumonia. The baby required oxygen treatment (20 to 60 per cent oxygen during the first week of life).

At 3 days of age, the acid-base disturbance had been corrected and within the first week, gavage and bottle feeding were started. There was no adequate record of the urine output of the infant during the first 3 days of life.

At 2 weeks of age, urinalysis revealed 16 to 20 red blood cells per high power field (HPF), 11 to 20 white blood cells per HPF, coarse granular casts, 15 mg. of albumin per ml., and a 4+ orthotoludine reaction. Urine culture grew 70,000 colonies per ml. of *E. coli grunthali*.

The mode of "urine collection" at the time was not specified in the infant's hospital chart, but a note has been made of severe phimosis which at least partially invalidates the urinary findings.

Blood urea nitrogen determinations were only done during the period of recovery (at 1 week of age, 39 mg. per cent; 3 weeks of age 32 mg. per cent).

During the acute phase of the disease, the patient had no roentgenologic investigation of the urinary tract. Renal abnormality had not been suspected.

Excretory urography was performed at 3 weeks of age, when the baby was well hydrated and was not in acute distress. The roentgenologic findings were compatible with renal papillary necrosis involving both kidneys (Fig. 1, *A* and *B*).

The patient was discharged at 1 month of age and followed thereafter in the out-patient service.

At 13 months of age, the baby was admitted for circumcision. There was a history of inflammation of the prepuce on at least 2 occasions. Slow development was questioned, but retardation was not diagnosed.

During the second admission, a chest roentgenogram was taken which revealed bilateral adrenal calcification (not illustrated).

On subsequent visits to the clinic, it gradually became obvious that the child was mentally retarded, hyperactive, becoming a behavior problem. At 6 years of age he was enuretic. Assessment by the psychology service resulted in the diagnosis of severe mental retardation and the child was placed in a special class. There was no documented urinary tract infection following the first admission.

Urographic examination was done at 6 years of age (Fig. 2, *A* and *B*) which revealed bilateral adrenal calcification, irregular contour of the kidneys with recognizable focal scarring. Most of the medullary cavities which were present at 3 weeks of age had disappeared, some per-

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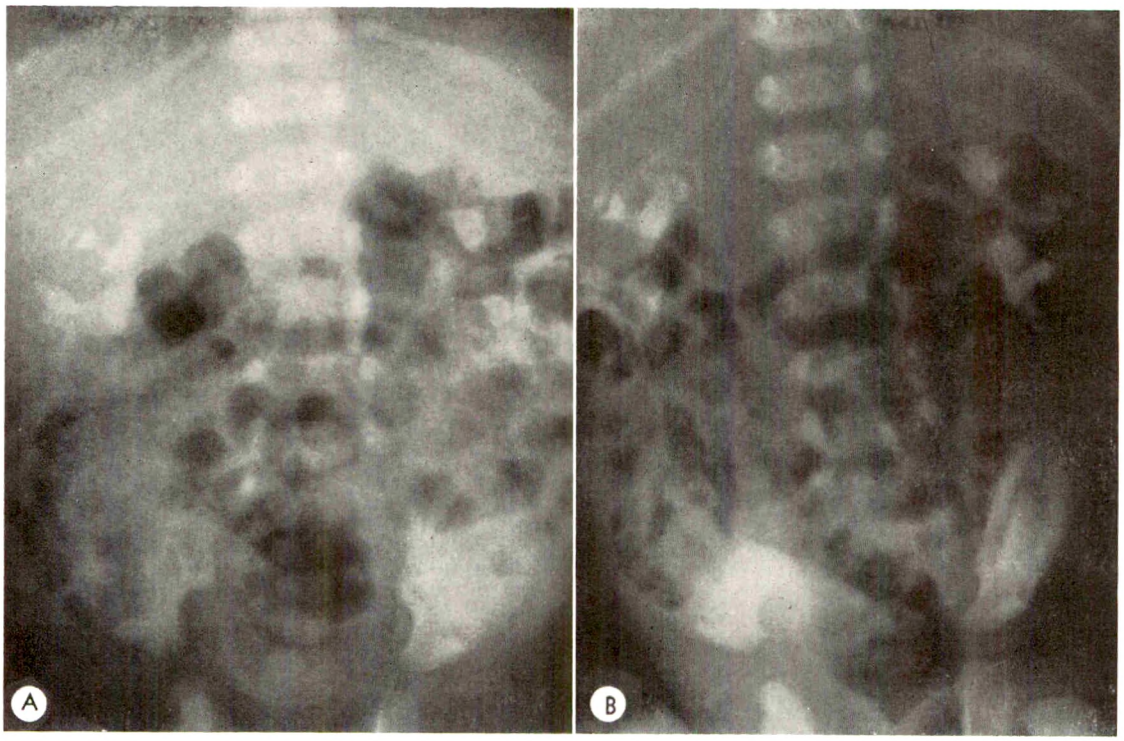


FIG. 1. (*A* and *B*) Urograms at 3 weeks of age. The kidneys are large, their contour is smooth but irregular cavities are filled in the renal papillae adjacent to every minor calyx visualized. There is dilatation and blunting of the minor calyces.

sisted but diminished in size. With ureteric compression and a second injection (Fig. 2*B*) excellent nephrogram and collecting system visualization were obtained. The major calyces are shown to be fusiform in shape, the minor calyces are narrow and have an amputated appearance. There is no normal papillary contour recognizable in either kidney.

DISCUSSION

The evolution of renal papillary necrosis, when it occurs in the newborn period, is not known. The urographic appearance of the kidneys in survivors at a later age is not described in the literature, as far as we know. Chrispin *et al.*[1] reported on the radiologic and histologic findings in 3 young infants, who had renal papillary necrosis associated with severe gastroenteritis. It has been postulated that late sequelae of renal papillary necrosis may be misinterpreted radiologically for pyelonephritic scarring or renal dysplasia.[1,3]

The evaluation of the renal parenchymal changes in these infants is impossible without urography, since kidney abnormality may not be suspected. In 18 cases postmortem diagnosis of medullary necrosis was made in the series of Davies *et al.*[2] The condition was not suspected in any of those before death. Only in 5 patients was there any clinical or laboratory evidence of renal damage. In the early phase, *i.e.*, during the period of acute renal shutdown, the urographic findings are nonspecific.[3,5] In a few weeks, the multiple irregular cavities which are pathognomonic of renal papillary necrosis may be demonstrated.[1,3–5] In the recent literature 31 cases of renal papillary necrosis are reported in children by Murvanidze and Kapanadze.[4] Their youngest patient (apparently documented radio-

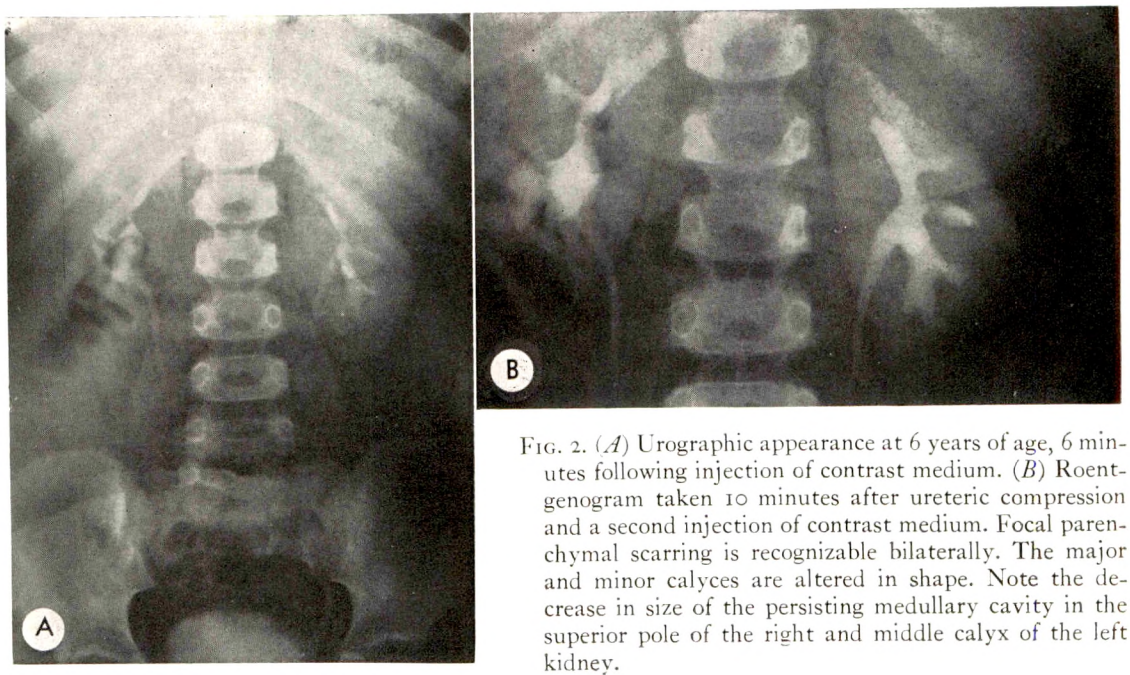


Fig. 2. (*A*) Urographic appearance at 6 years of age, 6 minutes following injection of contrast medium. (*B*) Roentgenogram taken 10 minutes after ureteric compression and a second injection of contrast medium. Focal parenchymal scarring is recognizable bilaterally. The major and minor calyces are altered in shape. Note the decrease in size of the persisting medullary cavity in the superior pole of the right and middle calyx of the left kidney.

logically but not illustrated in the publication) was 8 months old. In their series a 3 year old child who had papillary necrosis developed hypertension.

In our reported case, the roentgenographic findings at 3 weeks of age were typical of renal papillary necrosis, but at 6 years of age the roentgen appearance has significantly altered. Whether the urographic appearance of late sequelae of papillary necrosis is pathognomonic, remains to be answered.

SUMMARY

The changing urographic appearance of the kidney parenchyma in a survivor of renal papillary necrosis of newborn is presented.

The roentgen appearance of the late sequelae of the disease may cause diagnostic problems to the radiologist.

In a patient with known history of perinatal hypoxia and/or acute renal shutdown, renal papillary necrosis should be included in the differential diagnosis of chronic renal parenchymal changes.

It seems to us that documentation of roentgenologically recognizable sequelae should lead to more accurate diagnosis of renal parenchymal damage in childhood.

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REFERENCES

1. Chrispin, A. R., Hull, D., Lillie, J. G., and Ridson, R. A. Renal tubular necrosis and papillary necrosis after gastroenteritis in infants. *Brit. M. J.*, 1970, *1*, 410–412.
2. Davies, D. J., Kennedy, A., and Roberts, C. Renal medullary necrosis in infancy and childhood. *J. Path.*, 1969, *99*, 125–130.
3. Mauer, M., and Nogrady, M. B. Renal papillary and cortical necrosis in newborn infant: report of survivor with roentgenologic documentation. *J. Pediat.*, 1969, *74*, 750–754.
4. Murvanidze, D. D., and Kapanadze, G. I. Necrosis of renal papillae in children. *Urologiia I Nefrologiia* (Moskva), 1970, *35*, 7–12.
5. Perreault, G. Personal communication.

OVARIAN CYSTS IN THE NEWBORN*

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OVARIAN cysts are rarely seen in the newborn period (first month of life). The first case was reported in 1889[8] in a stillborn premature. While several additional cases were found in stillborns in the next decades, it was not until 1942[5] that the first successful removal of a neonatal ovarian cyst was recorded. To date, not counting stillborns, only 28 instances of ovarian cyst have been reported in the newborn period (Table I), almost all as single case reports. The 9 cases presented here, collected over the last 25 years, are by far the largest series of this rare entity.

CASE MATERIAL

An analysis of our cases is presented in Table II. Of the 9 cases, 6 were mentioned in a 1965 summary[13] of 117 neonatal abdominal masses seen at this hospital, and 2 of these 6 had in turn been included in an earlier summary.[20]

Only 2 of these 9 patients presented with signs or symptoms other than the mass itself. Case I had tachypnea, improving after aspiration of intraabdominal fluid, which was apparently free in the peritoneal cavity after rupture of the cyst. Case VII entered with vomiting, of uncertain cause. Most of the previously reported cases have also been asymptomatic except for abdominal distention and a palpable mass. All 9 of our cases were found on the first day of life. No child had any other congenital disease.

No predilection to involvement of one ovary over the other was found in our series or on review of the literature (Table I). Four of our cases were right-sided, 4 were on the left and 1 was bilateral.

All of the masses in this series were firm and smooth, as is true of previously reported cases. Three infants (Cases IV, V and IX) had cysts large enough to occupy both sides of the abdomen, and 1 of these cysts filled almost the entire abdomen. In 2 infants, the mass presented contralateral to its site of origin (Cases I and III). These cysts were both twisted, and during the torsion had apparently moved over to the other side of the abdomen. This phenomenon has been noted previously.[15] None of these cysts were palpably mobile. The side of presentation actually meant little, since usually an extendable anterior abdominal incision was used. However, the knowledge that the mass was unlikely to be of urinary origin prevented an unfortunate flank approach.

Intravenous urography was the chief and usually the only radiologic study in this series. The urinary tract was uniformly shown to be normal, apart from mild ureteral dilatation in one case. The total body opacification effect[23,26] was positive in 8 of these 9 masses and showed them to be avascular, presumably cystic, and probably benign (Fig. 1–3). In most cases the wall of the mass was shown by this effect to be thin and smooth; in 1 case, the corpus luteum cyst, the wall was demonstrably of variable thickness (Fig. 1), as was confirmed pathologically. The smallest cyst, Case VI, measuring 2.8 cm. in greatest diameter, failed to show the total body opacification effect.

Barium enema roentgenograms, obtained in 3 patients (Cases II, III and VII), all showed moderate upward displacement of the right colon; in all 3 cases the mass lay predominantly on the right by physical examination.

One of the cysts transilluminated by flashlight in a dark room. Only a single other case of transillumination of an ovar-

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TABLE I

PREVIOUSLY REPORTED OVARIAN CYSTS IN NEWBORNS

Pathology	Side	Size (cm.)	Location	Surgical Findings	Comment
Cystadenoma[9]	L	15×13	RLQ		
Follicular cyst[24]	L	12×10×5	LLQ		Polyhydramnios
Follicular cyst[18]	R	10×9×6	Right abdomen		
Follicular cyst[12]		19×12	Entire abdomen	Torsion of pedicle	Dystocia secondary to abdominal mass
Serous or simple cyst[32]	R	6×4×2	RLQ	Ileum adherent to mass	
Follicular cyst[27]	L	11	Left abdomen		
Simple cyst[36]	L	10×8×5			Transilluminated
Theca-lutein cyst[4]	R	10	Central		
Simple cyst[10]	R	8×6×4.5	Left abdomen		
Theca-lutein cyst[34]	R	11	Entire abdomen	Ruptured; 320 cc. ascites	
Corpus luteum cyst[35]	R	13	Right abdomen		
Follicular cyst[28]		9×9			
Follicular cyst[15]	R	6.5	Left abdomen	Torsion	Vomiting; no gas beyond ligament of Treitz
Follicular cyst[3]	L	7	LLQ	Twisted pedicle	
Corpus luteum cyst[3]	Bilateral	9 & 10	Entire abdomen		
Follicular cyst[7]	R	14	Low abdomen		
Follicular cyst[11]	R	13	Entire abdomen		
Follicular cyst[11]	L	9	—		
Follicular cyst[31]	R	—	—		
Follicular cyst[33]		—	—		
Paraovarian cyst[33]		—	—		
Hilar cell cyst[29]		8.5×5	Right abdomen	Rupture: 300 cc. ascites	
Simple cystoma[22]	R	14	—		
Cyst of unstated type[16]		—	—		
Follicular cyst[2]	L	12	—		
Theca-lutein cyst[21]	R	8	—	Ruptured	Polyhydramnios; small left cyst normal at herniorrhaphy 4 months later
Teratoma (benign)[6]	L	10	RLQ	Torsion	Patient died 3 days
Simple or serous cyst[17]	R	9×12×5	LLQ		

L = left; R = right; LLQ = left lower quadrant; RLQ = right lower quadrant.

ian cyst has been reported.[36] Transillumination of the head is widely used in hydrocephalus, subdural hematoma, porencephalic cyst, and hydranencephaly; it probably would be equally useful in the diagnosis of neonatal abdominal masses.

Almost all of the cases were operated on shortly after birth. The smallest cyst was not excised until 1 month after birth, it being so small that its actual existence was doubted. Four of the patients had transverse incisions, the rest being paramedian. In Case I, the cyst had ruptured; 3 other such cases have been reported.[21,29,34] In Cases I, III and VIII, torsion of the cyst had occurred, and the cyst of Case I was necrotic. Torsion with or without necrosis has been reported on 4 previous occasions.[3,6,12,15]

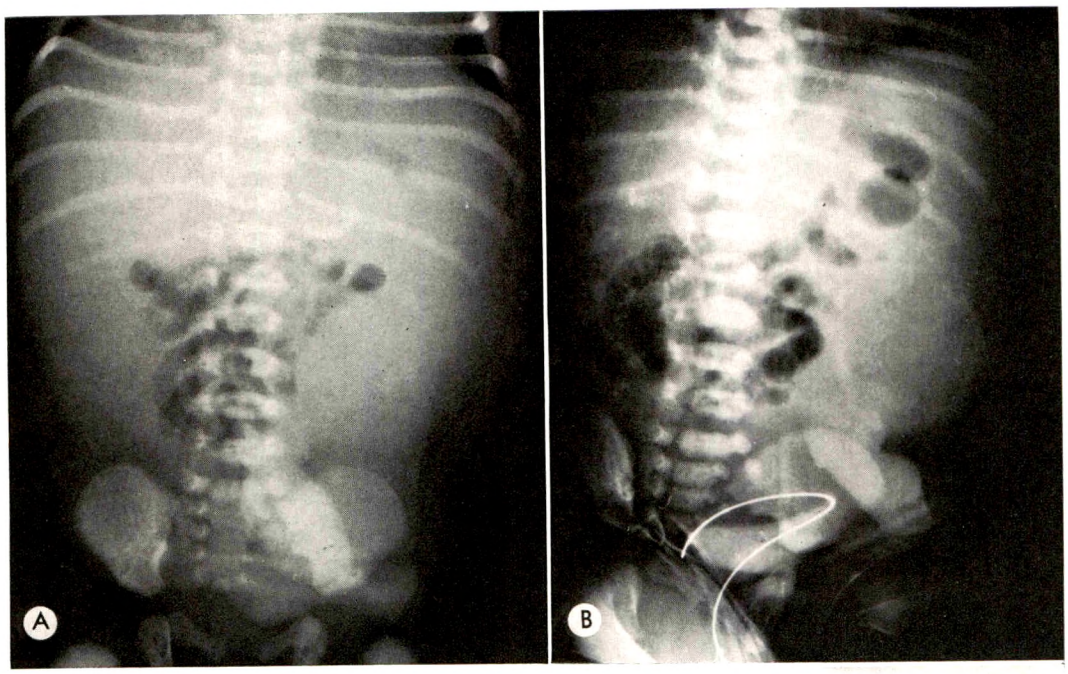
Because of the difficulty of separating the cyst from it, the stretched, distorted ovarian remnant was removed in all cases, and the ipsilateral fallopian tube was excised in

TABLE II

PRESENT SERIES OF NEONATAL OVARIAN CYSTS

Case	Pathology	Side	Size (cm.)	Location of Mass by Physical Examination	Total Body Opacification	Barium Enema Study	Surgical Findings	Comment
I.	Corpus luteum cyst	R	5×3×3	Left paraumbilical	3+ (Fig. 1, *A–C*)		Necrosis suggesting torsion; rupture; 450 cc. ascites	Polyhydramnios; placenta resembled that of maternal diabetes;[9] tachypnea, relieved by aspiration of ascitic fluid
II.	Follicular cyst	R	10	RLQ	3+ (Fig. 2, *A–C*)	Right colon displaced superiorly		
III.	Simple cyst	L	5	Right paraumbilical	3+ (Fig. 3, *A* and *B*)	Right colon elevated	Torsion and infarction	
IV.	Hemorrhagic cyst (probably a follicular cyst originally)	L	6.5×7	Extended from RLQ to LUQ	1+			Idiopathic jaundice and omphalitis; small contralateral cyst not removed; child well 13 years later. Caused moderate ureteral dilatation
V.	Theca-lutein cyst	L	11×7×7	Filled most of the abdomen especially the left side	1+			
VI.	Hemorrhagic cyst (probably a follicular cyst originally)	L	2.8×1.8×1	LLQ	0			
VII.	Follicular cyst	Bilateral	11.5×8.5×3.5 and 3.8×1	RUQ	1+	Cecum elevated		Vomiting; maternal pre-eclampsia
VIII.	Paraovarian cyst	R	7.5×3	Right abdomen	3+		Torsion 720°; adherent to terminal ileum	
IX.	Simple cyst	R	11×7×8	Lower abdomen	2+			Polyhydramnios; mass transilluminated

LUQ= left upper quadrant; RUQ= right upper quadrant.



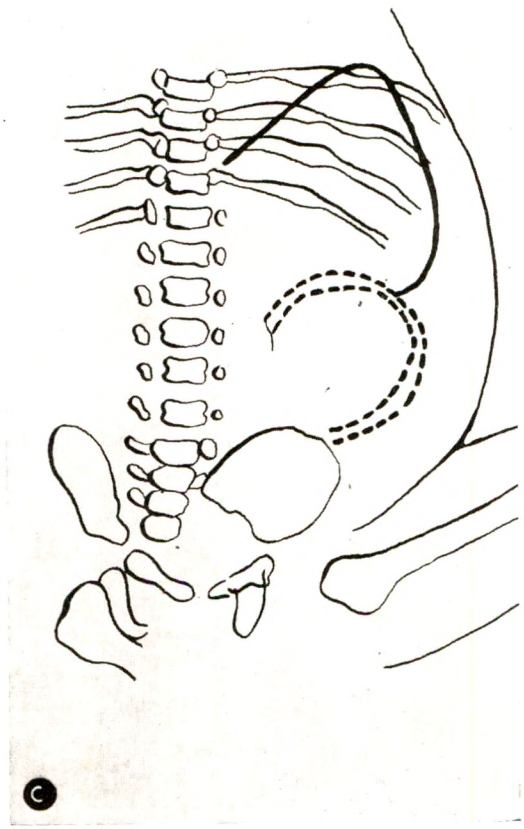


FIG. 1. Case 1. (*A*) Roentgenogram of the abdomen showing central localization of intestinal gas somewhat suggestive of ascites. In addition, a left-sided mass is seen. (*B*) Five minute roentgenogram from a high dose intravenous pyelography showing total body opacification. A mass with a wall of variable thickness and a lucent (avascular) center is seen in the left paraumbilical area. (*C*) Line drawing showing the cyst just below the spleen.

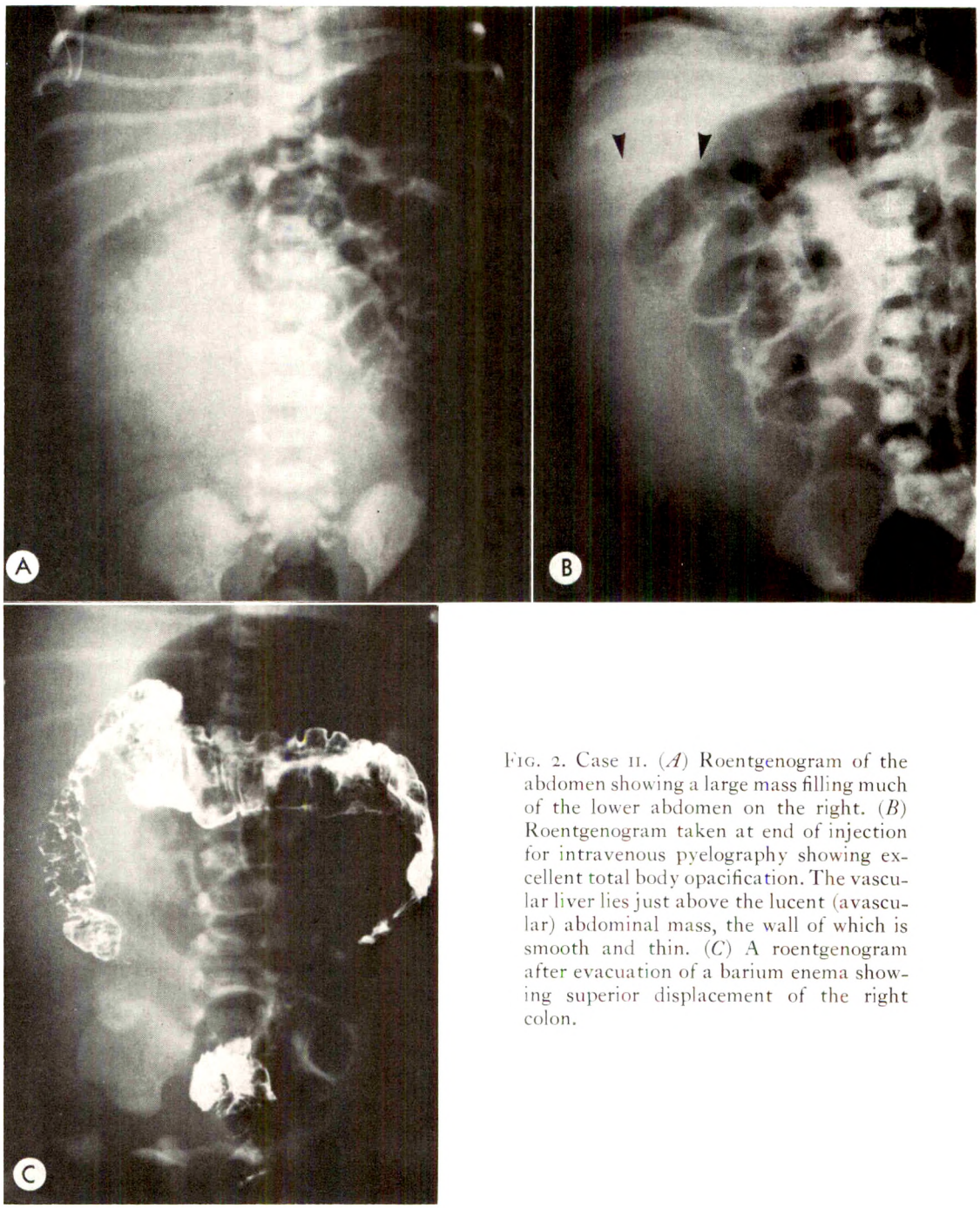


FIG. 2. Case II. (*A*) Roentgenogram of the abdomen showing a large mass filling much of the lower abdomen on the right. (*B*) Roentgenogram taken at end of injection for intravenous pyelography showing excellent total body opacification. The vascular liver lies just above the lucent (avascular) abdominal mass, the wall of which is smooth and thin. (*C*) A roentgenogram after evacuation of a barium enema showing superior displacement of the right colon.

about half the cases. In Case VII, the infant with bilateral involvement, the right ovary with its large cyst was removed, and the smaller cyst of the left ovary was also removed. As the patient may well have been castrated by the procedure, ovarian transplantation was contemplated. Unfortunately the patient's family was rift with social problems, and she was lost to follow-up. By comparison, a small contralateral cyst in Case IV was ignored; this cyst presumably regressed (Table II). Resolution of

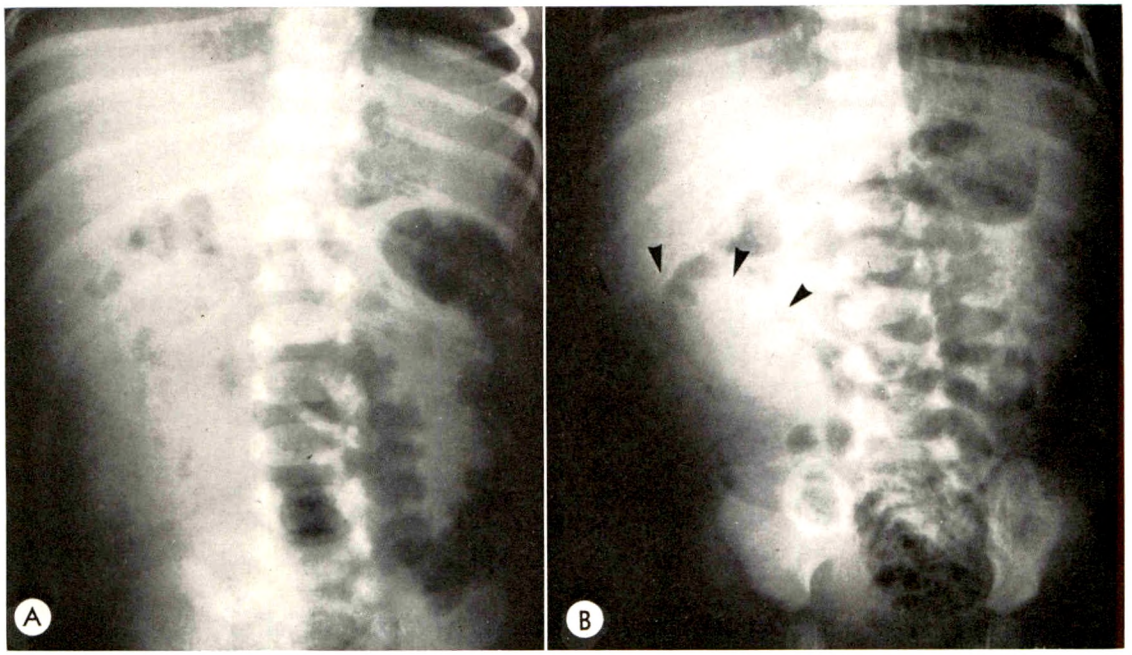


FIG. 3. Case III. (*A*) Roentgenogram of the abdomen showing a right-sided abdominal mass. (*B*) One minute pyelogram showing total body opacification. The vascular liver and kidney are noted above and behind the lucent (avascular) cyst.

a neonatal ovarian cyst is not uncommon.[7,21] All our patients had an uneventful postoperative recovery.

In 2 of these newborns (including the child with involvement of both ovaries) the cysts were follicular. Bleeding into a cystic atretic ovarian follicle is common in adults,[25] and the 2 hemorrhagic cysts probably were also originally follicular cysts, although they could perhaps have been infarcted serous cysts. Two simple cysts and 1 theca-lutein cyst were found. A single corpus luteum cyst developed in a patient with no history of maternal hormonal medication; the cellular lining was exactly the same as would be found in a corpus luteum cyst of pregnancy. The final case was felt to be a paraovarian cyst, because of the absence of any ovarian tissue in the cyst. The smallest cyst measured 2.8×1.8×1 cm., and the largest 3 were approximately 11×8 cm. The largest neonatal cysts recorded in the literature measured 19×12 cm.[12,17] (The crown-heel length of the average newborn is only 50 cm.)

Follow-up data could be obtained on only 5 of the 9 patients. All were doing well and had no further problems. The mothers of these patients had had no other children with ovarian cysts or any other congenital disease. One of the children had started normal menses; the others had not yet reached menarche.

DISCUSSION

Marshall[22] categorized 4 types of neonatal ovarian cysts, microscopically distinguished by their lining cells: (1) benign cysts of germinal or graafian epithelial origin such as simple cysts, serous cysts, cystomas, cystadenomas, follicular cysts, theca-lutein cysts and corpus luteum cysts; (2) benign and malignant granulosa cell tumors; (3) benign cystic teratomas; and (4) mesonephromas (paraovarian cysts). Granulosa cell tumors, teratomas, and paraovarian cysts are very uncommon in newborns, and the first general category includes the great majority of neonatal ovarian cysts now reported (Tables I and II). Simple or serous cysts, theca-lutein cysts, and corpus luteum cysts each ac-

count for about 10 per cent of the total. It should be noted that when the lining of a follicular cyst is destroyed by hydrostatic pressure, it becomes a simple cyst.[15]

The causes of the various types of neonatal ovarian cysts are uncertain and may be diverse. Some authors believe that maternal hormones are the predominant factor and that true graafian follicle development rarely occurs in the fetus.[11] Potter,[30] however, considers the presence of graafian follicles in the newborn to be a common finding. In addition, she disagrees with the general feeling that the development of follicles depends entirely on maternal hormones, since that would leave the presence of follicles in infancy and childhood unexplained. The occurrence of theca-lutein cysts in adults with chorioepithelioma and hydatidiform mole[25] suggests that some neonatal theca-lutein cysts may also be related to elevated chorionic gonadotropin levels. Ovarian cysts have been produced in rats injected with chorionic gonadotropin.[14] The development of polycystic ovaries after treatment with norethynodrel (enovid) suggests that pituitary dysfunction may be involved in cyst formation.[34] A series of autopsies on stillborns showed luteinizing cysts in 3 out of 5 infants of diabetic mothers, but in only 8 per cent of the group with nondiabetic mothers, while the mother of our newborn (Case 1) with a corpus luteum cyst was not diabetic, the placenta did show many changes typical of diabetes.[9] In conclusion, although the development of ovarian cysts may be only an exaggeration of normal follicular development, many bits of evidence suggest that maternal hormones play a part.

Paraovarian cysts originate from remnants of the mesonephros and paramesonephros, which come to be located in the mesosalpinx. Hydatids of Morgagni, Kobelt's cysts, epoöphoron and paroöphoron cysts, cysts of the rete ovarii, and Müllerian cysts are included in this generic term. Exact site of origin, therefore, plays a large part in determining the type of paraovarian cysts. Paraovarian cysts are thin-walled with only a single layer of columnar or cuboidal epithelium.[28] Although usually small, they may become very large. Several cases of paraovarian cysts in the pediatric age group have been reported,[28,31,33] but only 1 was a newborn.[33] Our Case VIII could be distinguished from a follicular cyst by its origin from the mesosalpinx rather than from the ovary itself as well as by the absence of any ovarian tissue in the cyst wall.

Intravenous urography is critical in the diagnosis of a neonatal abdominal mass. The majority of such masses are renal; hydronephrosis and multicystic kidney account for about 40 per cent of the total.[13] The usefulness of urography is not limited to urinary tract masses, moreover, since the total body opacification effect[23,26] is an excellent means of identifying and further characterizing abdominal masses. This effect is merely the apparent lucency developed by avascular masses during the circulation of injected contrast material through adjacent well-vascularized structures (Fig. 1–3). Such a lucency was recognized on the early urograms of 8 of the present 9 cases; in Case 1, the effect correctly predicted that the wall was thick rather than thin. Total body opacification is peculiarly useful in neonatal abdominal masses for reasons discussed elsewhere.[23] The roentgenologic evaluation of a neonatal abdominal mass generally ceases with a well-conducted intravenous urography,[13] although cystography and barium studies are sometimes useful and arteriography will be needed in rare cases.

The mothers of two of our patients with very large cysts had polyhydramnios (Cases 1 and IX). Perhaps the masses caused pressure on the small bowel and inhibited the swallowing and absorption of amniotic fluid. Polyhydramnios is fairly frequent in intrinsic gastrointestinal obstruction. Two other cases of polyhydramnios occurring with ovarian cysts have been reported.[17,24]

Probably many ovarian cysts resolve spontaneously and cause no problem. Some cysts are found incidentally at laparotomy.[31] Mainolfi *et al.*[21] noted a small cyst of

the left ovary when removing an 8 cm. cyst from the right side. At exploration during inguinal herniorrhaphy 4 months later, the cyst was no longer visible. These findings encourage surgical conservatism, especially when both ovaries contain cysts. In the future some techniques of aspiration of cyst contents may make removal of any remaining normal ovarian tissue unnecessary.

SUMMARY

Nine newborns with ovarian cysts of various histologic type are reported, and 28 similar cases from the literature are reviewed.

All nine cases were found on the first day of life. No child had any other congenital anomaly. The mothers of 2 of these girls had polyhydramnios.

The key diagnostic procedure is intravenous urography. The total body opacification effect, indicating avascularity, occurred during urography in 8 infants. Torsion, infarction, hemorrhage, necrosis, and rupture each occurred in 1 or more of these 9 cases. In 1 infant, cysts arose from both ovaries.

Surgical removal was readily accomplished and none of the cysts was malignant.

No further problems have arisen in those 5 children it has been possible to follow.

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REFERENCES

1. Ahlvin, R. C., and Bauer, W. C. Luteinized cysts in ovaries of infants born of diabetic mothers. *A.M.A. Am. J. Dis. Child.*, 1957, *93*, 107–109.
2. Astedt, B. Ovarian tumors in newborns. *Nord med.*, 1967, *77*, 87–88.
3. Boles, E. T., Jr., Hardacre, J. M., and Newton, W. A. Ovarian tumors and cysts in infants and children. *A.M.A. Arch. Surg.*, 1961, *83*, 580–592.
4. Brune, W. H., Pulaski, E. J., and Shvey, H. E. Giant ovarian cyst: report of case in premature infant. *New England J. Med.*, 1957, *257*, 876–878.
5. Bulfamonte, J. C. Large ovarian cyst in newborn child. *Am. J. Surg.*, 1942, *55*, 175–176.
6. Cox, H. D., Campbell, R. M., Vishniarsky, S., and Arayata, F. R. Huge ovarian cyst with torsion in newborn infant. *Virginia M. Month.*, 1969, *96*, 96–100.
7. Davis, A. E., Jr. Giant ovarian cyst of newborn. *Minnesota Med.*, 1961, *44*, 330–333.
8. Doran, A. H. Large ovarian tumors in 7 month old child. *Trans. Path. Soc.*, London, 1889.
9. Driscoll, S. Personal communication.
10. Finola, G. C., Quitangon, L., and Pontius, G. U. Large ovarian cyst in newborn infant. *Am. J. Obst. & Gynec.*, 1957, *73*, 690–692.
11. Garfinkle, B., and Rosenthal, A. H. Teratomas and follicular cysts of ovary in children. *Am. J. Obst. & Gynec.*, 1962, *83*, 101–104.
12. Graves, G. Y., McIlroy, D. B., and Hudson, G. W. Ovarian cyst in premature infant. *A.M.A. Am. J. Dis. Child.*, 1951, *81*, 256–258.
13. Griscom, N. T. Roentgenology of neonatal abdominal masses. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1965, *93*, 447–463.
14. Gold, J. J., Castillo, E., Borashek, S., and Scommegna, A. Factors in polycystic ovary development. *Fertil. & Steril.*, 1965, *16*, 560–569.
15. Karrer, F. W., and Swenson, S. A. Twisted ovarian cyst in newborn infant: report of case. *A.M.A. Arch. Surg.*, 1961, *83*, 921–924.
16. Kilman, J. W., Waldhauser, J. A., Vellios, F., and Battersby, J. S. Ovarian tumors in infants and childhood. *Am. J. Surg.*, 1967, *113*, 772–776.
17. Korobkin, M., de Lorimier, A. A., and Gooding, C. A. Ovarian cysts presenting in the neonatal period. *Brit. J. Radiol.*, 1970, *43*, 820–823.
18. Kundstadter, R. H., Schultz, A., and Strauss, A. A. Large ovarian cyst in newborn infant. *Am. J. Dis. Child.*, 1950, *80*, 993–998.
19. Lorrain, J. Ovaries polykystiques à la suite d'un traitment au norethynodrel. *Union méd. Canada*, 1966, *95*, 1053–1055.
20. Longino, L. A., and Martin, L. W. Abdominal masses in newborn infant. *Pediatrics*, 1958, *21*, 596–608.
21. Mainolfi, F. G., Standiford, J. W., and Hubbard, T. B., Jr. Ruptured ovarian cyst in newborn. *J. Pediat.*, 1968, *3*, 612–616.
22. Marshall, J. R. Ovarian enlargements in first year of life. *Ann. Surg.*, 1965, *161*, 372–377.
23. Martin, D. J., Griscom, N. T., and Neuhauser, E. B. D. Further look at total body opacification effect. To be published.
24. Neikirk, W. I., and Hudson, H. W., Jr. Giant

ovarian cyst in newborn infant. *New England J. Med.*, 1948, *239*, 468–469.

25. Novak, E. Gynecological and Obstetrical Pathology. W. B. Saunders Company, Philadelphia, 1962, pp. 319–320.
26. O'Conner, J. F., and Neuhauser, E. B. D. Total body opacification in conventional and high dose intravenous urography in infancy. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1963, *90*, 63–71.
27. Parry, N. G. Large ovarian cyst in newborn infant. *Quart. Bull. Northwestern Univ. M. School*, 1955, *29*, 396–399.
28. Perry, R. W. Ovarian tumors in pediatric patient. *Harper Hosp. Bull.*, 1961, *19*, 209–221.
29. Pierson, M., Dellestable, P., Colligon, P., and Duprez, A. Rupture d'un volumineux kyste congénital de l'ovaire chez un nouveau-né de 9 heures. *Bull. Féd. soc. gynéc. et obst.*, 1964, *16*, 183–188.
30. Potter, E. L. Pathology of the Fetus and Infant. Year Book Publishers, Inc., Chicago, 1962, pp. 466–468.
31. Reis, R. L., and Koop, C. E. Ovarian tumors in infants and children. *J. Pediat.*, 1962, *60*, 96–102.
32. Smith, R. C. Simple ovarian cyst in newborn infant. *Pediatrics*, 1954, *14*, 232–234.
33. Thatcher, D. S. Ovarian cysts and tumors in children. *Surg., Gynec. & Obst.*, 1903, *117*, 477–483.
34. Tietz, R. G., and Davis, J. B. Ruptured ovarian cyst in newborn infant. *J. Pediat.*, 1957, *51*, 564–565.
35. Tyson, W. T., Kegan, J. M., and Gold, D. Ovarian cyst in newborn infant. *J. Pediat.*, 1957, *51*, 564–565.
36. Watson, E. A., and Henderson, A. T. Giant ovarian cyst of newborn. *Nebraska M. J.*, 1955, *40*, 397–399.

OVARIAN TERATOMA IN CHILDHOOD*

DIAGNOSTIC ULTRASONIC AND ROENTGENOGRAPHIC CORRELATION

By ROGER A. HYMAN, M.D., LAJOS I. VON MICSKY, M.D., *and* NATHANIEL FINBY, M.D.
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CYSTIC teratomas of the ovary are infrequently encountered prior to puberty.[4] Approximately 1 per cent of tumors in female children are ovarian in origin, and, of these, approximately one-half are teratomas.[4,6,7] The vast majority of these are benign and cystic. Solid, rapidly growing malignant ovarian teratomas are rarely encountered. The term "dermoid cyst" is reserved for these tumors whose elements are entirely derived from ectoderm. Many pathologists believe that this rarely occurs and that if sufficient histologic sections are taken, elements from all 3 germ layers will be identified.[2,7] The term "benign cystic teratoma" is therefore thought to be preferable.

Ovarian teratomas are usually not discovered until they grow large enough to cause a mass or to twist on their vascular pedicles and cause pain. Although this pathologic entity occurs infrequently compared to appendicitis, with which it is frequently confused in children when no mass is palpable, it should be considered in the differential diagnosis of abdominal pain in female children.

The present case demonstrates that an accurate preoperative diagnosis can be made in the clinical absence of a mass, including the determination of whether the tumor is cystic or solid, and therefore its malignant potential,[2] when roentgenographic and ultrasonic studies are utilized.

REPORT OF A CASE

J.L., an 8 year old girl, was admitted to the St. Luke's Hospital Center with a 4 day history of left-sided abdominal pain that was crampy in nature. She had a low grade fever, and vomited upon eating or drinking. She had not moved her bowels for 2 days. Physical examination revealed the child lying on her right side with both legs flexed. The abdomen was tender, particularly in the left lower quadrant, but there was no rebound. No masses were palpable. The white blood cell count was 12,600.

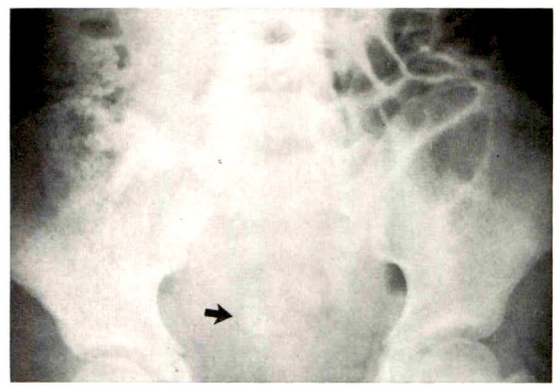


FIG. 1. A supine roentgenogram of the abdomen shows an area of amorphous calcification (arrow) overlying the right lower portion of the sacrum.

Roentgenographic and Ultrasonic Findings. A supine roentgenogram of the abdomen (Fig. 1) demonstrated an area of amorphous calcification overlying the right lower portion of the sacrum. Haziness in the pelvis suggested the possibility of a mass. Intravenous pyelography was then performed and demonstrated dilatation of the right collecting system from the renal pelvis to the level of the pelvic brim, as well as the calcification and mass effect in the pelvis seen previously (Fig. 2). A lateral roentgenogram obtained during the intravenous pyelography (Fig. 3) showed the abnormal calcium density to be located just posterior to the anterior abdominal wall. The roentgenographic findings of the pelvic mass with dense amorphous calcification causing partial obstruction of the right ureter strongly suggested the diagnosis of ovarian teratoma.

Diagnostic ultrasonic studies were then performed to confirm this impression, and to de-

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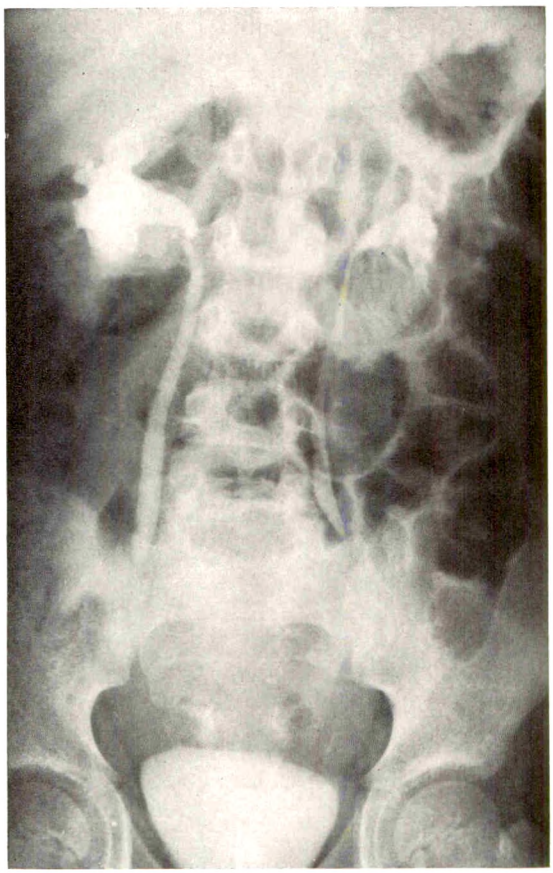


FIG. 2. An intravenous pyelogram demonstrates dilatation of the right collecting system to the level of the pelvic brim as well as the amorphous calcific density.

termine if the mass was solid or cystic.

Sonograms of the abdomen and pelvis (Fig. 4, *A–C*) showed a moderately distended bladder with an oval sonolucent mass cephalad to it and just posterior to the anterior abdominal wall. The mass measured 9×6×6 cm. sonographically. The wall-content interface of the mass was smooth and well-defined except in the midportion of its anterior wall where a sessile sono-opaque structure projected into the lumen of the cystic mass. The mass was moderately transonic. The position of the sono-opaque projection correlated well with the position of the amorphous calcific density seen roentgenographically. The ultrasonic findings, therefore, supported the diagnosis suspected roentgenographically, and, in addition, demonstrated the cystic quality of the mass.

Surgical and Pathologic Findings. At operation, an oval cystic mass was found attached to the left ovary which had undergone torsion. It had a smooth, glistening surface and contained serous fluid. Histologic section showed a delicate cyst wall lined by a stratified cuboidal to squamous epithelium. No malignant changes were identified. Thickened areas within the wall contained bone, cartilage, hair follicles, keratin cysts, sweat glands and a few hyperplastic thyroid acini. These findings established the pathologic diagnosis of benign cystic teratoma.

DISCUSSION

In the present case, the amorphous calcification seen in the pelvis on the roentgenograms suggested the diagnosis of ovarian teratoma. However, only about one-quarter to one-half of reported cases show calcification.[1,6,7] In addition, as in our case, occasionally no mass is palpable.[6,7] This is usually due to the difficulty in performing adequate abdominal and pelvic examina-

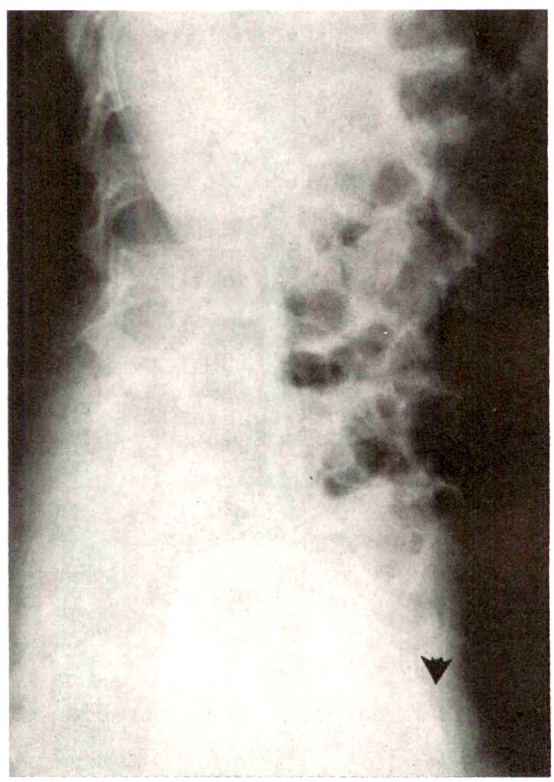


FIG. 3. A lateral roentgenogram obtained during intravenous pyelography shows the calcific density (arrow) to be located just posterior to the lower anterior abdominal wall.

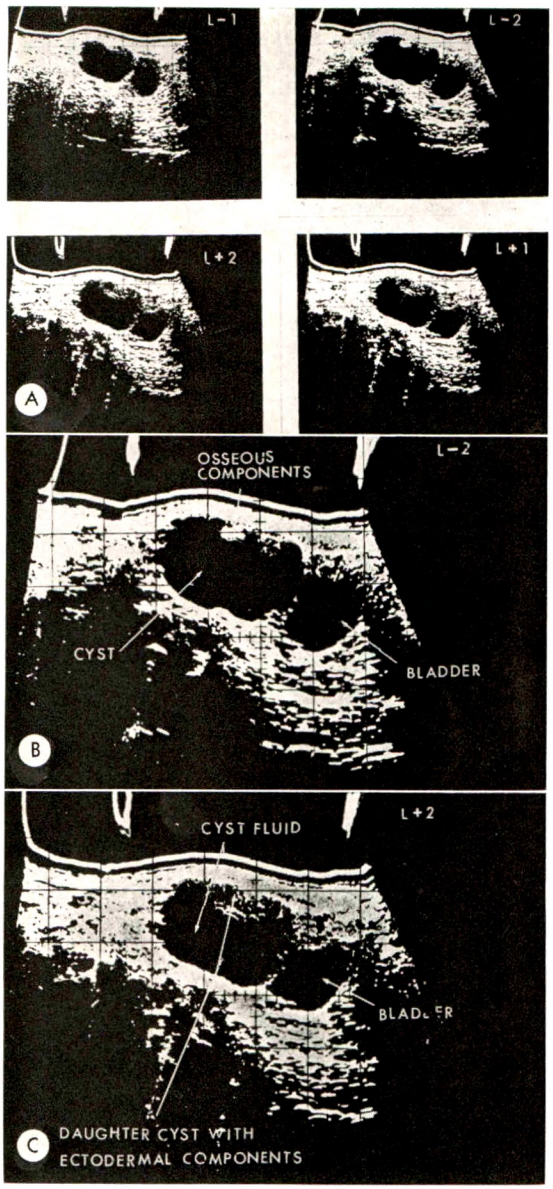


FIG. 4. (*A–C*) Longitudinal ultrasonic sections through the abdomen and pelvis demonstrate a sonolucent mass superior to the bladder. A sessile projection (*B*) within the mass can be seen in the same position where the amorphous calcification was shown on the lateral roentgenogram of the intravenous pyelography (Fig. 3).

tions in the presence of marked guarding which is frequently encountered with torsion of ovarian neoplasms. Therefore, occasions arise in which there is neither evidence of calcification in the pelvis roentgenographically, nor a mass on physical examination. When these children present with pain, then the diagnosis of acute appendicitis is frequently made, particularly since ovarian teratomas are more common on the right than on the left.[3,5] Ultrasonic evaluation has a contribution to make in this entity, whether the patient presents with pelvic calcification roentgenographically, a mass on physical examination, both, or neither. It is particularly helpful in fully establishing a preoperative diagnosis when no mass is palpable, as occurred in our case.

The recommendations of routine ultrasonic evaluation of the abdomen and pelvis in children presenting with acute abdominal pain is not always practical; however, ultrasonic studies can be extremely useful in selected cases of abdominal pain in children. In addition, when there is a known mass, its usefulness in determining whether the mass is cystic or solid is now well established.

Ultrasound represents a diagnostic modality which can be used in conjunction with other diagnostic procedures, particularly conventional diagnostic roentgenology, in order to obtain as much pertinent information as possible prior to surgery.

SUMMARY

A case of cystic ovarian teratoma with torsion of its pedicle is reported in a child.

Roentgenographic and ultrasonic findings are demonstrated and the value of ultrasonic evaluation in conjunction with routine roentgenography is discussed.

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The authors wish to thank Dr. Robert E. McCabe, Jr., for referring this patient for roentgenographic evaluation.

REFERENCES

1. BOLES, E. T., JR., HARDACRE, J. M., and NEWTON, W. A., JR. Ovarian tumors and cysts in infants and children. *A.M.A. Arch. Surg.*, 1961, *83*, 580–592.

2. DARGEON, H. W. Tumors of Childhood. Paul B. Hoeber, Inc., New York, 1960, pp. 252–257.
3. EIN, S. H., DARTE, J. M. M., and STEPHENS, C. A. Cystic and solid ovarian tumors in children: 44 year review. *J. Pediat. Surg.*, 1970, *5*, 148–156.
4. FERGUSON, C. C., and GOODHAND, T. K. Dermoid cyst of ovary presenting as cause of acute abdominal pain in childhood. *Pediatrics*, 1959, *23*, 948–950.
5. FORSHALL, I. Ovarian neoplasms in children. *Arch. Dis. Childhood*, 1960, *35*, 17–21.
6. IRONS, G. B., JR., HOGE, R. H., and SALZBERG, A. M. Ovarian teratomas in children. *Clin. Pediat.*, 1966, *5*, 151–153.
7. KILMAN, J. W., WALDHAUSEN, J. A., VELLIOS, F., and BATTERSBY, J. S. Ovarian tumors in infants and children. *Am. J. Surg.*, 1967, *113*, 772–776.

Vol. 116, No. 3

ULTRASONIC EVALUATION OF MASSES IN PEDIATRIC PATIENTS*

By BARRY B. GOLDBERG, M.D., MARIE A. CAPITANIO, M.D., *and* JOHN A. KIRKPATRICK, M.D.
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ULTRASOUND has been used extensively to evaluate masses in adults;[2,3,5] however, there are only scattered reports regarding its use in pediatric patients.[4,6,7] Over a 2 year period, 45 patients with masses were examined utilizing ultrasound; these included patients in whom the lesions were superficial, mediastinal, abdominal, and retroperitoneal. By means of ultrasound, an attempt was made to determine if a mass was cystic, solid, or both cystic and solid. In almost all instances the results of the ultrasonic examination were correlated with the characteristics of the lesion obtained either at surgery or autopsy.

TECHNIQUE

A commercially available ultrasonic machine with a standard 2.25 megaHertz transducer was utilized in the study. An A-mode type of ultrasonic display was used in which the reflections (echoes) produced at various tissue interfaces were displayed on an oscilloscopic screen as vertical deflections. The base-line is calibrated in centimeters, allowing for easy measurement of the size and depth of the mass in question. A water-soluble gel was used between the transducer and the skin to eliminate any trapped air which would prevent adequate transmission of the ultrasonic beam. The ultrasonic tracings obtained were recorded from the oscilloscope using self-developing film.

After the localization of a mass by either palpation or roentgenographic examination, the ultrasonic transducer was placed on the skin over the area of interest and echoes were obtained. The sensitivity was then adjusted so that at a low gain setting, the reflections from the anterior and posterior walls of the mass were recorded. A second examination was made at a high gain setting, and at this level the pattern produced by a solid mass filled in with echoes, whereas the pattern of a cystic mass remained relatively echo-free. A mass with solid and cystic components resulted in a mixed (complex) pattern (Fig. 1). In order to establish the proper sensitivity setting, evaluation of a full urinary bladder can be used as a substitute for a cystic mass and a kidney for a solid one. Initially and at the proper low gain setting, only the anterior and posterior walls of these structures should be recorded. The echoes from the posterior wall of the bladder will be numerous due to minimal attenuation of the ultrasonic beam as it passes through the fluid. The gain is then increased until the echo-free zone starts to fill in slightly with echoes from the edges. At this high gain setting, the echo-free zone from the kidney should completely fill in. Incidentally, it was found that the settings used were the same as those previously established for detecting and evaluating masses in adults.[2,4] However, whereas in adults the smallest deep cystic lesion that can be detected was found to approximate $2\frac{1}{2}$ cm. due to the presence of reverberations from the overlying structures, in infants and children a deep cystic mass as small as $1\frac{1}{2}$ cm. can be detected. When a cystic ultrasonic pattern was thought to be due to ascites or pleural effusion, the patient was turned without changing the position of

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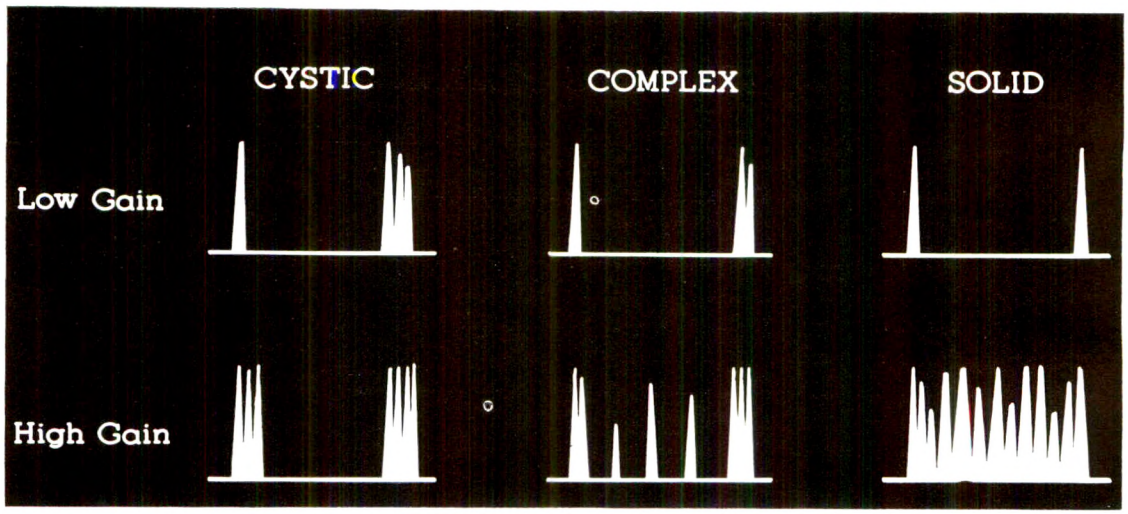


FIG. 1. Diagrammatic representation of ultrasonic patterns.

the transducer; if the fluid was not loculated it would shift and the echo-free zone would disappear.[1]

RESULTS

Forty-five masses were examined in infants and children ranging in age from the newborn period to 14 years. The masses were classified into 3 distinct ultrasonic patterns as cystic, solid, and complex (a combination of the 2). Twenty-seven of them were retroperitoneal in location, 9 were intraabdominal, 3 were mediastinal, and 3 were superficial. In addition, there were 2 patients who had ascites and 1 who had a pleural effusion. Of the 45 masses,

TABLE I

LOCATION OF MASS

	No.	Cystic	Solid	Complex
A. Retroperitoneal	27	9	8	10
B. Intraabdominal	9	2	6	1
C. Chest and Mediastinum	3	—	3	—
D. Superficial	3	—	3	—
E. Free Fluid Collections	3	3	—	—
Total	45	14	20	11

TABLE IIA

TYPE OF MASS

	No.	Cystic	Solid	Complex
A. *Retroperitoneal*				
1. Renal				
a. Normal	6	—	6	—
b. Hydronephrosis	3	3	—	—
c. Multicystic	5	5	—	—
d. Polycystic	3	—	—	3
e. Wilm's tumor	4	—	—	4
f. Leiomyomatous hamartoma	2	—	—	2
g. Neuroblastoma	2	—	2	—
2. Adrenal				
Carcinoma with pseudocyst	1	—	—	1
3. Retroperitoneal				
Chronic hematoma	1	1	—	—

FIG. 3. *Wilms' Tumor.* Intravenous urogram reveals distortion of the right calyceal system. Lead marker denotes region from which ultrasonic tracings were obtained. Upper pair of ultrasonic tracings (*A*) obtained from the abnormal kidney revealed a complex ultrasonic pattern. Lower pair of ultrasonic tracings (*B*) obtained from the normal left kidney revealed, as expected, a solid ultrasonic pattern.

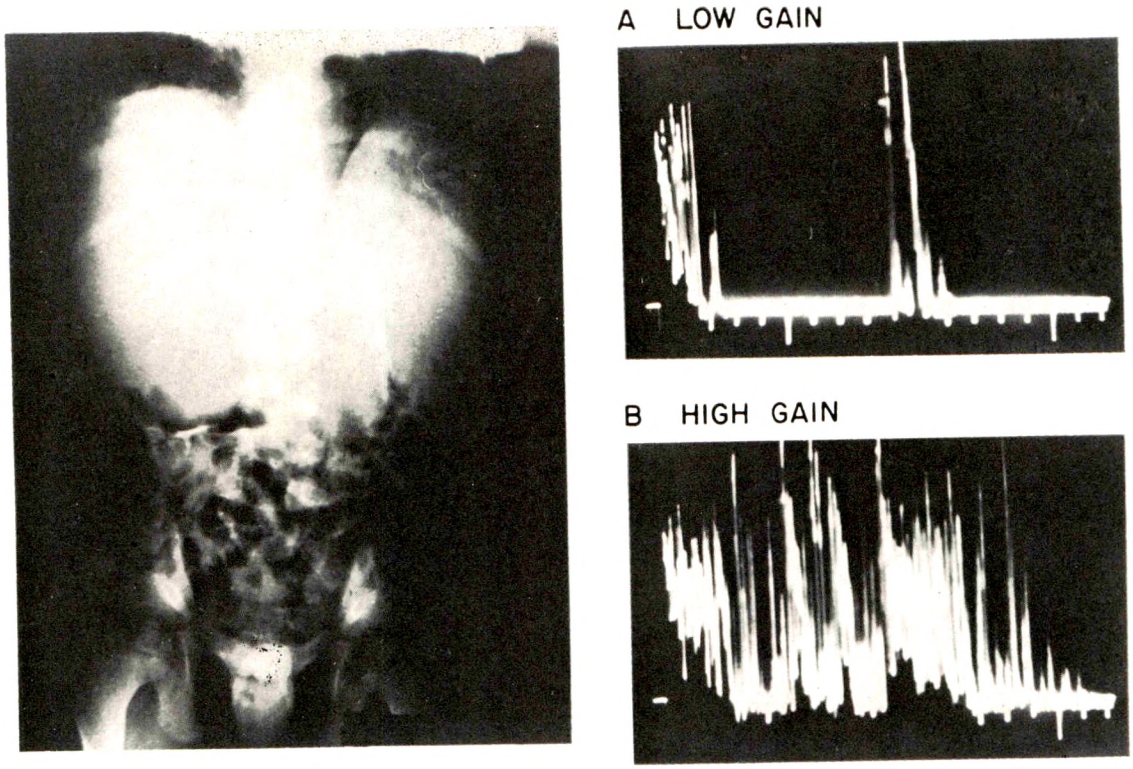
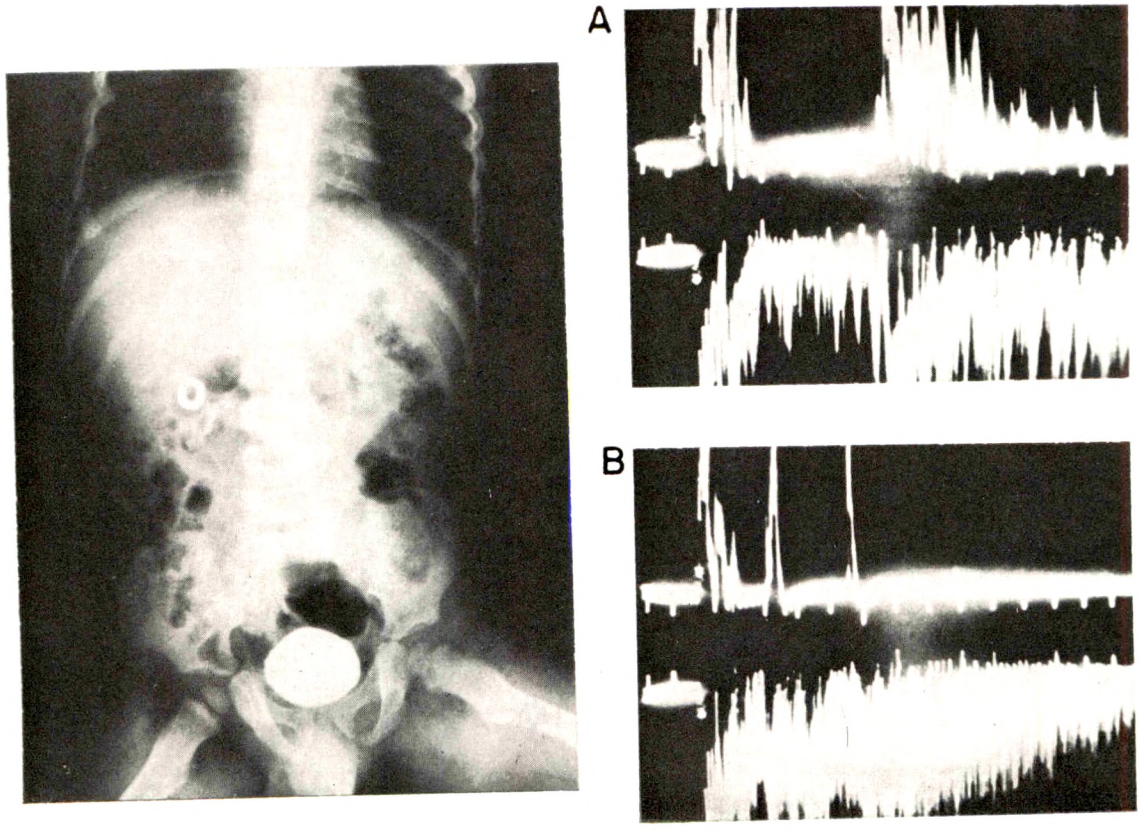


FIG. 2. *Neuroblastoma.* Intravenous urogram reveals a right suprarenal mass. Ultrasonic tracings are those of a typical solid ultrasonic pattern with filling in of the echo-free zone at high gain.



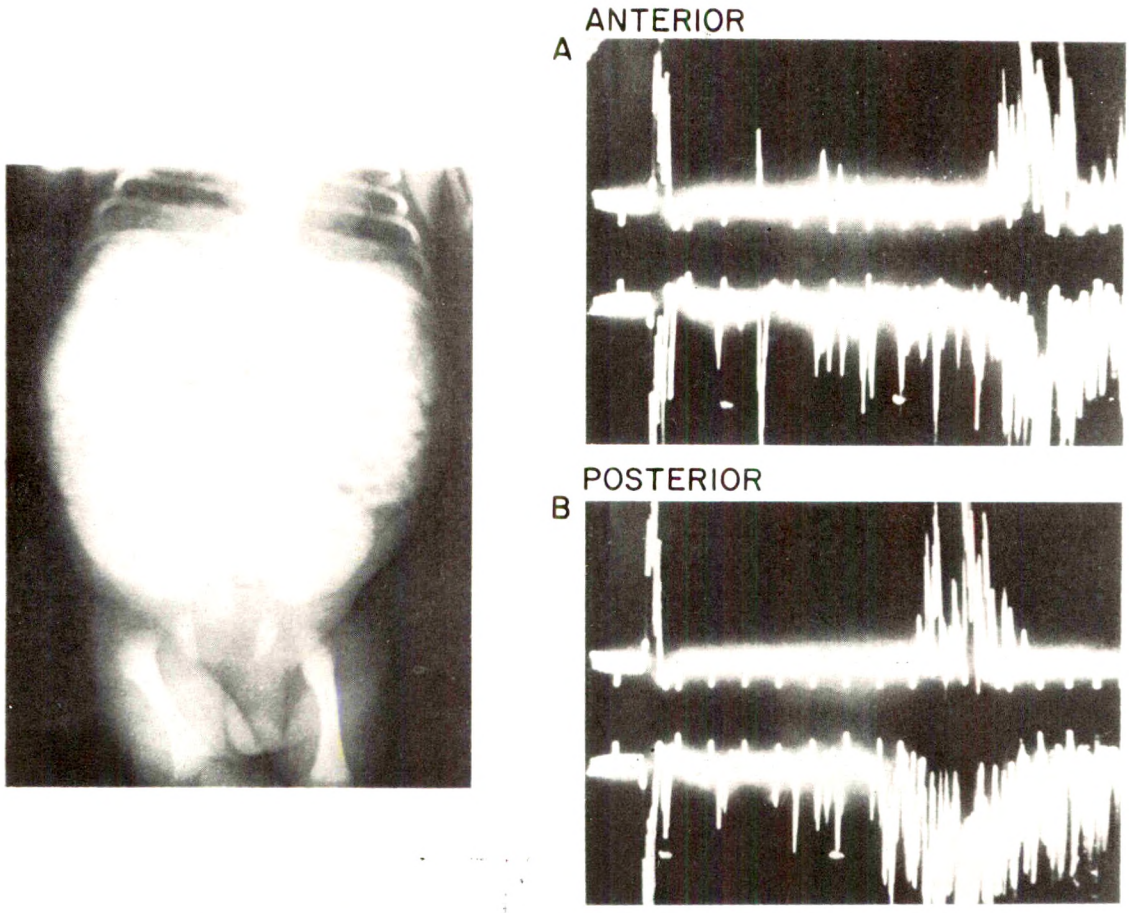


FIG. 4. *Polycystic Kidneys.* Intravenous urogram reveals bilateral infantile polycystic disease of the kidneys. Upper (*A*) and lower (*B*) pair of ultrasonic tracings are typical complex ultrasonic tracings.

14 produced cystic patterns, 20 produced solid patterns, and 11 produced complex patterns (Table 1).

RETROPERITONEAL MASSES

The majority of the masses examined were retroperitoneal. In this group of 27, 9 were cystic, 8 were solid, and 10 were complex (Table IIA). As would be expected, the normal kidney and the 2 neuroblastomas produced a solid ultrasonic pattern (Fig. 2). Wilms' tumor and leiomyomatous hamartoma of the kidney produced a complex ultrasonic pattern, which in the Wilms' tumor was due to areas of necrosis and hemorrhage and in the leiomyomatous hamartoma was caused by scattered cystic areas (Fig. 3). In 3 patients with infantile polycystic disease of the kidney, there was a complex ultrasonic pattern due to the minute cysts associated with a proportionally larger solid component (Fig. 4). This was not the case with the 3 hydronephrotic kidneys and 5 multicystic kidneys in which a cystic ultrasonic pattern was predominant, although in several patients, echoes were recorded from septa between the cysts (Fig. 5). As would be expected, an adrenal carcinoma with an associated cyst produced a complex pattern. A retroperitoneal hematoma which followed nephrectomy was accurately measured before and after aspiration (Fig. 6). The collection of fluid did not change configuration with changes in position of the

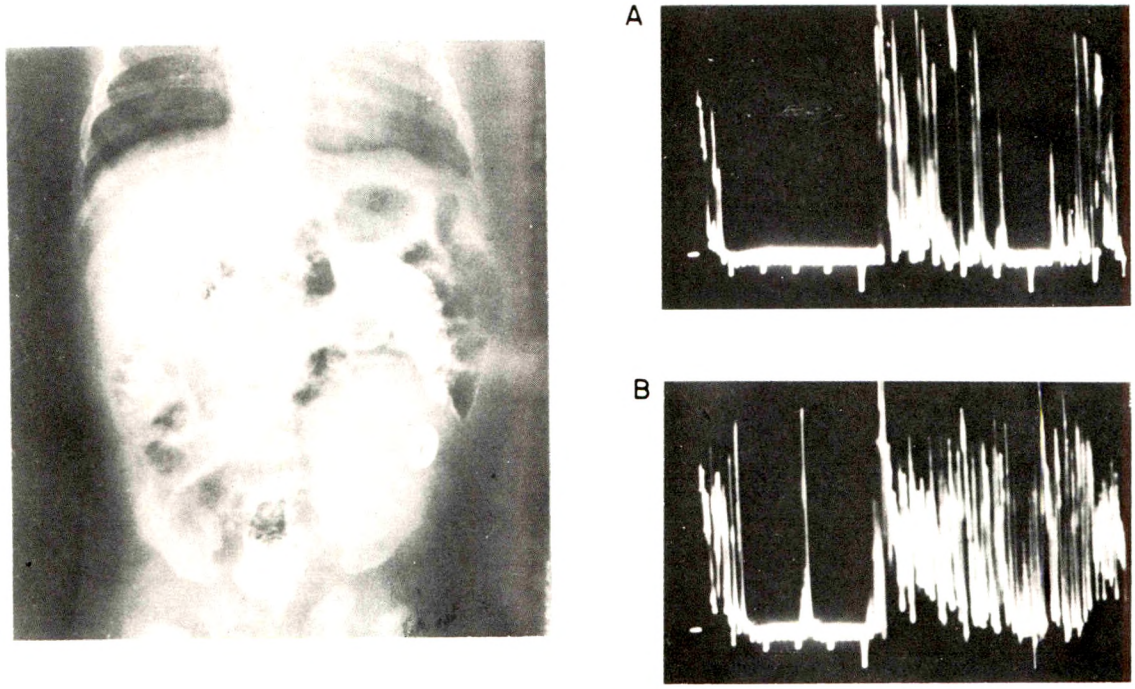


FIG. 5. *Multicystic Kidney*. Roentgenogram reveals a left-sided mass displacing the bowel. Lead marker denotes region from which ultrasonic tracing was obtained. Ultrasonic tracings, low gain (*A*) and high gain (*B*), are those of a predominantly cystic ultrasonic pattern.

patient as would be expected with free fluid.

INTRAABDOMINAL MASSES

There were 9 patients in this group, in 2 the mass was within the pelvis (Table IIB). Six masses produced a solid ultrasonic pattern, 2 of which were sarcomas. A sarcoma which arose in the pelvis had a fluid component which was not appreciated and this is considered to be a missed diagnosis, since a complex ultrasonic pattern should have been detected. A solid pattern was obtained from the normal liver, a complex pattern from the hepatic abscess, and a cystic pattern from the cystic mesenchymal hamartoma of the liver (Fig. 6). In 1 patient in whom biliary atresia was suspected, ultrasound was utilized in order to determine the presence of a gallbladder. A cystic structure was not found, and absence of the gallbladder was confirmed at surgery. A cystic pelvic mass in a female proved to be an ovarian cyst at surgery.

CHEST AND MEDIASTINAL MASSES

The masses encountered in the thorax in this series included 2 neuroblastomas and 1 sarcoma; each produced a solid ultrasonic pattern (Table IIC). Air prevents adequate penetration of the ultrasonic beam

TABLE IIB

TYPE OF MASS

	No.	Cystic	Solid	Complex
B. *Intraabdominal*				
1. Liver				
a. Normal	2	—	2	—
b. Mesenchymal cystic hamartoma	1	1	—	—
c. Abscess	1	—	—	1
d. Biliary atresia	1	—	1	—
e. Neuroblastoma	1	—	1	—
2. Ovarian cyst	1	1	—	—
3. Sarcoma	2	—	2*	—

* One case was incorrectly diagnosed as being solid. At surgery, there was a cystic area; therefore, the mass should have been classified as complex.

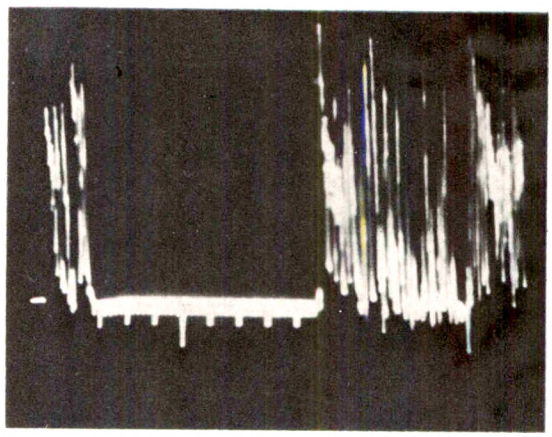


TABLE IIC

TYPE OF MASS

	No.	Cystic	Solid	Complex
C. *Chest and Mediastinum*				
1. Neuroblastoma	2	—	2	—
2. Sarcoma	1	—	1	—

and thus it is not possible to examine lesions within the lungs; on the other hand, lesions involving the pleura or mediastinum can be evaluated by ultrasound.

SUPERFICIAL MASSES

All 3 superficial masses produced solid ultrasonic patterns consistent with the surgical findings (Table IID).

FREE FLUID

In 3 patients with free fluid in the pleural or peritoneal cavities, a cystic ultra-

TABLE IID

TYPE OF MASS

	No.	Cystic	Solid	Complex
D. *Superficial*				
1. Lipoma	2	—	2	—
2. Encephalocele	1	—	1	—

TABLE IIE

TYPE OF MASS

	No.	Cystic	Solid	Complex
E. *Free Fluid Collections*				
1. Pleural effusion	1	1	—	—
2. Ascites	2	2	—	—

FIG. 6. *Retroperitoneal Hematoma.* Serial ultrasonic tracings were obtained during the percutaneous aspiration of fluid from a left-sided retroperitoneal hematoma. Note the progressive decrease in the size of the echo-free zone.

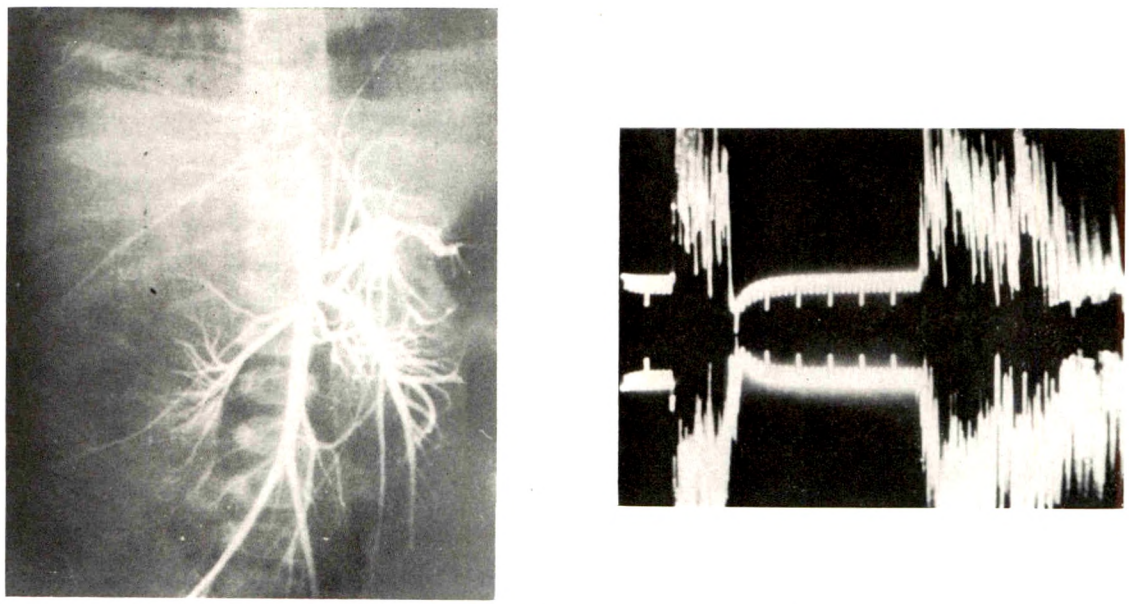


FIG. 7. *Cystic Hamartoma.* Ultrasonic tracings are those of a typical cystic ultrasonic pattern recorded from the right upper quadrant of the abdomen. Arteriogram confirms the presence of an avascular lesion in the liver.

sonic pattern was found (Table IIE). However, unlike loculated fluid or cystic masses, there was a significant decrease or disappearance of the echo-free zone when the patient was moved without changing the placement of the transducer (Fig. 7).

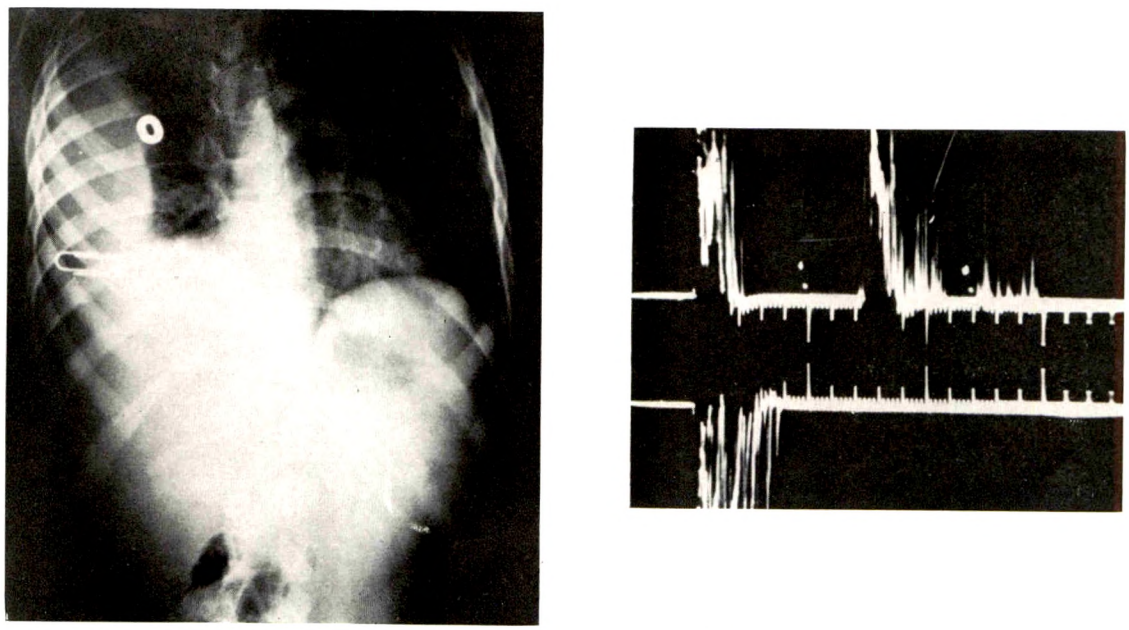


FIG. 8. *Pleural Effusion.* Roentgenogram reveals pleural density in right hemithorax. A cystic ultrasonic pattern is evident in the upper ultrasonic tracing. The lower (inverted) ultrasonic tracing demonstrates disappearance of the echo-free zone confirming the presence of free pleural fluid.

CONCLUSIONS

It is generally agreed that prompt diagnosis and treatment are essential in infants and children who have mass lesions of any kind. The addition of an ultrasonic examination in such patients has proved to be of diagnostic value. Ultrasonic evaluation requires no preliminary preparation; it is safe, rapid, and requires no compression or palpation of the mass. Ultrasound, of course, does not establish the pathologic characteristics of a mass lesion, although it does provide information as to the depth and size of the mass as well as its characteristics; *i.e.*, cystic, solid, or a combination of the 2. The latter is of some significance as cystic masses are likely to be benign, solid masses are apt to be malignant, and complex masses may be either benign or malignant. The examination is also helpful in demonstrating the presence of free fluid in the pleural or peritoneal cavity.

SUMMARY

Ultrasound was utilized in the evaluation of masses encountered in 45 patients who ranged in age from the newborn period to 14 years.

After localization of a mass by palpation or roentgenographic examination, the ultrasonic transducer was placed on the skin over it. At the proper instrument settings, fluid-filled masses produced no echoes, whereas solid masses produced multiple echoes.

Using this technique, it was possible to differentiate between cystic and solid masses as well as complex ones (a combination of the 2) with an accuracy of 98 per cent.

The information supplied by ultrasonic examination when used in combination with clinical and other laboratory data proved to be of diagnostic value.

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REFERENCES

1. Goldberg, B. B., Goodman, G. A., and Clearfield, H. R. Evaluation of ascites by ultrasound. *Radiology*, 1970, *96*, 15–22.
2. Goldberg, B. B., and Lehman, J. S. Some observations on practical uses of A-mode ultrasound. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *107*, 198–205.
3. Goldberg, B. B., Ostrum, B. J., and Isard, H. J. Nephrosonography: ultrasound differentiation of renal masses. *Radiology*, 1968, *90*, 1113–1118.
4. Goldberg, B. B., and Pollack, H. M. Differentiation of renal masses using A-mode ultrasound. *J. Urol.*, 1971, *105*, 765–771.
5. Howry, D. H. Brief atlas of diagnostic ultrasonic radiologic results. *Radiol. Clin. North America*, 1965, *3*, 433–452.
6. Lister, J., and Levick, R. K. Errors in diagnosis in Wilms' tumor. *J. Pediat. Surg.*, 1966, *1*, 488–497.
7. Suruga, K., Hirai, Y., and Nagashima, K. Ultrasonic echo examination as aid in diagnosis of congenital bile duct lesions. *J. Pediat. Surg.*, 1969, *4*, 452–456.

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Seventy-third Annual Meeting: Washington-Hilton Hotel, Washington, D. C., October 3–6, 1972.

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Representative on the Board of Chancellors of the American College of Radiology: Juan A. del Regato.

Fifty-fifth Annual Meeting: Broadmoor Hotel, Colorado Springs, Colorado, April 22–26, 1973.

EDITORIAL

PEDIATRIC RADIOLOGISTS—QUO IBIMUS HINC?

THIS is the eleventh editorial on the subject of Pediatric Radiology which has appeared in the JOURNAL over the past 10 years—the second one during 1972. These editorials, most of which document the spectacular growth of this subspecialty over the past decade, are highly complimentary; and one gets the impression that the only possible way to attract attention in writing about it would be to declare that it is vastly overrated.

We jest! In the first place, we are not about to bite the hand that has fed us so generously, if not sumptuously, for the past 30 years. Secondly, and more importantly, there appears to be no sound justification for blanket criticism of a thriving branch of radiology which continues to generate enthusiastic support from all directions.

After all, there are close to 250 members of the 14 year old *Society for Pediatric Radiology* in the United States, and nearly 300 registrants participated in the recent Ninth Annual Meeting of the *European Society for Pediatric Radiology* in Paris. On a purely local level, no less than 9 University of Michigan physicians who, during the past year and a half, spent several weeks or months in the Radiology Division of C. S. Mott Children's Hospital are currently in full-scale training in pediatric radiology (5) or have taken positive steps to obtain such training (4). Two others are seriously considering such a move. All of this has taken place at an institution which has no fellowship training program of its own.

On the face of it, this appears to be an impressive, albeit fortuitous, recruiting program, aided and abetted by a new hospital and interesting case material. Although we do not know precisely what is happening along this line at other medical centers, there still appears to be a steadily increasing interest among young physicians to enter Pediatric Radiology. So it would seem appropriate that we should gird ourselves for more intensive efforts toward convincing these young people that the pediatric approach unquestionably is the way for them to go, opening up new fellowship training programs, as we do so.

There are, however, several good reasons why we should not go at full velocity in this direction until we have taken a long, hard look at some cold, sobering facts. First of all, there are no more than 48 Children's Hospitals in the United States and Canada with full-time pediatric radiologists. In these 48 hospitals there are, at most, 105 radiology positions, virtually all of which are occupied at the present writing. Of even greater significance is the fact that the average age of the Directors of these Radiology Departments is only 47. Understandably, the average age of Associate Staff Members is somewhat less. It is quite obvious that very few of this group will soon be rushing to take advantage of the improved Social Security benefits recently passed by Congress, so there is no pressing need for mass replacement of the incumbents.

What about full-time pediatric radiologists working in medical centers which do not have separate Children's Hospitals or those practicing in large General Hospitals? There are approximately 50 such individuals in the United States, and their average age is just below 40. It is clearly evident that, for some years to come, retirement attrition will be very low in this group also.

Furthermore, there is limited room for expansion in this category as long as pediatric radiologists refuse to share in the examination of adult patients.

And so we come to a possible answer to the question posed in the title of this editorial, namely: "Where do we go from here?" The suggested solution may sound heretical to some pediatric radiologists and utterly impractical to others, but it is not completely unrealistic; in fact, it has much to recommend it.

It has been said that pediatric radiologists supervise only 20 per cent of children's roentgenologic examinations, the remaining 80 per cent being done by general radiologists as well as by family physicians, orthopedists, and other nonradiologic private practitioners. Even if this group were willing to refer all of their child patients to existing pediatric radiologic facilities, it is just not feasible to do this in the United States, although some progress is being made in this direction through the blossoming programs of Community Medicine in which certain hospitals are designated as Pediatric Centers or as Obstetric and Gynecologic Centers for cities, counties, or even groups of counties. These programs are promising at the moment, but little more than that.

It appears that the largest underdeveloped resource for pediatric radiologists lies in the remaining medical schools, the several hundred large general hospitals, and the growing number of sizable private-practice groups which do not yet have a sufficiently large pediatric clientele to warrant a full-time radiologic specialist in the pediatric field. If young, well-trained pediatric radiologists will temporarily forget their pride and express a willingness to share, however lightly, the adult patient load, they will find themselves in great demand. The general radiologists in these groups must, of course, cooperate in seeing to it that the pediatric radiologists' hard-earned talents are not diluted to any significant degree during the transitional period leading to full-time pediatric activity. There absolutely must be adequate facilities, including superb equipment and the all-important technologists especially trained in the care of infants and children.

If such a cooperative program can be consummated, considerable inroads can be made into the 80 per cent of infants and children who at the present time are not receiving the ultimate radiologic care they deserve. At the same time those engaged in the training of pediatric radiologists may proceed in their activities with clear conscience and a sense of moral obligation fulfilled.

John F. Holt, M.D.

C. S. Mott Children's Hospital
University of Michigan
Medical Center
Ann Arbor, Michigan 48104

NEWS ITEMS

AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE

The American Association of Physicists in Medicine will hold its winter meeting at the Sheraton-Blackstone Hotel, Chicago, Illinois, November 30–December 1, 1972.

For further information please contact Mr. Martin Rozenfeld, Department of Radiology, University of Chicago, Chicago, Illinois 66037.

SYMPOSIUM ON POLYTOMOGRAPHY OF THE TEMPORAL BONE

The 6th Symposium on Polytomography of the Temporal Bone will be given under the auspices of The Wright Institute of Otology at the Community Hospital, Indianapolis, Indiana, on February 3 and 4, 1973.

The subjects covered are: Basic Anatomy of the Temporal Bone; and Technique of Polytomography of the Temporal Bone with demonstrations of normal tomograms.

Pathologic conditions revealed by polytomography, such as cholesteatoma, ossicular chain problems, otosclerosis, fractures, foreign bodies, tumors, and congenital anomalies will be shown on original tomograms and the clinical applications discussed.

The number of registrants is limited to 18.

Inquiries should be directed to: Clifford C. Taylor, M.D., The Wright Institute of Otology, Inc., Community Hospital of Indianapolis, Inc., 1500 North Ritter Avenue, Indianapolis, Indiana 46219.

THIRD ANNUAL ASPEN RADIOLOGY CONFERENCE

The Third Annual Aspen Radiology Conference will be held March 5–9, 1973 at the Aspen Institute for Humanistic Studies, Aspen, Colorado.

The Conference is designed for physicians and scientists interested in diagnostic radiology, nuclear medicine and radiation therapy and will explore the impact of clinical and technological advances on radiologic practice.

The topics for discussions will include advances in bone, chest, gastrointestinal, and neuroradiology involving a triradiologic approach. Each day will survey the advances in a single radiology subdivision with independent diagnostic, nuclear medicine and therapy sessions. Small group discussions and refresher courses on these topics in each of the radiologic subdivisions will be held in the afternoons.

The Conference Faculty includes: Herbert Abrams, M.D., Harvard University; Bengt Bjarngard, Ph.D., Harvard University; Frederick Bonte, M.D., University of Texas; Donald Brown, M.D., University of Colorado; Robert Shalek, Ph.D., M. D. Anderson Hospital; Marvin Daves, M.D., University of Colorado; Alexander Gottschalk, M.D., University of Chicago; C. J. Karzmark, Ph.D., Stanford University; Bowen Keller, Ph.D., University of Rochester; Merle K. Loken, M.D., University of Minnesota; William Martel, M.D., University of Michigan; Harry Mellins, M.D., Harvard University; Mary Lou Meurk, B.A., Mount Zion Hospital, San Francisco; Thomas H. Newton, M.D., University of California; Theodore Phillips, M.D., University of California; James Quinn, III, M.D., Northwestern University; Philip Rubin, M.D., University of Rochester, New York; and James Youker, M.D., Medical College of Wisconsin.

Further information may be obtained from Maurice O'Connor, M.D., Conference Director, Division of Radiology, Denver General Hospital, Denver, Colorado 80204.

SIXTEENTH ANNUAL POSTGRADUATE COURSE IN DIAGNOSTIC RADIOLOGY

The Department of Radiology and Continuing Education in Health Sciences of the University of California, San Francisco,

announces the Sixteenth Annual Postgraduate Course in Diagnostic Radiology which will be held at the Fairmont Hotel, San Francisco, March 12–16, 1973.

This Course is designed to provide a survey of selected subdivisions of diagnostic radiology for the radiologist in clinical practice. The faculty of this Course is comprised of the Faculty of the Department of Radiology, University of California School of Medicine, San Francisco and an international Guest Faculty.

The Guest Faculty includes: Wylie J. Dodds, M.D., Milwaukee, Wisconsin; Kent Ellis, M.D., New York, New York; Melvin M. Figley, M.D., Seattle, Washington; Robert G. Fraser, M.D., Montreal, Canada; John A. Kirkpatrick, Jr., M.D., Philadelphia, Pennsylvania; George R. Leopold, M.D., San Diego, California; William Martel, M.D., Ann Arbor, Michigan; Harry Z. Mellins, M.D., Boston, Massachusetts; Leo G. Rigler, M.D., Los Angeles, California; Professor Eric Samuel, Edinburgh, Scotland; Edward B. Singleton, M.D., Houston, Texas; David M. Witten, M.D., Birmingham, Alabama; and William B. Young, M.D., London, England.

AN INTRODUCTION TO DIAGNOSTIC ULTRASOUND

Columbia University College of Physicians and Surgeons presents a postgraduate medicine course titled, "An Introduction to Diagnostic Ultrasound," April 12 and 13, 1973 under the direction of Donald L. King, M.D., of the Department of Radiology.

Presentations will include discussion of the physics, technology and instrumentation, and applications of pulse-echo ultrasound, with emphasis on echoencephalography, echocardiography and ultrasonography.

The presentations on ultrasonic imaging applications will include discussion of imaging renal and other abdominal masses and organs, a discussion of imaging in obstetrics and gynecology and discussion of a new application, ultrasonic imaging of the heart.

For further information and application forms, please write to Melvin D. Yahr, M.D., Associate Dean, College of Physicians and Surgeons, Columbia University, 630 West 168th Street, New York, New York 10032.

THIRD INTERNATIONAL CONGRESS OF THE INTERNATIONAL RADIATION PROTECTION ASSOCIATION (IRPA)

This Congress will be held in the Washington-Hilton Hotel, Washington, D.C., September 9–14, 1973.

The IRPA was formed in 1966 to promote international contacts and cooperation among those engaged in health physics and radiation protection work; to provide for discussion of the scientific and practical aspects of the protection of man and his environment from ionizing radiations; to serve as an international clearing house of scientific activities in radiation protection; and thereby to facilitate the utilization of radiation and nuclear energy for the benefit of mankind.

IRPA accepts as members individuals who are members of affiliated national or regional societies. It does not infringe upon the autonomy of the affiliated society, which is free to function effectively in radiation protection activities of local, national, or regional interest. Approximately, 6,300 members in some 60 countries, representing 21 professional societies are currently affiliated with IRPA.

The Third International Congress is hosted by the Health Physics Society of the United States of America.

The Scientific Sessions and the Exhibition of the Congress will cover all aspects of radiologic protection from both ionizing and non-ionizing radiations. The details of the sessions will depend upon the papers accepted.

A modified rapporteur system of presentation is planned for the great majority of papers accepted, combined with panels, symposia, and discussion groups in which the authors are expected to play a very

important role in contributing to lively discussion. It is anticipated that relatively few papers will be presented orally by their authors. All accepted papers, however, will be published in their full form in the Proceedings of the Congress.

Abstracts must be submitted before January 15, 1973.

For further information please contact Mr. John C. Villforth, Secretary, Bureau of Radiological Health, FDA, 5600 Fishers Lane, Rockville, Maryland 20852, USA.

FIFTH INTERNATIONAL CONGRESS OF RADIATION RESEARCH

The Fifth International Congress of Radiation Research will be held in Seattle, Washington, USA, July 14–20, 1974.

A brochure has recently been mailed to a large list of radiation research workers.

If you have not received a brochure and are interested in obtaining details of this meeting, please write to: Dr. W. K. Sinclair, Secretary-General, Argonne National Laboratory, Argonne, Illinois 60439, USA.

BOOK REVIEW

Progress in Pediatric Radiology. Volume 3: Genito-urinary Tract. Edited by H. K. Kaufmann, Basel. Cloth. Pp. 380, with 209 figures. Price, $29.00. Year Book Medical Publishers, 35 East Wacker Drive, Chicago, Ill. 60601, 1970.

The editor of this book has accomplished a remarkable task in presenting a true look at the progress in pediatric radiology, as the title indicates. He has been able to collect the experience and opinions of many well known authors and incorporate them in a book which serves a much needed gap in this field. All topics discussed are unusually well covered but several deserve special mention because the information is quite pertinent.

Pediatric radiographic technique is an area of considerable difference of opinion among Radiologists and Technicians. The discussions presented in the book represent those techniques which men of experience have found most useful in their practice. The chapter on Radiation Exposure due to Urinary Tract Disease is especially valuable. It gives a very vivid description of the comparative radiation dose for all methods of recording of the radiographic image in use today and again emphasizes the extreme low dose required in video tape recording.

The reader will also find interesting reading and illustrations on arteriography, inferior venacavagraphy, intersex, congenital anomalies, and the intra-uterine fetus, just to mention a few areas covered.

James F. Martin, M.D.

BOOKS RECEIVED

Evaluation of Renal Function and Disease with Radionuclides. Editor, M. Donald Blaufox, Albert Einstein College of Medicine, Bronx, N. Y. Volume 2 of Progress in Nuclear Medicine. Cloth. Pp. 315, with some figures. Price, $24.50. University Park Press, Chamber of Commerce Building, Baltimore, Md. 21202, 1972.

The Upper Cervical Spine: Regional Anatomy, Pathology and Traumatology; a Systematic Radiological Atlas and Textbook. By Detlef von Torklus and Walter Gehle, Orthopaedic Clinic and Outpatient Department, University Hospital, Hamburg. Translated by L. S. Michaelis. Cloth. Pp. 101, with many illustrations. Price, $15.00. Grune & Stratton, Inc., 111 Fifth Avenue, New York, N. Y. 10003, 1972.

Commissariat à l'Énergie Atomique. Rapport annuel 1971. Paper. Pp. 183, with many illustrations. Commissariat à l'Énergie Atomique, 29–33 Rue de la Fédération, Paris XV, France, 1972.

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SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

United States of America

American Roentgen Ray Society
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga. 30322. Annual Meeting: Washington-Hilton Hotel, Washington, D. C., October 3–6, 1972.

American Radium Society
Secretary, Dr. Felix N. Rutledge, M. D. Anderson Hospital and Tumor Institute, Houston, Texas 77025. Annual Meeting: Broadmoor Hotel, Colorado Springs, Colorado, April 22–26, 1973.

Radiological Society of North America
Secretary, Dr. Hillier L. Baker, Jr., Mayo Clinic, Rochester, Minn. 55901. Annual Meeting: Palmer House, Chicago, Ill., November 26–December 1, 1972.

American College of Radiology
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago, Ill. 60606. Annual meeting: San Francisco, Calif., April 2–7, 1973.

Section on Radiology, American Medical Association
Secretary, Dr. Antolin Raventos, Davis, Calif. Annual Meeting: New York, N. Y., June 24–28, 1973.

American Board of Radiology
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.

Oral examinations will be held in the following cities during the next 2 years: Atlanta, Ga., Dec. 4–8, 1972, Sheraton-Biltmore Hotel; Chicago, Ill., June 4–8, 1973, Marriott Motor Hotel; and Dallas, Tex., Dec. 3–7, 1973, Statler Hilton Hotel.

Written examinations are scheduled in 13 large centers.

Applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.

Deadline for filing applications for any examination in 1973 was September 30, 1972.

American Board of Nuclear Medicine, Inc. A Conjoint Board of the American Boards of Internal Medicine, Pathology and Radiology and sponsored by the Society of Nuclear Medicine.
Chairman, Dr. Merrill A. Bender, Suite 2402, 211 E. 43rd St., New York, N. Y. 10017.

Examination for certification in the Specialty of Nuclear Medicine will be given April 7, 1973. The deadline for filing application forms is January 1, 1973.

American Association of Physicists in Medicine
Secretary, D. John Wright, Ph.D., Department of Radiology, Temple Univ. Health Sciences Center, Philadelphia, Pa. 19140. Annual Meeting in Chicago, Ill., Sheraton-Blackstone Hotel, Nov. 30 and Dec. 1, 1972.

American Society of Therapeutic Radiologists
Secretary, Dr. Herman D. Suit, Radiation Medicine, Massachusetts General Hospital, Boston, Mass. 02114. Annual Meeting: Biltmore Hotel, Phoenix, Ariz., November 1–4, 1972.

American Institute of Ultrasound in Medicine
Secretary, John M. Reid, Ph.D., 556 18th Ave., Seattle, Wash. 98122. Annual Meeting.

American Society of Neuroradiology
Secretary, Dr. David O. Davis, Department of Radiology, 901 Twenty-third St., N. W., Washington, D. C. 20037. Annual Meeting, Boston, Mass., April 26–28, 1973.

Thirteenth International Congress of Radiology
President-Elect, Dr. Juan Gómez López. *Secretary General*, Dr. José Bonmatí, Lagasca, 27 Madrid 1, España. Meeting: Madrid, Spain, Oct. 15–20, 1973.

International Society of Radiology
Hon. Secretary-Treasurer, Prof. Eric Samuel, Radiologist-in-Charge, The Royal Infirmary, Edinburgh EH3 9Y-W, Scotland.

Eleventh Inter-American Congress of Radiology
Counselor for the United States, Dr. Juan A. del Regato, Penrose Cancer Hospital, 2215 North Cascade Ave., Colorado Springs, Colorado 80907. Meeting: Bogotá, Colombia in 1975.

Inter-American College of Radiology
President, Dr. Miguel Dao y Dao, Apartado Postal 14213, Candelaria, Caracas, Venezuela, S.A.
Secretary General, Dr. Rubén Merenfeld, Instituto Diagnóstico, Apartado Postal 14213, Candelaria, Caracas, Venezuela, S.A. Meeting: Bogotá, Colombia in 1975.

Third Congress of the European Association of Radiology
Meeting in June, 1975 in Edinburgh, Scotland.

Second Asian and Oceanian Congress of Radiology
Honorary Secretary, Dr. J. J. Martin, Box 805 F., G.P.O., Melbourne, 3001, Australia.
Meeting in 1975.

Alabama Chapter of ACR
Secretary-Treasurer, Dr. Lowry R. Young, Jr., 905 Madison St., Huntsville, Ala. 35801. Meets time and place of Alabama State Medical Association.

Alaska Radiological Society
Secretary, Dr. Bruce C. Wright, Providence Hospital, Anchorage, Alaska. Meets third Wednesday each month.

American Thermographic Society
Secretary-Treasurer, Dr. Marc. S. Lapayowker, Department of Radiology, Temple Univ. Hosp., 3401 N. St., Philadelphia, Pa. 19140. Meets at New York-Hilton Hotel, June 23 and 24, 1973.

Arizona Radiological Society, Chapter of ACR
Secretary, Dr. Wesley S. Fee, 2421 E. 6th St., Tucson, Ariz. 85719. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

Ark-La-Tex Radiological Society
Secretary, Dr. Erich K. Lang, Confederate Memorial Medical Center, LSU School of Medicine, Shreveport, La. 71101.

Arkansas Chapter of ACR
Secretary-Treasurer, Dr. David H. Newbern, 4301 W. Markham, Little Rock, Ark. 72205. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

Association of University Radiologists
Secretary-Treasurer, Dr. Melvyn H. Schreiber, 2909 Dominique Dr., Galveston, Tex. Annual Meeting, Vancouver, B. C., Canada, May 9–12, 1973.

Atlanta Radiological Society
Secretary-Treasurer, Dr. Ernst Ruder, Dept., of Radiol., Crawford Long Memorial Hosp., Atlanta, Ga. 30308. Meets on four Thursday evenings during the academic year at a time announced in early September of each year, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

Bavarian-American Radiologic Society
Secretary, Lucien F. Burke, Maj. MC, Radiol. Service, US Army Gen. Hosp., Frankfurt, APO, New York 09757.

Blockley Radiological Society
Secretary-Treasurer, Dr. R. John Gould, 41 Lombardy Rd., Drexel Hill, Pa. 19026.

Bluegrass Radiological Society
Secretary-Treasurer, Dr. Eugene Slusher, Lexington

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

Clinic, 1221 S. Broadway, Lexington, Ky. 40504. The Society meets once each month during the school year.

BRONX RADIOLOGICAL SOCIETY, NEW YORK STATE, CHAPTER ACR
Secretary-Treasurer, Dr. Leon J. Corbin, 1369 Rosedale Ave., Bronx, N. Y. 10472. Meets 4 times a year.

BROOKLYN RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Sarah Lieberson, 1298 St. Marks Ave., Brooklyn, N. Y. 11213. Meets first Thursday of each month, October through June.

BUFFALO RADIOLOGICAL SOCIETY
Secretary, Dr. Ru-Kan Lin, 191 North St., Buffalo, N. Y. 14201. Meets second Monday evening each month, October to May inclusive, at University Club.

CALIFORNIA RADIATION THERAPY ASSOCIATION
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets semiannually in San Francisco and Los Angeles.

CALIFORNIA RADIOLOGICAL SOCIETY, CALIFORNIA CHAPTER OF ACR
Secretary-Treasurer, Dr. John L. Gwinn, 4650 Sunset Blvd., Los Angeles, Calif. 90027.

CATAWBA VALLEY RADIOLOGICAL SOCIETY
Secretary, Dr. Emmett R. White, P.O. Box 10, Rutherford College, N. C. 28671. Meets every Thursday, Dept. of Radiology, Valdese General Hosp., Valdese, N. C., at 12:30 P.M.

CENTRAL NEW YORK RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. John Sanborn, Radiology Department, Crouse-Irving Memorial Hosp., Irving Ave., Syracuse, N. Y. 13210. Meets first Monday each month October through May.

CENTRAL OHIO RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Terry Meyer, 175 S. Merkle Rd., Columbus, Ohio 43209. Meets second Thursday in October, November, January, and March 15, and May 19 at Fort Hayes Hotel, Columbus, Ohio.

CHICAGO RADIOLOGICAL SOCIETY, DIVISION OF THE ILLINOIS RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Raymond L. Del Fava, 355 Ridge Ave., Evanston, Ill. 60201. Meets third Thursday of each month, October to April, except December, at the Bismarck Hotel, Chicago, Ill.

CLEVELAND RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Daniel E. Wertman, 11311 Shaker Blvd., Cleveland, Ohio 44104. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and April.

COLORADO RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Jerome D. Sutherland, 3705 E. Colfax Ave., Denver, Colo. 80206. Meets third Friday of each month at Denver Athletic Club from September through May.

CONNECTICUT VALLEY RADIOLOGIC SOCIETY
Secretary, Dr. Carl W. Scheer, 335 Cook Ave., Meriden, Conn. 06450. Meets in April and October.

DALLAS-FORT WORTH RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Norman L. Presley, 222 Medical Tower, 1550 W. Rosedale, Fort Worth, Tex. 76104. Meets the 3rd Monday of every month at 6:30 P.M., at the Cibola Inn, Arlington, Tex.

DELAWARE CHAPTER OF ACR
Secretary, Dr. Seymour R. Kaplan, Kent Gen. Hosp., Dover, Del. 19901.

EAST BAY RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Peter E. Kane, Children's Hosp. Med. Ctr., Oakland, Calif. 94609. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

EAST TENNESSEE RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. George H. Wood, 205 Medical Arts Bldg., Knoxville, Tenn. 37902. Meets in January and September.

FLORIDA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Ralph C. Aye, Tampa General Hospital, Tampa, Fla. 33606. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

FLORIDA WEST COAST RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. J. C. Hewitt, 1 Davis Blvd., Tampa, Fla. 33606. Meets in January, March, May, July, September and November.

GEORGIA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Richard S. Colvin, Emory University Clinic, Atlanta, Ga. 30322. Meets in spring and fall at Annual State Society Meeting.

GREATER CINCINNATI RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

GREATER LOUISVILLE RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

GREATER MIAMI RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Stanley I. Worton, 1400 N.W. 10th Avenue, Miami, Florida 33136. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

GREATER ST. LOUIS SOCIETY OF RADIOLOGISTS
Secretary-Treasurer, John J. Lang, 507 Fairways Circle, St. Louis, Mo. 63141.

HAWAII RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Ghim L. Yeoh, 1481 King St., Ste. 202, Honolulu, Hawaii 96814. Meets third Monday of each month at 7:30 P.M.

HEALTH PHYSICS SOCIETY
Secretary, John H. Pingel, Argonne National Laboratory, 9700 S. Cass Ave., Argonne, Ill. 60439. Annual Meeting: Deauville Hotel, Miami Beach, Fla., June 17–21, 1973.

HOUSTON RADIOLOGICAL SOCIETY
Secretary, Dr. Neill B. Longley, 6436 Fannin, Suite 601, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025.

IDAHO STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Hugh P. Smith, Jr., 130 E. Bannock, Boise, Id. 83702. Meets in the spring and fall.

ILLINOIS RADIOLOGICAL SOCIETY, INC., CHAPTER OF ACR
Secretary, Dr. B. Jay Hill, Mercy Hospital, Stevenson at King Drive, Chicago, Ill. 60616. Meets in the spring and fall.

INDIANA ROENTGEN SOCIETY, INC., CHAPTER OF ACR
Secretary, Dr. L. Ray Stewart, 852 S. Alvord Blvd., Evansville, Ind. 47714.

IOWA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. John Huston Jr., 1948 First Ave. N.E., Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

KANSAS RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Ralph H. Baehr, 310 Medical Arts Bldg., Topeka, Kan. 66604. Meets in spring with State Medical Society and in winter on call.

KENTUCKY CHAPTER OF ACR
Secretary-Treasurer, Dr. Joseph G. Whelan, Jr., 1313 St. Antony Place, Louisville, Ky. 40204. Meets in April and September.

KINGS COUNTY RADIOLOGICAL SOCIETY
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N.Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

KNOXVILLE RADIOLOGICAL SOCIETY
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

LONG ISLAND RADIOLOGICAL SOCIETY
Secretary, Dr. Jerome Zwanger, 1200 Stewart Ave., Garden City, N. Y. 11533. Meets monthly.

Los Angeles Radiological Society
Secretary, Dr. Gabriel H. Wilson, UCLA Center for Medical Sciences, Los Angeles, Calif. 90024. Meets second Wednesday of month in September, November, Janary, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif. Midwinter Radiological Conference, Century Plaza Hotel, Los Angeles, Calif. 90067, Feb. 2–4, 1973.

Louisiana Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Robert T. Cook, Southern Baptist Hosp., 2700 Napoleon Ave., New Orleans, La. 70115.

Louisiana-Texas Gulf Coast Radiological Society
Secretary-Treasurer, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Tex. 77701.

Maine Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Richard W. Taylor, Radiology Department, St. Mary's General Hospital, Lewiston, Maine 04240. Meets in June, September, December and April.

Maryland Radiological Society, Chapter of ACR
Secretary, Dr. George Burke, 4000 North Charles St., Baltimore, Md. 21218.

Massachusetts Radiological Society, Chapter of ACR
Secretary, Dr. Norman L. Sadowsky, The Faulkner Hosp., 1153 Centre St., Jamaica Plain, Mass. 02130.

Memphis Roentgen Society
Secretary-Treasurer, Dr. Jerry C. Phillips, The University of Tennessee College of Medicine, Department of Radiology, Walter C. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38103. Meets first Monday Oct. through May at the University Club.

Miami Valley Radiological Society
Secretary, Dr. Darwood B. Hance, Reid Memorial Hospital, Richmond, Ind. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

Michigan Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. George A. Kling, Harper Hospital, 3825 Brush St., Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

Mid-Hudson Radiological Society
Secretary-Treasurer, Dr. Herbert S. Berlin, Hopewell Junction, N. Y. Meets 7:00 P.M., first Wednesday of each month, September to May.

Milwaukee Roentgen Ray Society
Secretary-Treasurer, Dr. Thomas C. Lipscomb, 1004 North Tenth St., Milwaukee, Wis. 53233. Meets monthly on fourth Monday, October through May, at University Club.

Minnesota Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Harlan W. Hawkinson, 6 Buffalo Rd., St. Paul, Minn. 55110. Meets twice annually, fall and winter.

Mississippi State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Nancy W. Burrow, 1327 Peachtree St., Jackson, Miss. 39202. Meets third Thursday of each month at the Heidelberg Hotel, Jackson, at 6:00 P.M.

Missouri Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ronald G. Evens, Mallinckrodt Institute of Radiology, 510 S. Kingshighway, St. Louis, Mo. 63110.

Montana Radiological Society, Chapter of ACR
Secretary, Dr. Dexter W. Johnson, Columbus Hosp., Great Falls, Mont. 59401.

Nebraska Chapter of ACR
Secretary-Treasurer, Dr. John T. McGreer, III, 924 Sharp Bldg., Lincoln, Nebr. 68508. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

Nevada Radiological Society, Chapter of ACR
Secretary, Dr. William G. Arbonies, 887 Marsh, Reno, Nev. 89520.

New England Roentgen Ray Society
Secretary, Dr. Stefan C. Schatzki, 1180 Beacon St. Brookline Mass. 02146. Meets the third Friday of each month, October through April, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 4:30 P.M.

New Hampshire Roentgen Ray Society, Chapter of ACR
Secretary-Treasurer, Dr. Edward P. Kane, Claremont Gen. Hosp., Claremont, N. H. 03743. Meets four to six times yearly.

New Mexico Society of Radiologists, Chapter of ACR
Secretary, Dr. Carole R. Simmons, Dept. of Radiol., Presbyterian Hospital Center, Albuquerque, N. M. 87106. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

New Orleans Radiological Society, Inc.
Secretary-Treasurer, Dr. Jimmy L. Mains, P.O. Box 446, Gretna, La. 70053. Meets bimonthly at local restaurants selected by the President.

New York Roentgen Society
Secretary-Treasurer, Dr. Howard R. Gould, 153 West 11th Street, New York, N. Y. 10011. Meets monthly on the 3rd Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Conference, Waldorf-Astoria Hotel, New York, N. Y., Apr. 26–28, 1973. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

New York State Chapter of ACR
Secretary-Treasurer, Dr. John J. Magovern, 74 N. Village Ave., Rockville Centre, N. Y. 11570.

North Carolina Chapter of ACR
Secretary-Treasurer, Dr. James F. Martin, 300 S. Hawthorne Road, Winston-Salem, N. C. 27103.

North Dakota Radiological Society, Chapter of ACR
Secretary, Dr. Marshall Landa, 1702 13th St., So., Fargo, N. D. 58102. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

North Florida Radiological Society
Secretary, Dr. David F. Bew, University Hospital of Jacksonville, 655 W. Eighth Street, P.O. Box 2751, Jacksonville, Florida 32203. Meets quarterly in March, June, September and December.

Northeastern Oklahoma Radiological Society
Secretary-Treasurer, Dr. Theodore J. Brickner, Jr., 5919 S. Gary Place, Tulsa, Okla. 74110.

Northeastern New York Radiological Society
Secretary, Dr. Robert C. Friedman, 325 Presidential Way, Guilderland, N. Y. 12085. Meets in Albany area on third Wednesday of October, November, March, April, and May.

Northern California Radiation Therapy Association
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets quarterly.

Northern California Radiological Society
Secretary, Dr. Julian B. Holt, 3704 Mission Ave., Suite 26, Carmichael, Calif. 95608. Meets fourth Monday of Sept., Nov., Jan., March and May at Aldo's Restaurant in Sacramento.

Northwestern New York Radiological Society
Secretary, Dr. Barbara E. Chick, Glens Falls Hospital, Glens Falls, N. Y. 12801.

Northwestern Ohio Radiological Society
Secretary, Dr. Vito J. Zupa, Mercy Hospital, Department of Radiology, Toledo, Ohio.

Ohio State Radiological Society, Chapter of ACR
Secretary, Dr. Thomas F. Ulrich, Barberton Citizens Hospital, Barberton, Ohio 44203.

Oklahoma State Radiological Society, Chapter of ACR
Secretary, Dr. George H. Ladd, 4616 Denison, Muskogee, Okla. 74401. Meets in January, May and October.

Orange County Radiological Society
Secretary, Dr. Norman Arnett, 400 North Pamona, Fullerton, Calif. 92632. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at the Orange County Medical Association Bldg., Orange, Calif.

Oregon State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ronald W. Philips, 2305 Washington, Milwaukie, Ore. 97222. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary-Treasurer, Dr. Robert S. Miller, 13753 S.W. Farmington Rd., Beaverton, Oregon 97005. Annual meeting: Benson Hotel, Portland, Ore., May 11–13, 1973.

Pennsylvania Radiological Society, Chapter of ACR
Secretary, Dr. Theodore A. Tristan, Harrisburg Polyclinic Hosp., Harrisburg, Pa. 17105.

Philadelphia Roentgen Ray Society
Secretary, Dr. Howard M. Pollack, Department of Radiology, Episcopal Hospital, Philadelphia, Pa. 19125. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Stephen C. Bruno, Shadyside Hospital, 5230 Centre Ave., Pittsburgh, Pa. 15232. Meets second Wednesday of month, October through June, at Park Schenley Restaurant.

Radiation Research Society
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016. Annual Meeting: St. Louis, Mo., April 29–May 2, 1973.

Radiological Society of Connecticut, Inc., Chapter of ACR
Secretary-Treasurer, Dr. Carl W. Scheer, 335 Cook Ave., Meriden, Conn. 06450. Meetings are held quarterly.

Radiological Society of Greater Cincinnati
Secretary-Treasurer, Dr. Alvin Nathan, c/o Academy of Medicine, 320 Broadway, Cincinnati, Ohio 45202. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of Greater Kansas City
President-Secretary, Dr. Arch W. Templeton, Univ. of Kansas Med. Ctr., Kansas City, Kansas 66103. Meets 5 times a year on given dates.

Radiological Society of Louisiana, Chapter of ACR
Secretary-Treasurer, Dr. Robert T. Cook, 1820 Octavia St., New Orleans, La. 70115. Meets semiannually during Louisiana State Medical Society meeting and 6 months later.

Radiological Society of New Jersey, Chapter of ACR
Secretary, Dr. Sidney Ketyer, St. Elizabeth Hosp., 225 Williamson St., Elizabeth, N. J. 07207. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

Radiological Society of Rhode Island, Chapter of ACR
Secretary-Treasurer, Dr. James B. Leach, Jr., 115 Cass Ave., Woonsocket, R. I. 02895.

Radiological Society of Southern California
Secretary-Treasurer, Dr. Marshall Rowen, Dept. Radiol., St. Joseph Hosp., 1100 Stewart Dr., Orange, Calif. 92668. Meets three times a year, usually October, February and May.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

Redwood Empire Radiological Society
Secretary, Dr. Lee F. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. Donald P. King, 218 Gun Club Rd., Richmond, Va. 23221. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary-Treasurer, Dr. Kenneth E. Robinson, 1441 East Avenue, Rochester, New York 14610. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

Rocky Mountain Radiological Society
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual meeting: Brown Palace Hotel, Denver, Colo., Aug. 1973.

San Antonio-Civilian Military Radiological Society
Secretary, Dr. Robert M. Maurer, Santa Rosa Medical Center, San Antonio, Tex. 78207. Meets third Wednesday of each month at Fort Sam Houston Officers' Club at 6:30 P.M.

San Diego Radiological Society
President-Secretary, Dr. James M. Lee, Scripps Clinic and Research Found., 476 Prospect St., La Jolla, Calif. 92037. Meets first Wednesday of each month at the Town & Country Hotel.

San Francisco Radiological Society
Secretary-Treasurer, Dr. James G. Moore, Mary's Help Hospital, 1900 Sullivan Ave., Daly City, Calif. 94015.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
Secretary-Treasurer, Dr. Louis Wener, Cafritz Memorial Hosp., 1310 Southern Ave., S.E., Washington, D. C. 20032. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section on Radiology, Southern Medical Association
Secretary, Dr. Mario A. Calonje, 1514 Jefferson Highway, New Orleans, La. 70121. Annual Meeting, New Orleans, La., Nov. 13–16, 1972.

Section on Radiology, Texas Medical Association
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

Shreveport Radiological Club
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

Society for Pediatric Radiology
Secretary, Dr. John L. Gwinn, Children's Hospital, 4614 Sunset Blvd., Los Angeles, Calif. 90027. Annual meeting: Washington-Hilton Hotel, Washington, D. C., October 1–2, 1972.

Society of Gastrointestinal Radiologists
Secretary-Treasurer, Dr. Wm. B. Seaman, The Presbyterian Hospital, Columbia-Presbyterian Medical Center, 622 W. 168th St., New York, N. Y. 10032.

Society of Nuclear Medicine
Secretary, Dr. E. James Potchen, Edward Mallinckrodt Institute of Radiology, 660 S. Euclid Ave., St. Louis, Mo. 63110. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. Annual meeting.

South Bay Radiological Society
Secretary, Dr. A. D. Benjamin, 596 Carroll St., Sunnyvale, Calif. 94086. Meets the 2nd Monday of each month at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

South Carolina Radiological Society, Chapter of ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Coast Radiological Society
Secretary-Treasurer, Dr. James T. McClintock, P. O. Box 837, Santa Barbara, Calif. 93102. Meets fourth Monday of the month, Oct. through May.

South Dakota Radiological Society, Chapter of ACR
Secretary, Dr. M. F. Petereit, 2416 S. Phillips, Sioux Falls, S. D. 57105. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Society
Secretary-Treasurer, Dr. John Byfield, 1000 West Carson St., Torrance, Calif. 90502. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 7544, Mobile, Ala. 36607. Seventeenth Annual Meeting: Grand Hotel, Point Clear, Ala., Jan. 26–28, 1973.

Southwestern Radiological Society
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Carl T. Stubblefield, Bedford County Hospital, Columbia, Tenn. 38401. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas State Radiological Society, Chapter of ACR
Secretary, Dr. Herman C. Sehested, 815 Medical Tower, Room 100, 1550 W. Rosedale St., Fort Worth, Tex. 76104. Annual meeting.

The Fleischner Society
President, Dr. Robert G. Fraser, 1555 Summerhill Ave., Montreal 109, P.Q., Canada. Annual meeting.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John F. Stucki, Department of Radiology, McKay-Dee Hosp., 3939 Harrison Blvd., Ogden, Utah 84403. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society, Chapter of ACR
Secretary, Dr. J. Lorimer Holm, R.R.#3, Barre, Vt. 05641.

Virginia Chapter of ACR
Secretary-Treasurer, Dr. James S. Redmond, Suite 7, Medical Center, Lynchburg, Va. 24501. Next Meeting: Nov. 6, 1972 at the Conference Ctr., Williamsburg, Va.

Washington, D. C., Chapter of ACR
Secretary-Treasurer, Dr. Albert M. Zelna, 9110 Seven Locks Rd., Bethesda, Md. 20014.

Washington State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John N. Roehr, 1011 Medical Dental Bldg., Seattle, Wash. 98101.

West Virginia Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Andrew W. Goodwin, II, 200 Med. Arts Bldg., Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society; other meetings arranged by program committee.

Westchester County Radiological Society
Secretary, Dr. Max C. King, 218 Forest Ave., New Rochelle, N. Y. 10804. Meets on second Tuesday of October, December, February and May.

Wisconsin Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Marvin L. Hinke, 630 S. Central Ave., Marshfield, Wisc. 54449. Meets twice a year, May and September.

Wisconsin Society of Therapeutic Radiologists
Secretary, Dr. Robert W. Edland, Gundersen Clinic, LaCrosse, Wisc. 54601. Meets quarterly.

Wyoming Radiological Society, Chapter of ACR
Secretary, Dr. Ross J. Collie, Box 96, Lander, Wyo. 82520.
Meets in fall with State Medical Society and in spring on call of President.

Mexico, Puerto Rico and Central America

Asociación Costarricense de Radiología
Secretary, Dr. Jorge Vargas Segura, Apartado 5367, San José, Costa Rica.

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá
Secretary-General, Dr. Francisco Mirambell, Apartado 3352, San José, Costa Rica, Central America.
Meets annually in a rotating manner in the six countries.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad de Radiología de El Salvador
Secretary, Dr. Carlos Mejia, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

Sociedad Mexicana de Radiología, A.C.
Coahuila No. 35, México 7, D.F., México.
Secretary-General, Dr. Jorge Cano Coquí. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. Heriberto Pagán Sáez, Box 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
Moyne, 121 Boul. Taschereau, Greenfield Park, P.Q., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretary, Dr. T. Kerr, 32 Welbeck St., London, W1M/7PG, England. Meets monthly from October until May.

Canadian Association of Physicists, Division of Medical and Biological Physics
Honorary Secretary-Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada.

Canadian Association of Radiologists
Honorary Secretary, Dr. Champlain Charest, 1555 Summerhill Ave., Montreal 109, Que., Canada. Annual meeting: Montreal, Que., Canada, March 11–17, 1973.

Edmonton and District Radiological Society
Secretary, Dr. L. A. Koller, Suite 360, Professional Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada. Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

Faculty of Radiologists
Honorary Secretary, Robert Morrison, 47 Lincoln's Inn Fields, London, W.C.2, England. The scientific meetings held in January, February and March 1973 will be combined sessions with the British Institute of Radiology and the Section of Radiology of The Royal Society of Medicine.

Faculty of Radiologists, Royal College of Surgeons in Ireland
Hon. Secretary, Dr. D. F. Canwell, 123 St. Stephens Green, Dublin 2, Ireland.

Montreal Radiological Study Club
Secretary, Dr. W. Paul Butt, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 4:45 p.m. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1, England.

Société Canadienne-Française de Radiologie
Secretary General, Dr. Hubert Sasseville, Verdun Hospital, Montréal, P.Q., Canada. Meets every third Tuesday from October to April.

The Hospital Physicists' Association
Honorary Secretary, J. B. Massey, B.Sc., Christie Hosp. and Holt Radium Institute, Physics Department, Withington, Manchester M20 9BX, England.

Toronto Radiological Society
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September through May.

Royal College of Radiologists of Australasia
Honorary Secretary, Dr. T. P. Loneragan, 147 Macquarie St., Sydney, N.S.W. 2000, Australia.

South America

Colégio Brasileiro de Radiologia
Secretary-General, Dr. Miguel Mario Céntola, Caixa Postal 5984, São Paulo, Brazil.

Federación Argentina de Sociedades de Radiología
Branches of the Federation are: Sociedad de Radiólogos de la Provincia de Córdoba; Sociedad Argentina de Radiología; Asociación Argentina de Radiología; Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario; Sociedad de Radiología y Medicina Nuclear del Centro y Sudeste de la Provincia de Buenos Aires (Mar del Plata); Sociedad Salteña de Radiología y Medicina Nuclear; Sociedad de Radiología de Tucumán; Sociedad de Radiologia del Nordeste Argentino; Sociedad de Radiología de La Plata; and Sociedad de Radiología de San Juan.
Secretary-General-Treasurer, Dr. Roberto Habichayn, Av. Colón 637, Córdoba, Argentina.

Sociedad Argentina de Radiología
Secretary-General, Dr. Armando B. de Onaindia, Santa Fé 1171, Buenos Aires, Argentina. Meets first Wednesday evening, April through December.

Sociedad Boliviana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Armando Rocha Amoédo, Caixa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 p.m. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. Claudio Cortés, Casilla, 13426 Santiago, Chile. Meets first Friday of each month at Av. Santa María 1810 at 7:00 p.m.

Sociedad Colombiana de Radiologia
Secretary-General, Dr. Rodrigo Caro Molina, Cali, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología
Secretary, Dr. Carlos Palau Jimenez, Casilla 5469, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Hugo Volpe Rios, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiologia
Secretary-General, Dr. Miguel Chiappori Cambana, Av. General Santa Cruz No. 315, Miraflores, Lima, Perú, Casilla Correo, 2306, Lima, Perú. Meets monthly except during January, February, and March

Sociedad de Radiología del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

Sociedad de Radiología del Nordeste Argentino
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Sociedad de Radiología de La Plata
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Sociedad de Radiología, Cancerología y Física Médica del Uruguay
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Secretary, Dr. Manoel Medeiros Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

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Secretary-General, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Córdoba, Argentina.

Sociedad de Radiología, Radioterapéutica y Medicina Nuclear, de Rosario
Secretary-General, Dr. Federico Frágola, Santa Fe 1798, Rosario, Argentina.

Sociedad Salteña de Radiología y Medicina Nuclear
Secretary, Dr. Julio Luis Baldi, Av. Sarmiento 536, Salta, Argentina.

Sociedad Venezolana de Radiología y Medicina Nuclear
Secretary-General, Dr. Salvador Itriago Borgas, Apartado Postal 9213, Candelaria, Caracas, Venezuela. Meets monthly, third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Röntgen-Gesellschaft
Secretary, Dr. Ernst Kotcher, Wiener Allgemeine Poliklinik Mariannengasse 10, A-1090 Wien, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik. The 1973 Annual Meeting is a joint meeting with the Deutsche Röntgengesellschaft and will be held in Vienna, April 12–14, 1973.

Société Royale Belge de Radiologie
General Secretary, Dr. Joseph Baeyens, Alost, Belgium. Meets in February, March, May, June, September, October, November and December.

Société Européenne de Radiologie Pédiatrique
Secretary, Dr. P. Hicken, The Children's Hosp., Ladywood Middleway, Birmingham B16 8ET, Great Britain. Tenth Annual Meeting, Birmingham, April 11–13, 1973.
Permanent Secretary, Clément Fauré, Hôpital des Enfants Malades, 149, rue de Sèvres, 75 Paris 15e, France.

Société Française de Radiologie Médicale, Médecine Nucléaire et Electrologie, and its branches: Société du Sud-Ouest, du Littoral méditerranéen, du Centre et Lyonnais, du Nord, de l'Ouest, de l'Est, et d'Alger et du Massif Central. Parent Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru Paris 8e, France.

Société Française de Neuroradiologie
Secretary-General, Professeur agr. R. Djindjian 16, rue de l'Université 75, Paris 7e, France. Annual meeting.

Československá Radiologická Společnost
Secretary, Associate Professor Jaromír Kolář, M.D., Sc.D., Radiological Clinic, Praha 2, U nemocnice 2, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

Deutsche Röntgengesellschaft
President, Professor Dr. med. Walter Frommhold, Director of Medizinisches Strahleninstitut der Universität Tübingen, Röntgenweg 11, 7400 Tübingen, Germany.

The 1973 Annual Meeting is a joint meeting with the Österreichische Röntgengesellschaft and will be held in Vienna, April 12–14, 1973.

Società Italiana di Radiologia Medica e Medicina Nucleare
Administrative Secretary, Prof. E. Conte, Ospedale Mauriziano, 10128 Torino, Italy. Meets annually.

Societas Radiologica Danica
Secretary, Dr. Bent Langefeldt, Tretommervej 20a, DK-8240 Risskov, Denmark.

Nederlandse Vereniging voor Radiologie
Secretary, Dr. J. D. Mulder, Dept. of Rad., Akademisch Ziekenhuis, Leiden, Netherlands.

Scandinavian Radiological Society
Secretary-General, Professor C. E. Unnérus, M.D., Hagalung-Tapiola, Havsvindsvägen 5 C., Finland.

Sociedad Española de Radiología y Electrología Médicas y de Medicina Nuclear
Secretary, Dr. José Bonmatí, Villanueva, 11, Madrid 1, España. Meets every second Friday of each month, Oct. to June, inclusive, in Madrid.

Sociedade Portuguesa de Radiologia e Medicina Nuclear
Secretary-General, Dr. Luís Aires de Sousa, Av. Elias Garcia, 123- 1, Esg.-Lisboa i-Portugal.

Schweizerische Vereiningung für Radiologie, Nuklearmedizin und Strahlenbiologie (Association Suisse de Radiologie, Médecine Nucléaire et de Radiobiologie)
Secretary, Dr. med. Gustav Schoch, Bethesdaspital, Gellerstrasse 144, 4000 Basel, Switzerland.

Asia

Indian Radiological Association
Secretary, Dr. L. R. Parthasarathy, 44 Constable Road, Madras—23, India.

Indonesian Radiological Society
Secretary, Dr. Gani Iljas Sasmitaatmadja, Radiology Dep., Faculty of Medicine, University of Indonesia, Salemba 6, Djakarta, Indonesia.

Iranian Radiological Society
Secretary, Dr. Issa Yaghmai, P.O. Box No. 14-1151, Teheran, Iran. The Society meets on the second Saturday of each month.

Israel Radiological Society
President, Dr. I. Izkovitch, 2 Wingate Ave., Beit Harofe, Haifa, Israel.

Japanese American Radiological Society
Secretary, Thomas J. Imray, Maj. MC, Chief, Radiol. Dep., US Army Hosp., Camp Zana, Japan, APO San Francisco, Calif. 96343. Meets 3rd Thursday, monthly at various locations in Tokyo.

Philippine College of Radiology
Secretary-Treasurer, Dr. Edmundo V. Villacorta, Box 1284, Commercial Center, Makati, Rizal D-708, Philippines. Meets every 3 weeks on Fridays. Annual convention: Jan. 1973, Manila.

Radiological Society of Thailand
Secretary, Prof. Rojana Suvansuthi, M.D., Ph.D., Depart. of Rad., Sirizaj Hospital, Faculty of Medicine Mahidol University, Bangkok 7, Thailand.

Africa

Association of Radiologists of West Africa
Honorary Secretary, Dr. T. M. Kolawole, University College Hospital, University of Ibadan, Ibadan, Nigeria.

Radiological Society of South Africa
Secretary, Dr. B. Rosenberg, P.O. Box 3200, Johannesburg, South Africa.

South African International Radiological Congress
Director, Dr. Paul Sneider, P.O. Box 4878, Johannesburg, South Africa.

ABSTRACTS OF RADIOLOGIC LITERATURE

E. FREDERICK LANG, M.D., Editor

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ROENTGEN DIAGNOSIS

Neck and Chest

Bandt, Paul D., Blank, Norman, and Castellino, Ronald A. Needle diagnosis of pneumonitis: value in high-risk patients. *J.A.M.A.*, June, 1972, *220*, 1578–1580. (Address: R. A. Castellino, M.D., 300 Pasteur Drive, Palo Alto, Calif. 94305.)

Twenty-five needle biopsy aspirations are reported in this article. These were biopsies on 21 acutely ill patients who had lung infections. All were patients who had been immunologically compromised by active immunosuppressive drug treatment. They were cardiac or renal transplantation patients, or were those with malignant lymphoma and leukemia, or those on steroid therapy. These patients had evidence of infection with constitutional symptoms and fever, but the exact type of infection had not been proven prior to the needle biopsies.

The biopsies were performed percutaneously with fluoroscopic guidance. Expiratory posteroanterior and lateral roentgenograms of the chest were taken immediately and at intervals of 1 hour and 24 hours after the biopsy. The specimens were smeared and studied for bacteria and fungi, and were inoculated on culture media.

The biopsies were done with 18 gauge thin walled spinal needles except in 3 cases, where the Franklin modification of the Vim Silverman needle was used.

A positive diagnosis was made in 76 per cent of the biopsies. The only complication occurring in those patients in whom the 18 gauge spinal needle was used was a pneumothorax in 1 patient. Two of the patients who had the Vim Silverman cutting needle used for biopsy had complications. There was hemoptysis requiring transfusion in one, and another had pneumothorax requiring chest tube drainage; this latter patient died 10 days after the biopsy.

The authors point out the risk of using the cutting needle in the presence of thrombocytopenia; they believe that this should be an absolute contraindication to biopsy with a cutting needle.—*Richard E. Kinzer, M.D.*

Bledsoe, Francis H., and Seymour, E. Quitman. Acute pulmonary edema associated with parathion poisoning. *Radiology*, Apr., 1972, *103*, 53–56. (From: Department of Radiology, Medical University of South Carolina, Charleston, S. C.)

Exposure to parathion, an organic phosphate derivative, is a major cause of fatal poisoning throughout the world; more than 50 deaths were recorded in Florida during one 6 month period.

The authors present 3 case reports on teen-age male farm workers who were exposed to parathion in the course of their duties. They all developed symptoms of parathion poisoning within 6–24 hours of their exposure. Each patient was acutely ill and comatose when hospitalized; however, all responded to specific therapy and were discharged symptom-free on the 4–6th day after admission.

Parathion is rapidly absorbed through the skin, mucous membranes and from the respiratory and gastrointestinal tracts. As little as a single drop in the eye may be fatal.

The authors describe the clinical symptoms and give a brief discussion of the pathophysiology of parathion poisoning.

Pulmonary edema appears to be a frequent occurrence in parathion poisoning, and has been attributed to the muscarinic action of the drug. It is also pointed out that pulmonary edema may occur in the postictal state and it is interesting that all of the described patients did convulse in the initial stages of their clinical course. All of the patients exhibited some degree of clinical hypoxia and this has also been suggested as a possible contributing factor in the development of pulmonary edema, similar to that seen in heroin and barbiturate poisoning.

The roentgenographic characteristics appear to be those of an alveolar-filling process and seem to be confined mainly to the perihilar area. The heart is not enlarged in contrast to the usual findings in patients with pulmonary edema associated with myocardial disease. The roentgenographic changes of pulmonary edema associated with parathion toxicity resolved rapidly with appropriate therapy, frequently within hours.

The sudden onset of pulmonary edema without cardiomegaly should at least raise the possibility of parathion poisoning, especially in a young and previously well patient.—*E. Jerome Schoolar, M.D.*

Rösch, Josef, Judkins, Melvin P., Green, Gerald S., and Kidd, Harold. Aortocoronary venous bypass grafts: angiographic study of 84 cases. *Radiology*, March, 1972, *102*, 567–573. (Address: Josef Rösch, M.D., Department of Radiology, University of Oregon Medical School, 3181 S.W. Sam Jackson Park Road, Portland, Oregon 97201.)

Aortocoronary venous bypass autograft is the most recent advancement in the surgical treatment of coronary disease. A reversed segment of a saphenous vein is interposed between the aorta and the coronary artery bridging a stenotic or an occluded segment of the coronary artery and thus revascularizing ischemic myocardium. Coronary arteriography, a decisive technique for selecting patients for surgery, is also important for evaluating the results of a bypass procedure. This communication presents the authors' experience with follow-up coronary and bypass graft angiography and includes a discussion of some of its technical aspects.

During the period from April, 1969 to February, 1971, 84 patients were angiographically examined, who had aortocoronary vein bypass grafts done at 4 different Northwest hospitals. In all, 117 various graftings were performed. A single graft was placed in 52 patients, double bypass grafting was done in 31 patients, and triple bypass grafts were used in 1 patient. In 6 patients bypass grafting was complelemented by other cardiac surgery, including resection of left ventricular aneurysm, Vineburg procedure, and aortic valve replacement. All 84 patients underwent preoperative and follow-up angiography with follow-up studies done at intervals from 10 days to 20 months after surgery. All preoperative and follow-up studies were performed by the Judkins' percutaneous transfemoral technique using polyurethane catheters. In the follow-up studies, a pigtailed left ventricular catheter was usually introduced first, and, after the aortic and left ventricular pressures were recorded, left ventricular cineangiography was performed in the right anterior oblique position. Bilateral selective coronary arteriography was then done with preshaped catheters and, as the final step, the bypass grafts were visualized.

Complete studies, including left ventriculography and selective coronary and graft visualization, were done in 75 patients. In 8, including those who had aortic valve replacement, only selective coronary and graft angiograms were obtained. Of 117 grafts examined, 82 grafts in 64 patients were patent and functioning well. In properly working grafts, the graft size was proportional to that of the grafted artery, anastomoses were wide open and there was prompt, complete wash out of the injected contrast medium with good distal filling of the grafted artery and its branches. Nine grafts, although patent, functioned poorly and 26 grafts were considered occluded. The selective coronary angiograms led to different findings depending on graft function. In a patent, well functioning graft, the grafted artery either did not fill or filled faintly beyond the anastomosis due to good but nonopaque flow through the graft. In 14 patients, substantial progress of the coronary disease was found in the proximal segment of the grafted artery. Coronary angiograms in patients whose grafts were considered occluded usually showed no substantial change from the presurgical examination. Cineventriculography showed improved ventricular contractions and/or decreased left ventricular and diastolic pressure compared to preoperative studies in 36 of 64 patients with patent grafts. The average end-diastolic pressure in these patients fell from 19.6 mm. Hg before surgery to 12.7 mm. Hg after surgery. The rest of 28 patients with patent grafts did not show any substantial change nor did 15 patients with stenotic or occluded grafts. Only in 5 patients with occluded grafts was there a deterioration of the left ventricular function with decreased contractility and/or increased end-diastolic pressure.

The objectives of this follow-up study were evaluation of the bypass grafts, their contribution to revascularization of ischemic myocardium, and their effects on coronary arteries. Selective catheterization and visualization of individual grafts using both cine and rapid serial film angiography gave the best information about the anatomy of the grafts and their function. For the graft catheterization, the transfemoral percutaneous approach using polyurethane Ducor catheters has been very effective in the authors' experience with the ability to selectively catheterize 88 to 91 patent grafts (96.7 per cent).

The key to selective graft catheterization is a properly shaped catheter which must correspond to the anatomy of the graft and its aortic anastomosis. The right sided graft is usually connected to the lower portion of the ascending aorta 2–4 cm. above the origin of the right coronary artery. It takes an almost straight caudad course, regardless whether it goes to the main right or posterior descending artery. For its selective catheterization, a modified head-hunter catheter (with straightened tip) has been most effective in their experience. The left sided graft is usually attached higher than the right graft and more laterally on the anterolateral part of the aorta in an anterolateral direction and forms a reversed C-shaped curve around the pulmonary artery. Because of the sharp angle between the left graft and aorta, catheters with a curved tip, such as the right coronary or head-hunter catheter, are the best choice. To minimize the need for catheter exchange, catheterization of left grafts was attempted with the right coronary catheter following its use in the right coronary artery. For safety during selective graft catheterization, and catheterization of the coronary arteries, catheter tip pressure is monitored constantly and injection is done only with the catheter in a free position. Should it be impossible to selectively catheterize the graft, useful information can still be obtained from a midstream injection into the ascending aorta.

Selective visualization of coronary arteries is an indispensable part of the follow-up study of patients with aortocoronary bypass grafts. It not only helps to evaluate the basic disease and its progress, but gives information about graft function and offers surgeons a good means for evaluation of their operative technique on these small vessels. Good graft function can be expected to result in a lack of filling or a faint filling of grafted artery and absence of the previously shown collaterals. The examination of left ventricular function is an important part of the follow-up study and demonstrates the effect of the grafts on the myocardium.

The authors emphasize that repeat angiography as a follow-up to aortocoronary venous bypass grafting constitutes an important extension to preoperative study. Follow-up studies are a little more complex than the preoperative angiography, but procedure

time is only minimally extended and the patients have tolerated it well.

In this reported series, there were no local or cardiac complications.—*Edward B. Best, M.D.*

Abdomen

Brown, Alastair R. Non-gangrenous ischaemic colitis: a review of 17 cases. *Brit. J. Surg.*, June, 1972, *59*, 463–473. (From: Department of Surgery, Sydney Hospital, Sydney, Australia.)

Ischemic colitis usually occurs after abdominal aortic surgery or as a terminal event in cardiac disease. Rarely, it occurs spontaneously.

The radiologic features are: "thumb printing" in the acute stage; tubular narrowing of the affected segment, usually clearly demarcated; rapid change to either normal appearing colon or a segmental stricture, in serial barium enemas; shortening of the affected segment, even after stricture; and the common occurrence of the lesion in the region of the splenic flexure. Clinically, there are severe abdominal pain, diarrhea, rectal bleeding, and an acute onset. In nongangrenous ischemic colitis, there is a remarkable paucity of physical signs, mild systemic disturbance, and self limited disease. In gangrenous ischemic colitis, signs of peritonitis and shock dominate. The author confines his investigation to nongangrenous colitis.

The severity of the symptoms often bears no relation to the severity and the extent of colonic damage as shown on the roentgenogram. The tendency for the radiologic changes to be reversible emphasizes the need for an early barium enema examination when ischemic colitis is suspected.

Seventeen patients are reported, and in 7, the initial diagnosis was ischemic colitis. In the others, ulcerative colitis, granulomatous colitis, carcinoma and obstruction were considered first. In some, the diagnosis was confirmed by histologic examination of either a resected specimen or, in a low lesion, a sigmoidoscopic biopsy specimen. In 15 patients normal colonic function returned; 1 patient died postoperatively; and in the other patients, mild constipation developed, probably as a result of the ischemia.

Radiologically, it was not possible to predict which patient would develop stricture and which would clear completely. Nine patients had repeat barium enema studies, and in 3 there was permanent colonic deformity, while there was no residual change in the other 6.

In 12 of the 17 patients, the splenic flexure was included in the region of involvement; in 2 of the others, the abnormal segment was in the left half of the transverse colon; while in the other 3, the abnormal segment was in the descending or sigmoid colon below the splenic flexure. Sixteen of the lesions occurred spontaneously and 1 followed anterior resection of the rectum.—*E. Frederick Lang, M.D.*

MacIver, A. G., and Whitehead, R. Zonal colonic aganglionosis, a variant of Hirschsprung's disease. *Arch. Dis. Childhood*, April, 1972, *47*, 233–237. (From: Department of Pathology, Radcliffe Infirmary, Oxford, England.)

Four of 15 cases of Hirschsprung's disease of the colon treated surgically showed an atypical pattern of aganglionosis, suggesting that this variant of the disease may not be as uncommon as previously suspected.

There was aganglionosis of both intermyenteric and submucous plexuses in the resected segments. In all cases there was a zone of aganglionosis in the sigmoid with normal ganglion cells present proximally and distally. In 1 of these cases there was a double zone of aganglionosis with the distal area extending to the anus and with normal ganglion cells between the 2 zones.

The clinical course of acute intestinal obstruction in 3 cases and of recurrent constipation in 1 case was common with that of typical Hirschsprung's disease.

The authors believe that zonal aganglionosis supports a theory that migration of neuroblasts or their subsequent development can proceed following local arrest and that development occurs in a craniocaudal direction from the neural crest rather than caudocranially.

Zonal aganglionosis may present as typical Hirschsprung's disease, but rectal biopsy may be normal. The possibility of a second area of aganglionosis is cases which respond poorly after resection must be kept in mind.—*Paul M. Dassel, M.D.*

Grundy, D. J., King, P. A., and Lloyd G. Comparative evaluation of preoperative and operative radiology in biliary-tract disease. *Brit. J. Surg.*, March, 1972, *59*, 205–208. (From: Chelsea and Kensington Group, St. Mary Abbots Hospital, London, England.)

One hundred consecutive patients with abnormal cholecystograms (3 gm. telepaque, repeated the following day if necessary) were studied further by the 4 day telepaque test, intravenous cholangiography, and operative cholangiography. T-tube cholangiography was done on the tenth to twelfth postoperative day.

Operative cholangiography was the most accurate study in the diagnosis of a stone in the common duct, being positive in 20 of 21 cases. This study is recommended in all patients undergoing cholecystectomy.

For the operative cholangiogram the patient was tilted 15° to the right of the Eric Samuel tunnel. The cystic duct was cannulated with a polyethylene catheter (size PE205), filled with saline, and connected to a syringe by a pyelostomy nozzle with a

tap. Three roentgenograms were taken using the pull-through technique after injections of water soluble contrast medium (45% hypaque or 60% urografin). The first injection was small, to demonstrate the normally narrowed terminal segment of the common bile duct.

The 4 day telepaque test (1 gm. iopanoic acid 3 times a day for 4 days) demonstrated stones in the gallbladder in 30 per cent of the patients with a nonfunctioning gallbladder on 2 oral cholecystographic examinations, thus materially improving the preoperative diagnosis of gallbladder stones. However, stones in the common duct were shown in only 32 per cent of the cases.

Intravenous cholangiography with 20 ml. biligrafin (iodipamide methylglucamine) demonstrated stones in 59 per cent of the cases with duct stones.

In view of the risk associated with biligrafin, and to a lesser extent with large doses of telepaque, it is concluded that neither of these tests is desirable prior to cholecystectomy in the good-risk patient, provided that a satisfactory operative cholangiogram is obtained. These 2 studies should be reserved for the poor-risk patient when the presence or absence of duct stones may influence the decision to operate. The combined technique of a 4 day telepaque test followed by intravenous cholangiography is not recommended in any patient.—*Paul M. Dassel, M.D.*

Lloyd-Jones, William, Mountain, John C., and Warren, Kenneth. Annular pancreas in the adult. *Ann. Surg.*, Aug., 1972, *176*, 163–170. (From: Department of Surgery, Lahey Clinic Foundation, Boston, Mass.)

Annular pancreas is a rare anatomic anomaly. If the resultant duodenal obstruction is of sufficient degree, immediate surgical intervention is required in the newborn. The obstruction may be of such a low degree that the anomaly is an incidental finding at autopsy. In the intermediate degrees, symptoms usually begin in the third or fourth decade of life with diagnosis usually in the fifth decade.

Annular or almost annular pancreas is the result of fixation of the free end on one of the two buds from which the pancreas develops. The ring of pancreatic tissue which develops, as the duodenum develops, produces obstruction in the descending segment. The annulus usually has a single duct which passes from in front to the right and behind the duodenum emptying into the main pancreatic duct of the larger pancreatic head. Pressure of the duodenum on the annulus frequently leads to pancreatitis.

The authors report 14 patients, and in 5 of these the anomaly was found only after previous operation for gallbladder disease. Only 1 patient had an additional congenital anomaly, but in infants, coexisting congenital anomalies are found in about 70 per cent of cases. In infants, an exact preoperative diagnosis cannot be made since differentiation from duodenal atresia or stenosis and constricting peritoneal bands from malrotation can produce the same clinical picture. In the adult the disease is characterized by chronic partial duodenal obstruction which progresses over a period of years.

Clinically, upper abdominal pain, postprandial fullness and bloating, vomiting, weight loss and gastrointestinal bleeding are encountered. The clinical picture is modified by complications of which the commonest is peptic ulceration, especially in the duodenum.

In the adult the diagnosis is entirely dependent on findings in the barium study of the gastrointestinal tract. The characteristic changes are a small annular filling defect in the descending duodenum, symmetric dilatation of the duodenum above this, and reverse peristalsis. The complicating duodenal ulceration may lead to scarring which may prevent the usual duodenal dilatation. Differentiation between annular pancreas and cholecystoduodenocolic bands, postbulbar peptic ulcer stricture, cicatrizing duodenitis, and primary neoplasm of the duodenum can usually be made.

The ring was complete in 13 of the patients, while it was only partial in the other. In 11 it was in the descending duodenum and in the other 3 it was at the junction of the descending and inferior transverse segments. If there is no complication the treatment of choice is a retrocolic duodenojejunostomy without division of the annulus. The complications may modify this. Division of the annulus may not correct the obstruction because of permanent stenosis associated with intermingling of the annular tissue with the duodenal wall, and in addition section of its duct may lead to pancreatitis and fistula. Unless it is sought for, the anomaly is easily overlooked at the time of surgery on the biliary tract.—*E. Frederick Lang, M.D.*

Genitourinary System

Smith, Lynwood H., Fromm, Hans, and Hofmann, Alan F. Acquired hyperoxaluria, nephrolithiasis, and intestinal disease: description of a syndrome. *New England J. Med.*, June, 1972, *286*, 1371–1375. (From: Mayo Clinic and Mayo Foundation, Rochester, Minn.)

Excessive urinary excretion of oxalate has been considered to be due to a genetic disorder of glyoxylic acid metabolism. There are 2 enzyme defects in this metabolic pathway that are known to result in accumulation of glyoxylic acid and its conversion to oxalic acid.

Acquired hyperoxaluria has been seen in several conditions including ingestion of ethylene glycol,

rhubarb gluttony, and in the excessive use of ascorbic acid. Pyridoxine deficiency is known to cause hyperoxaluria in a few cases.

Nephrolithiasis as a complication of bowel disorders has been noted many times.

The authors now describe a new syndrome of acquired hyperoxaluria, nephrolithiasis, and intestinal disease. Seven patients with resection of the distal ileum developed calcium oxalate nephrolithiasis and increased urinary excretion of oxalate. The etiology of hyperoxaluria in these patients is uncertain, but it is suggested that the hyperoxaluria results from the profound disturbance in bile acid glycine metabolism present in patients with intestinal resection or malabsorption in whom the bile acid is exposed to deconjugating bacteria.

The response to the administration to cholestyramine or taurine is good, making it worthwhile to measure oxalate excretion in all patients with intestinal disease and nephrolithiasis, since effective therapy is available.—*Richard E. Kinzer, M.D.*

Malek, Reza S., Greene, Laurence F., DeWeerd, James H., and Farrow, George M. Xanthogranulomatous pyelonephritis. *Brit. J. Urol.*, June, 1972, *44*, 296–308. (From: Mayo Clinic and Mayo Foundation, Rochester, Minn.)

Xanthogranulomatous pyelonephritis is a rare unilateral benign disorder, characterized by massive tissue necrosis followed by phagocytosis of liberated lipid material.

Most of the cases occur in the fourth through the sixth decade with the sexes equally affected.

Renal pain, low grade fever and generalized vague illness were present in most patients. About two-thirds of the patients had a history of obstructive uropathy, calculous disease or urologic procedures.

A renal mass was found in most cases, one-third had hepatomegaly and 1 had a draining flank sinus.

Cultures were always positive and usually showed *E. coli*, *Proteus* or *Pseudomonas*; however, the bladder urine was sterile in over half of the cases. Reversible hepatic dysfunction was present in about half of the cases mimicking hypernephroma.

The usual roentgenologic abnormality seen was a unilateral renal mass (with calcification in a few cases), hydronephrosis, and a functionless kidney.

The variable roentgenographic pattern, hepatic dysfunction and diverse manifestations of xanthogranulomatous pyelonephritis may closely mimic those of hypernephromas, perinephritic abscess, pyelonephritis with stone, renal carbuncle, and tuberculosis of the kidney.

The data in 18 cases of xanthogranulomatous pyelonephritis are tabulated. Reproductions are presented of roentgenograms (in 7 cases) and of gross and microscopic pathology.—*Paul M. Dassell, M.D.*

Blood and Lymph System

Yao, S. T., Hobbs, J. T., and Irvine, W. T. Peripheral arterial insufficiency of the lower extremities unassociated with organic occlusions. *Surg., Gynec. & Obst.*, June, 1972, *134*, 963–966. (From: Surgical Unit, St. Mary's Hospital, London, England.)

Seven patients with atypical, intermittent claudication, cold painful feet and normal peripheral pulses were studied by venous occlusive plethysmography and arteriography. It was postulated that these patients had a functional circulatory disorder without major occlusive disease.

No arterial blocks were demonstrated at arteriography, but early venous filling of veins in the thigh was noted. The hemodynamic data indicated reduced resting blood flow with increased peripheral vascular resistance.

It was concluded that the clinical symptoms and the angiographic and hemodynamic findings arose through increased tonicity of precapillary sphincter and a change in permeability of arteriovenous anastomosis.

Three patients in the study responded to lumbar sympathectomy with relief of symptoms and return of their hemodynamics to normal.—*Robert I. White, Jr., M.D.*

Rhodes, Buck A., Rutherford, R. B., Lopez-Majano, V., Greyson, N. D., and Wagner, Henry N., Jr. Arteriovenous shunt measurements in extremities. *J. Nuclear Med.*, June, 1972, *13*, 357–362. (Address: B. A. Rhodes, Division of Nuclear Medicine, Johns Hopkins School of Hygiene and Public Health, 615 North Wolfe Street, Baltimore, Md. 21205.)

The authors describe their technique for the measurement of peripheral arteriovenous shunting by arterial administration of albumin microspheres labeled with Tc^{99m}.

These studies were initially carried out in dogs and later in patients.

The usual patient dose was 2 to 4 mc of radioactivity or less than 1/300 the number of microspheres with 15 to 30 micron diameters found to produce hemodynamic changes. The amount of shunting was determined from sequential counts over the chest following the arterial and then intravenous administration of calibrated doses of microspheres.

More than 95 per cent of the patients without clinical evidence of shunting showed less than 3.2 per cent shunts (an average of 0.09 per cent).

In a patient with congenital arteriovenous fistula a 9.8 per cent shunt was found and in a patient with pulmonary osteoarthropathy the shunt value was 33.2 per cent.—*Merle K. Loken, M.D.*

RADIATION THERAPY

Marcove, Ralph C., Miké, Valerie, Hutter, Robert V. P., Huvos, Andrew G., Shoji, Hiromu, Miller, Theodore R., and Kosloff, Rochelle. Chondrosarcoma of the pelvis and upper end of the femur: an analysis of factors influencing survival time in one hundred and thirteen cases. *J. Bone & Joint Surg.*, Apr., 1972, *54-A*, 561–572. (Address: R. C. Marcove, M.D., Memorial Hospital for Cancer and Allied Diseases, New York, N. Y. 10021.)

The pelvis and upper end of the femur were the most common sites of chondrosarcoma (approximately 60 per cent of all cases). One hundred and thirteen patients and 152 operations were studied, out of a series of 121 consecutive cases that were treated.

The data analyzed were sex, age, preoperative duration of a recognized mass, preoperative duration of pain, site and size of the lesion, histologic grade of the tumor, and date and type of surgical treatment. The basic method of analysis was by means of survival curves. At the time of follow-up of 40 living patients, 5 still had disease and 35 showed no evidence of disease. Eleven of the 35 had no evidence of the disease more than 15 years after their last surgical procedure. The data show that it is much more meaningful to use a 10 or 15 year survival rate as an index of cure of chondrosarcoma than to use a 5 year survival rate (11 of 59 deaths from the disease occurred more than 5 years after the initial diagnosis).

Persistent pain which is often overlooked is an important sign in evaluating patients with cartilaginous tumors; 25 per cent of the patients reported 2 or more years of preoperative pain, some as many as 10 and 14 years. It is also to be noted that cartilaginous lesions in this region of the skeleton can grow to large proportions causing relatively few symptoms.

The findings in this study indicate that the cure rate and length of survival are directly related to the microscopic grading of the tumors (in contradistinction to the lack of value of such histologic grading of osteosarcomas). In this instance, careful studies of pathologic specimens of chondrosarcomas of the pelvis and upper end of the femur showed no difference in the histologic features of the lesions.

The prognosis is also improved by the type of surgery; *i.e.* there must be a wide margin of normal tissue between the tumor and the line of resection if the best cure rate is to be obtained.

The treatment of choice should be a hemipelvectomy and there is no contraindication to radical surgery even in the face of multiple local recurrences.

In 18 cases, where radiation therapy was given as a primary treatment with no surgery performed, the short survival of these patients is noteworthy.—*E. Nicholas Sargent, M.D.*

RADIOISOTOPES

Dittrich, Frank A., and Goodwin, David A. Early recognition of chronic airway disease by the ^{133}Xe lung scan. *J.A.M.A.*, May, 1972, *220*, 1120–1122. (From: Palo Alto Veterans Administration Hospital, Palo Alto, Calif.)

The authors present a case report of a 27 year old nurse with pleuritic chest pain whose chest roentgenogram showed a small infiltrate in the middle lobe of the right lung.

Conventional pulmonary function tests were found to be normal.

A xenon 133 lung "scan" demonstrated delay in washout of the radioactive gas from both lung bases, indicating increased small airway resistance in these lung regions. This finding suggested the possibility of α_1 antitrypsin deficiency which was later confirmed by serum studies.

Repeat xenon studies when the patient was asymptomatic showed persistance of delayed clearance of xenon gas from the lung bases which is a typical finding in patients with α_1 antitrypsin deficiency.—*Merle K. Loken, M.D.*

Lomas, Frederick, Dibos, Pablo E., and Wagner, Henry N., Jr. Increased specificity of liver scanning with the use of ^{67}gallium citrate. *New England J. Med.*, June, 1972, *286*, 1323–1329. (Address: P. E. Dibos, M.D., Division of Nuclear Medicine, 615 North Wolfe Street, Baltimore, Md. 21205.)

Sixty-one patients with defects in the liver as demonstrated by scans with Tc^{99m} sulfur colloid were also studied using gallium 67 citrate.

Twenty-three of 30 patients with cancer involving the liver showed definite accumulation of gallium in focal areas devoid of activity on the colloid scans. Of these, 11 of 12 had hepatocellular carcinoma, 4 of 9 had metastatic adenocarcinoma, 2 had malignant squamous cell carcinoma, 2 had malignant melanoma, 2 had Hodgkin's disease, and 1 each had undifferentiated carcinoma and hepatoblastoma.

Gallium was poorly taken up by 5 metastatic adenocarcinomas and 1 malignant carcinoid.

Four of 6 patients with liver abscess and 1 with cirrhosis had appreciable uptake of gallium in liver lesions indicating a lack of specificity of this agent for cancer.

Twenty-six other patients with other benign conditions producing focal defects on the colloid liver scans did not show appreciable gallium uptake in the liver lesions.

The gallium studies were said to be helpful in detecting hepatoma in patients with cirrhosis and in differentiating cancer and abscess from more benign causes of abnormal liver scans.

Five figures and one table are included.—*Merle K. Loken, M.D.*

Freedman, G. S. Gamma camera tomography: theory and preliminary clinical experience. *Radiology*, Feb., 1972, *102*, 365–369. (Ad-

dress: Division of Nuclear Medicine, Yale University School of Medicine, 333 Cedar Street, New Haven, Conn. 06510.)

A gamma camera tomography method is reported which requires a standard gamma camera detector, to which a special rotating collimator is mounted. The information from the detector is stored in a digital computer in a 64×64 array. Views are taken at 12 positions 30° apart and each digital image obtained is stored on magnetic tape. The data from these 12 separate images can be used by the computer to reconstruct any plane parallel to the plane of the crystal (longitudinal tomography). The image of the reconstructed plane is displayed on an oscilloscope and can be photographed for a permanent record.

The special inclined, parallel hole, rotating lead collimator is $1\frac{1}{2}$ inches thick, and contains 1,500 parallel hexagonal holes, each inclined 20° to the face of the gamma camera crystal. Studies using Tc^{99m} can usually be completed in less than 5 minutes, and the computer reconstructs a longitudinal image at any desired depth in approximately 10 seconds per tomographic plane.

There are several areas of potential application and usefulness of the above described system, which include the following: improved resolution in the Z plane; single-view organ imaging; determination of organ and lesion volumes; redistribution of overlying activity; transverse tomography; and color tomography.

The principal disadvantage of the system is the fact that the cross-section area seen in all 12 views diminishes with increasing depth; however, preliminary clinical experience with this method of gamma camera tomography has demonstrated the deep lesions not seen by conventional scanning methods.

Representative preliminary clinical results are illustrated in several figures.—*Philip D. Bourland, Ph.D.*

CHEMOTHERAPY

Harley, John B. (Chairman), Schilling, Albert (Chairman), and Glidewell, Oliver (Statistician), Acute Leukemia Group B and the Eastern Cooperative Oncology Group. Ineffectiveness of fluoride therapy in multiple myeloma. *New England F. Med.*, June, 1972, *286*, 1283–1288. (Address: John B. Harley, M.D., Division of Hematology, Department of Medicine, West Virginia University Medical Center, Morgantown, W. Va. 26506.)

This article presents the results obtained in a double-blind cooperative study concerning the use of fluoride in multiple myeloma. Eighteen institutions participated and the follow-up period was 5 years.

There were 215 patients who were randomized and of these 150 completed at least 6 months of therapy. They were divided into 3 subgroups: (a) 52 in the placebo group received 50 mg. of sodium chloride 4 times a day; (b) 51 patients in the low-dose group received 25 mg. of sodium fluoride and 25 mg. of sodium chloride 4 times a day; and (c) 47 patients in the high-dose group received 50 mg. of sodium fluoride 4 times a day.

Routine myeloma studies and skeletal surveys were obtained at regular intervals.

The radiation factors were duplicated in the follow-up roentgenograms.

All patients also received alkylating agents, steroids, hormones and radiation therapy as clinically indicated.

Evaluation of response to fluoride was based on the amount of pain and activity, loss of weight, pathologic fractures, need for roentgen-ray therapy, progressive disease, recalcification or fluorosis as shown by the roentgenograms and alterations in serum calcium and alkaline phosphatase, as well as survival.

Nausea occurred equally in the 3 groups even in those on placebos. There were several patients with arthralgia, diarrhea, retinopathy and gastrointestinal bleeding in each group including those on placebos. Arthralgia was considered to be due to fluorosis in several patients.

The study was planned to evaluate the effect of long-term administration of sodium fluoride as adjunctive therapy in multiple myeloma, since doses of 50 to 200 mg. of sodium fluoride daily have resulted in positive calcium balance and increased deposition of calcium in bone in patients with metabolic bone diseases. Patients with minimal or moderate osteosclerosis secondary to fluoride are usually asymptomatic. It was postulated that if fluorides could prevent the complications secondary to fractures and diffuse bone destruction in myeloma, then survival might be improved. However, survival in the 3 groups was similar; median survival in months was 36.9 in the placebo group; 40.0 in the low-fluoride group; and 32.3 in the high-fluoride group.

Roentgenograms showed increased density and coarsened trabeculae in: 12 per cent of the placebo group; 18 per cent of the low-dose patients; and 17 per cent of the high-dose patients. Subclinical fluorosis (increased alkaline phosphatase) was found in 17 per cent of the placebo group and in 43 per cent of the fluoride treated patients. Definite changes of fluorosis in 7 per cent of patients were not seen until 14 months and the median time before recognition was 30 months. Hypocalcemia or hypercalcemia was found in a few patients equally distributed in each group.

Clinically there was no difference in the number of patients showing deterioration in the 3 groups. However, if the median time from onset of the program to the time of progressive disease (objective or subjective) is compared, it was found to be 14 months in the high-dose group, 18 months in the low-dose and 28 months in the placebo group.

It was, therefore, shown in this double-blind study

that the use of fluorides in multiple myeloma did not result in increased longevity or alleviation of symptoms. The newly formed sclerotic bone was invaded by myeloma cells.—*Martha Mottram, M.D.*

ULTRASOUND

McIlroy, R. F. The routine use of ultrasound for the diagnosis of postoperative deep-vein thrombosis in a district general hospital. *Brit. J. Surg.*, Feb., 1972, *59*, 133–135. (From: Kent and Canterbury Hospital, Canterbury, Kent, England.)

A Sonicaid D.205 portable ultrasound apparatus was used to investigate patients with suspected deep-vein thrombosis. The machine consists of a transmitter operated at 2 MHz, a receiver, an audio-amplifier, and a loudspeaker. The transducer contains 2 ceramic crystals, one of which emits ultrasound and the other acts as a receiver.

The transducer, when placed over the femoral artery, emits a characteristic pulsatile sound indicative of major arterial flow. When the transducer is moved medially and the calf squeezed a characteristic venous roar caused by the Doppler effect is heard in the normal person.

Similar observations can be made over the inferior vena cava, the femoral vein in the adductor canal, and over the popliteal vein, thus localizing the site of obstruction. Obstruction is diagnosed if no sound is produced when the calf is squeezed.

Over a 6 month period, 322 legs were examined in patients who had undergone surgery, requiring hospitalization for more than 48 hours at varying intervals after surgery. Of 14 legs thought to have thrombosis on clinical grounds, only 50 per cent were confirmed by ultrasound. Nine asymptomatic legs were positive at ultrasound.

Four patients with deep-vein thrombosis diagnosed by ultrasound developed pulmonary emboli, this diagnosis being confirmed by clinical examination, chest roentgenography and electrocardiography. Two patients died from pulmonary emboli; deep-vein thrombosis had been diagnosed ante mortem by ultrasound in both patients.

No other technique was used to confirm the presence of deep-vein thrombosis, but the author considers the ultrasound reliable and simple and quotes 2 other series in which other techniques such as venography confirmed its accuracy.—*Roger C. Sanders, M.D.*

Tajik, Abdul J., Gau, Gerald T., and Schattenberg, Thomas T. Echocardiogram in total anomalous pulmonary venous drainage: report of case. *Mayo Clin. Proc.*, April, 1972, *47*, 247–250. (From: Mayo Graduate School of Medicine, University of Minnesota, Rochester, Minn.)

A 22 year old woman was shown by oxygen saturations to have an atrial septal defect. No angiocardiogram was obtained, but an echocardiogram showed a considerably increased right ventricular dimension and a left ventricular dimension at the upper limits of normal. The ventricular septum moved 'paradoxically' in the same direction as the posterior wall of the left ventricle in both systole and diastole.

At surgery, all the pulmonary veins were found to drain into a small posterior chamber which in turn drained into the right atrium. Total correction of the venous anomaly and the atrial septal defect was achieved.

Postoperative echocardiograms showed persistence of the paradoxical ventricular septal motion.

Paradoxical movement of the ventricular septum has been observed previously in patients with atrial septal defect and is considered to indicate right ventricular volume overload.

The authors believe this to be the first reported case in which echocardiography has been performed in a patient with total anomalous venous drainage.—*Roger C. Sanders, M.D.*

THERMOGRAPHY

Nathan, B. E., Burn, J. Ian, and MacErlean, D. P. Value of mammary thermography in differential diagnosis. *Brit. M. J.*, May, 1972, *2*, 316–319. (From: Hammersmith Hospital and Royal Postgraduate Medical School, London, England.)

Thermographic examination of the breasts was carried out on 359 women attending a breast clinic.

Ninety-seven were examined after the application of alcohol swabs for 5 to 10 minutes with a Bofors thermographic camera and the remainder were examined with an EMI thermoscan unit after sitting unclothed to the waist for 10 minutes.

Thermograms were read as abnormal, if there was a well defined heat spot or clear inequality between the breasts. Amongst 164 patients with normal thermograms, cancer of the breast was discovered in 7 patients, and 41 had histologically confirmed benign breast lesions—a false negative rate of 29 per cent.

Of the 195 patients with abnormal thermograms who were biopsied, 115 had no confirmed organic disease, 27 had cancer of the breast, and 53 had benign lesions. Seven biopsies were reported as normal breast tissue. There was therefore a 59 per cent false positive rate. No form of benign breast lesion was more likely to cause a thermographic abnormality than another.

Since the site of the breast lesion may not coincide with the thermographic lesion, it cannot be used as a guide to a biopsy site.

The authors conclude that thermography is not a satisfactory screening procedure, although once a base line thermogram has been obtained, the technique may be used for follow-up. Thermography represents "an interesting ancillary investigation but no more than that."—*Roger C. Sanders, M.D.*

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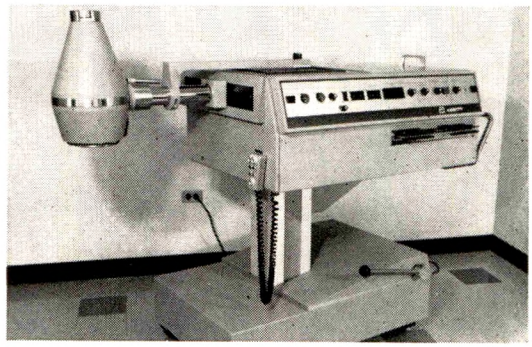
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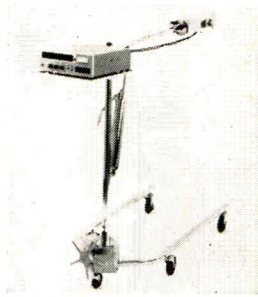
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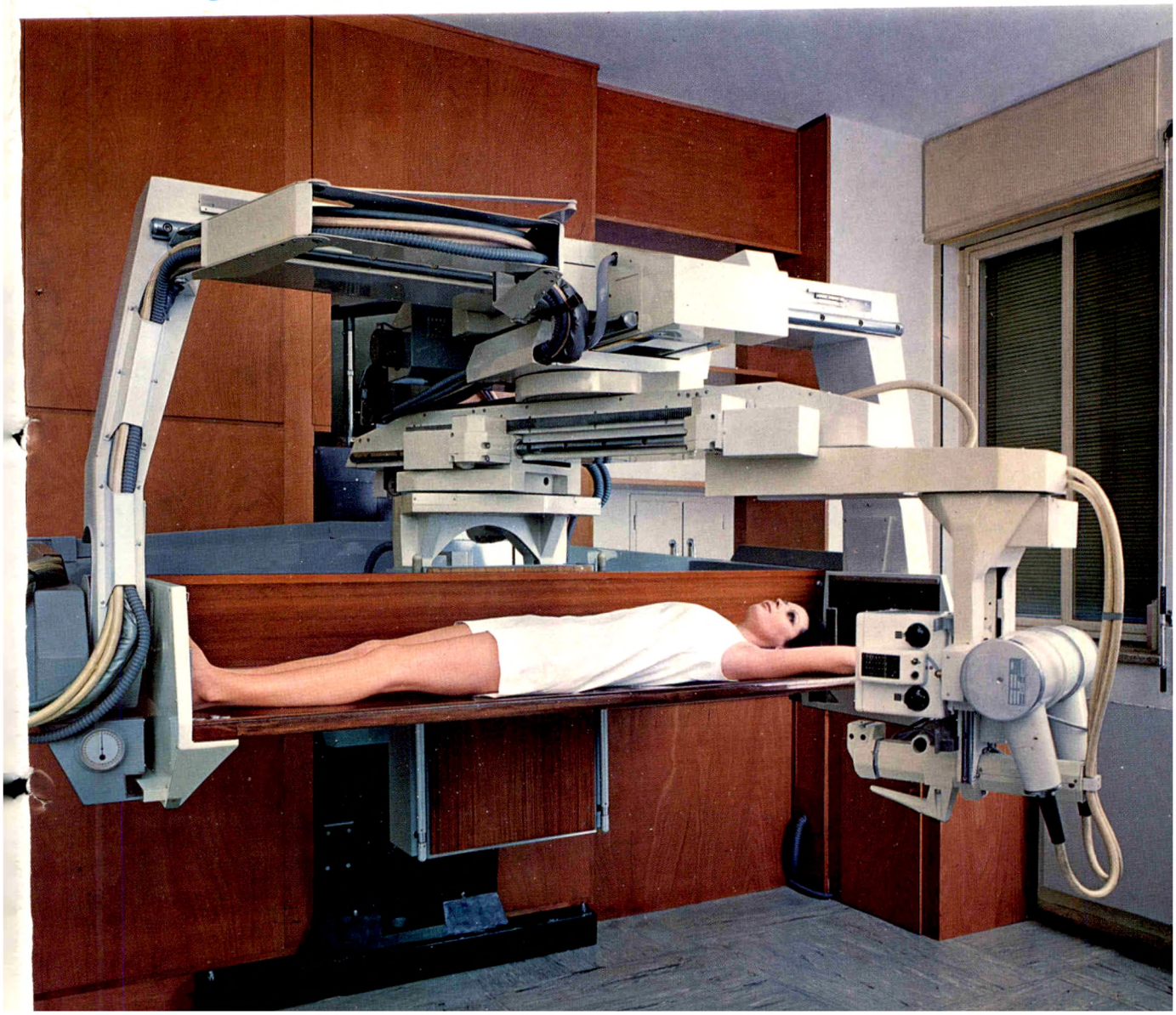
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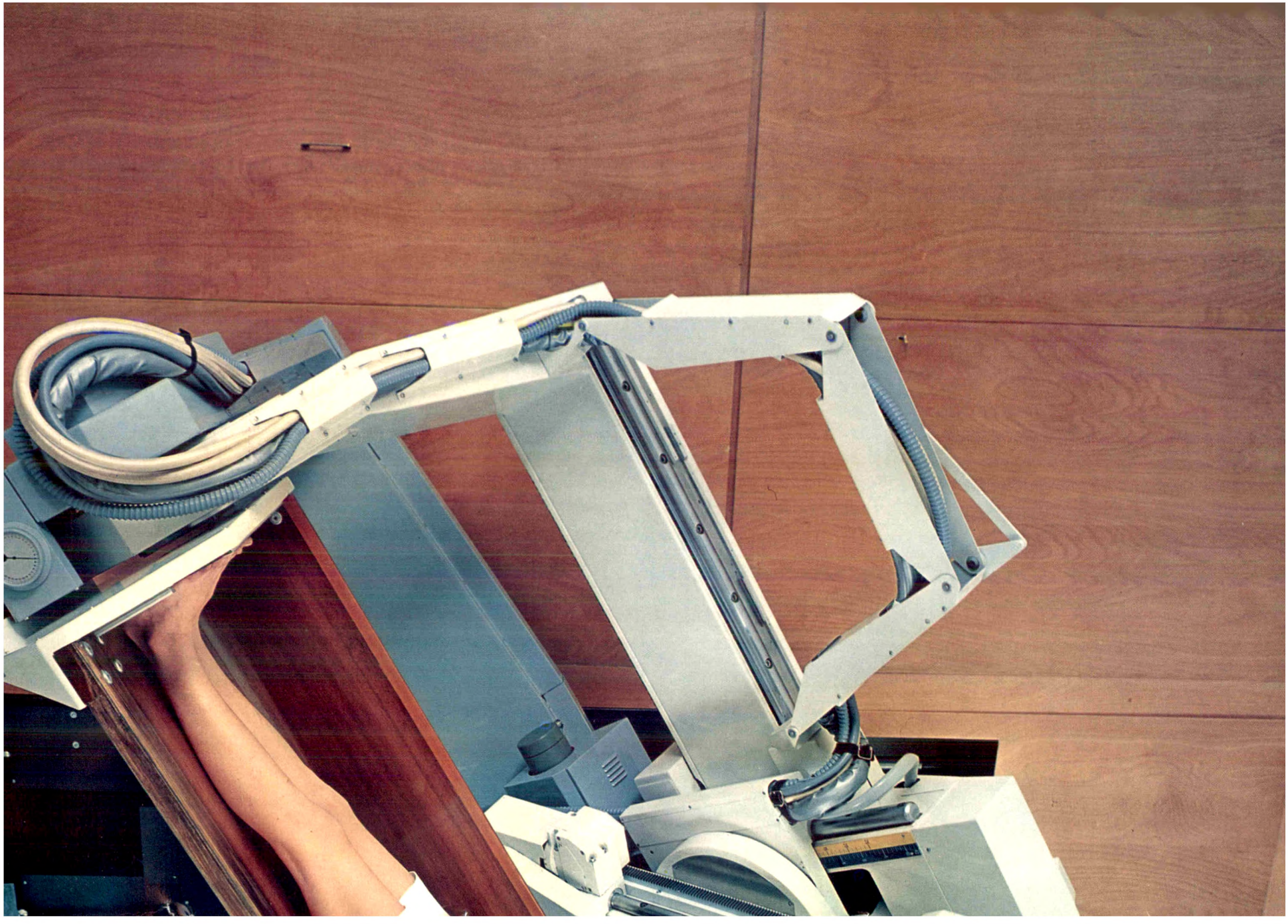
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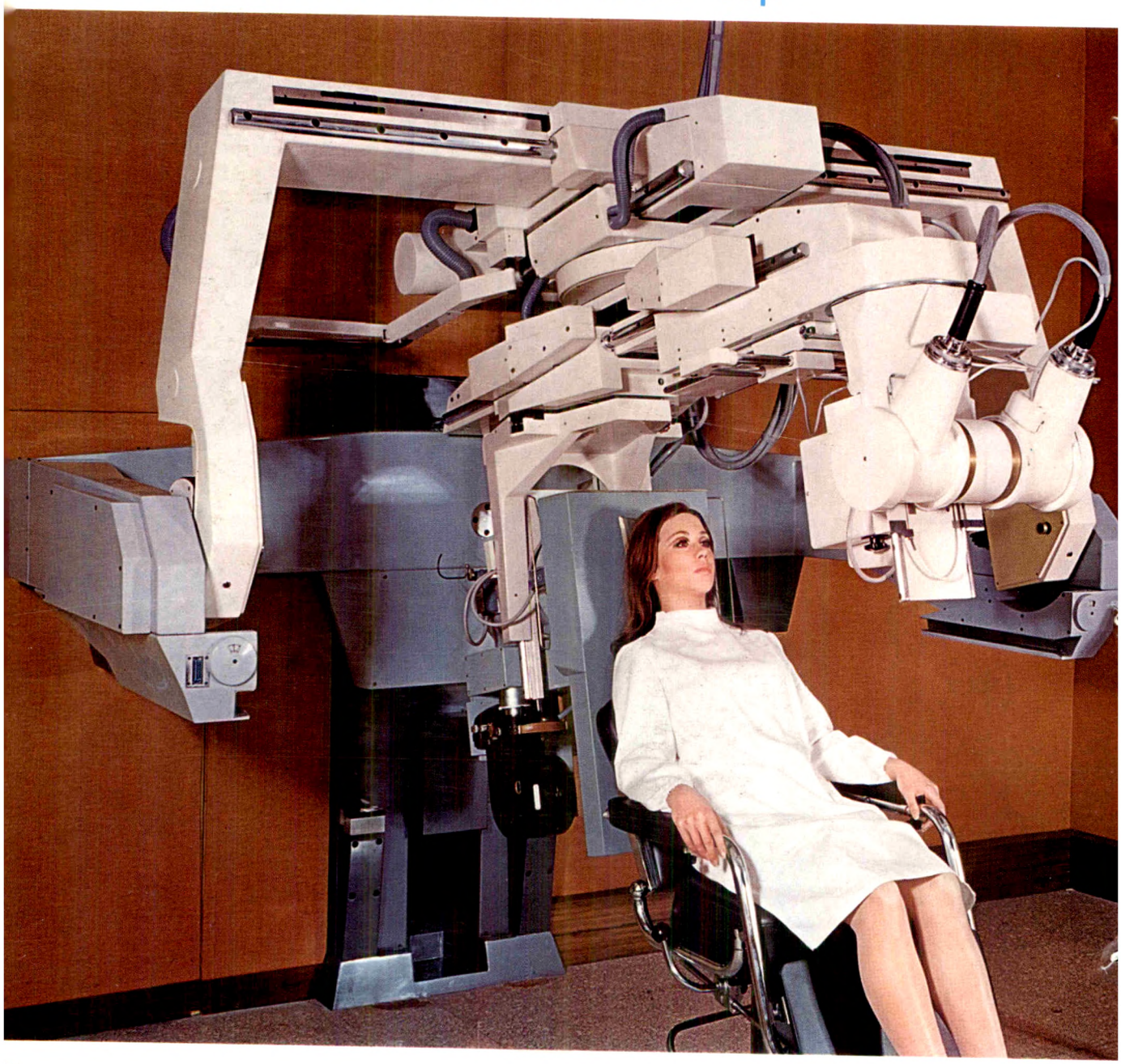
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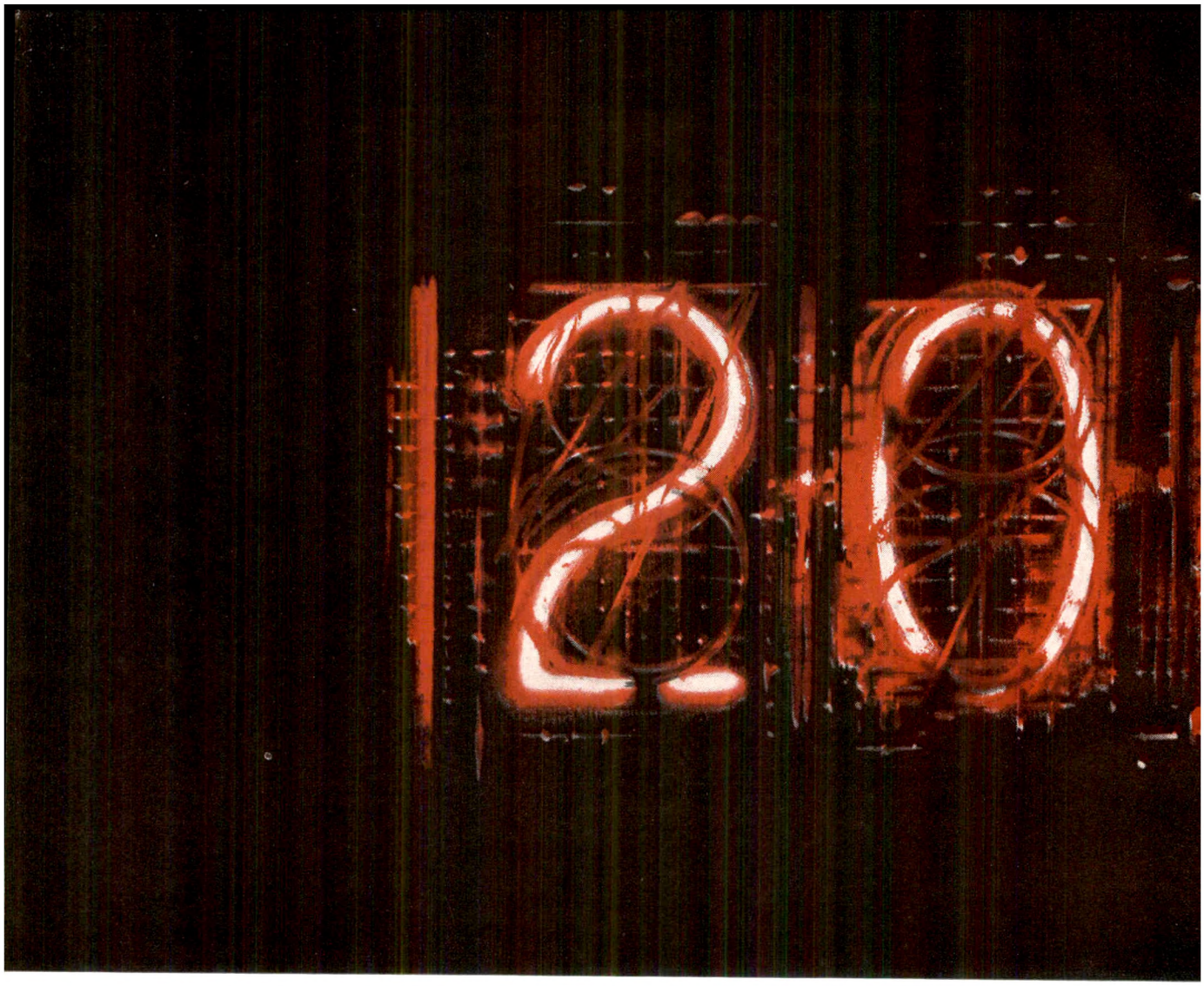
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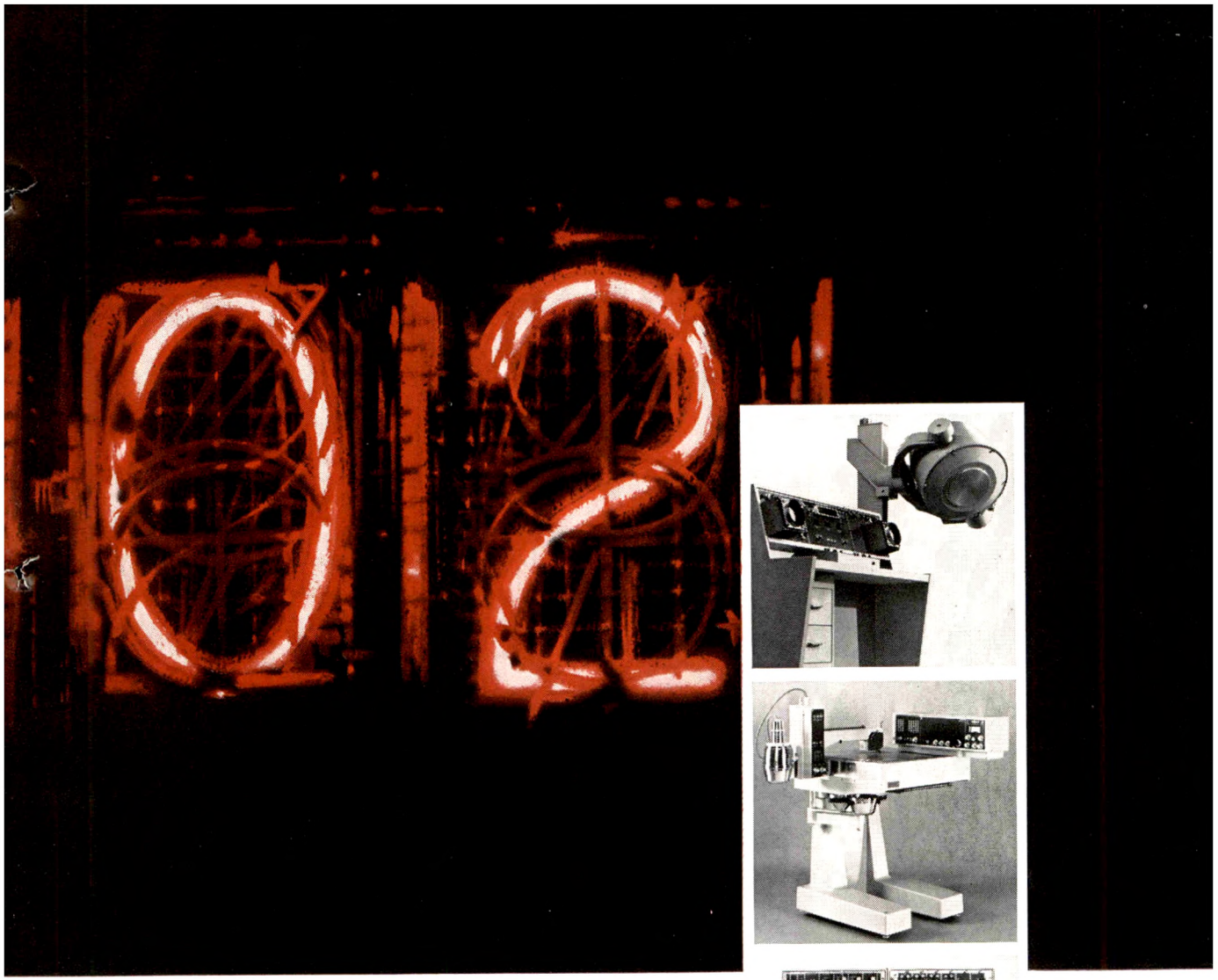
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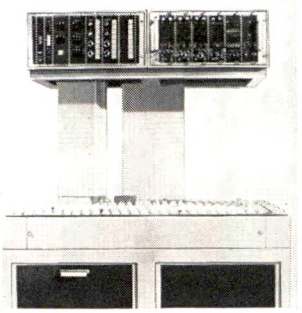
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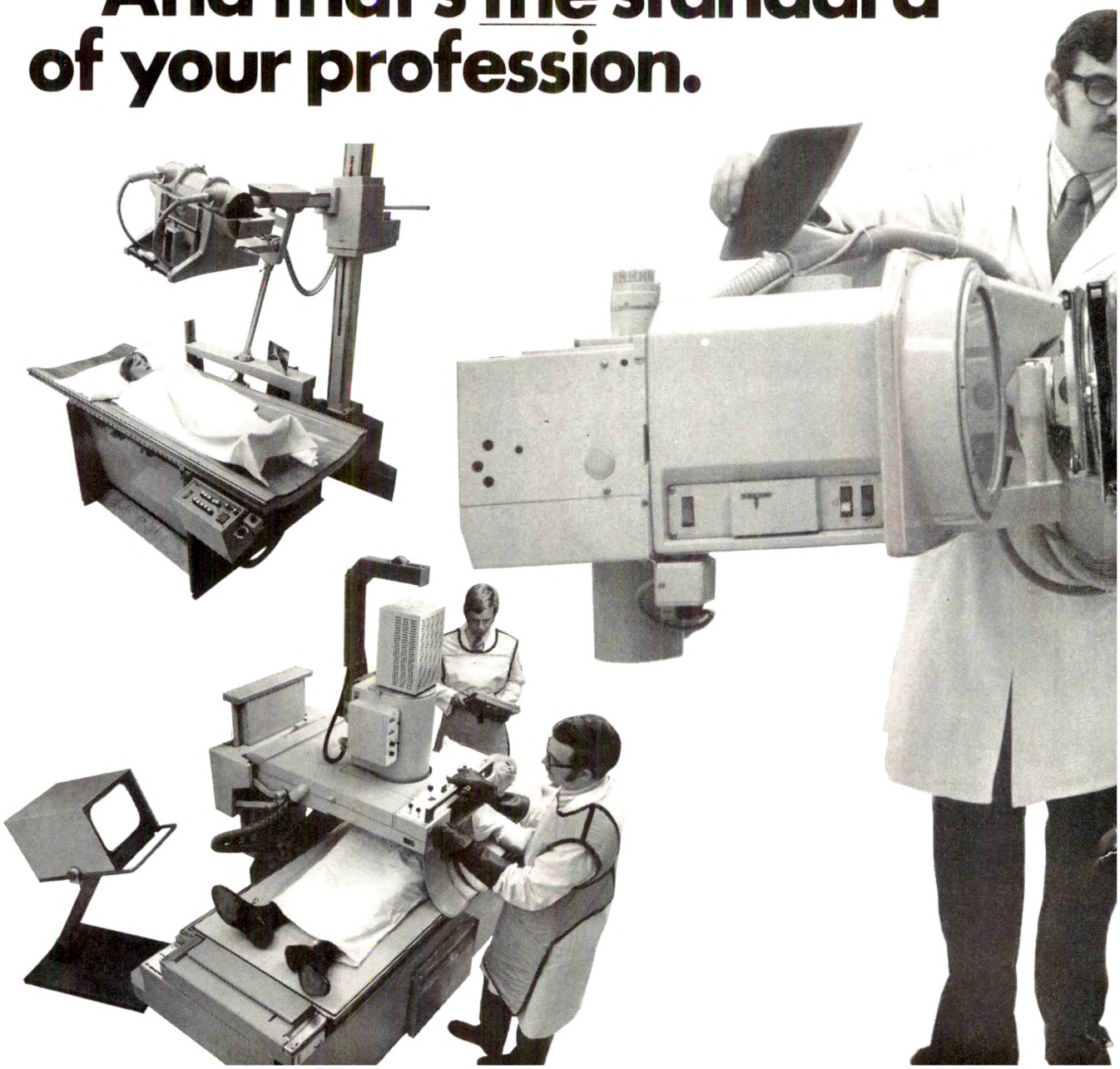
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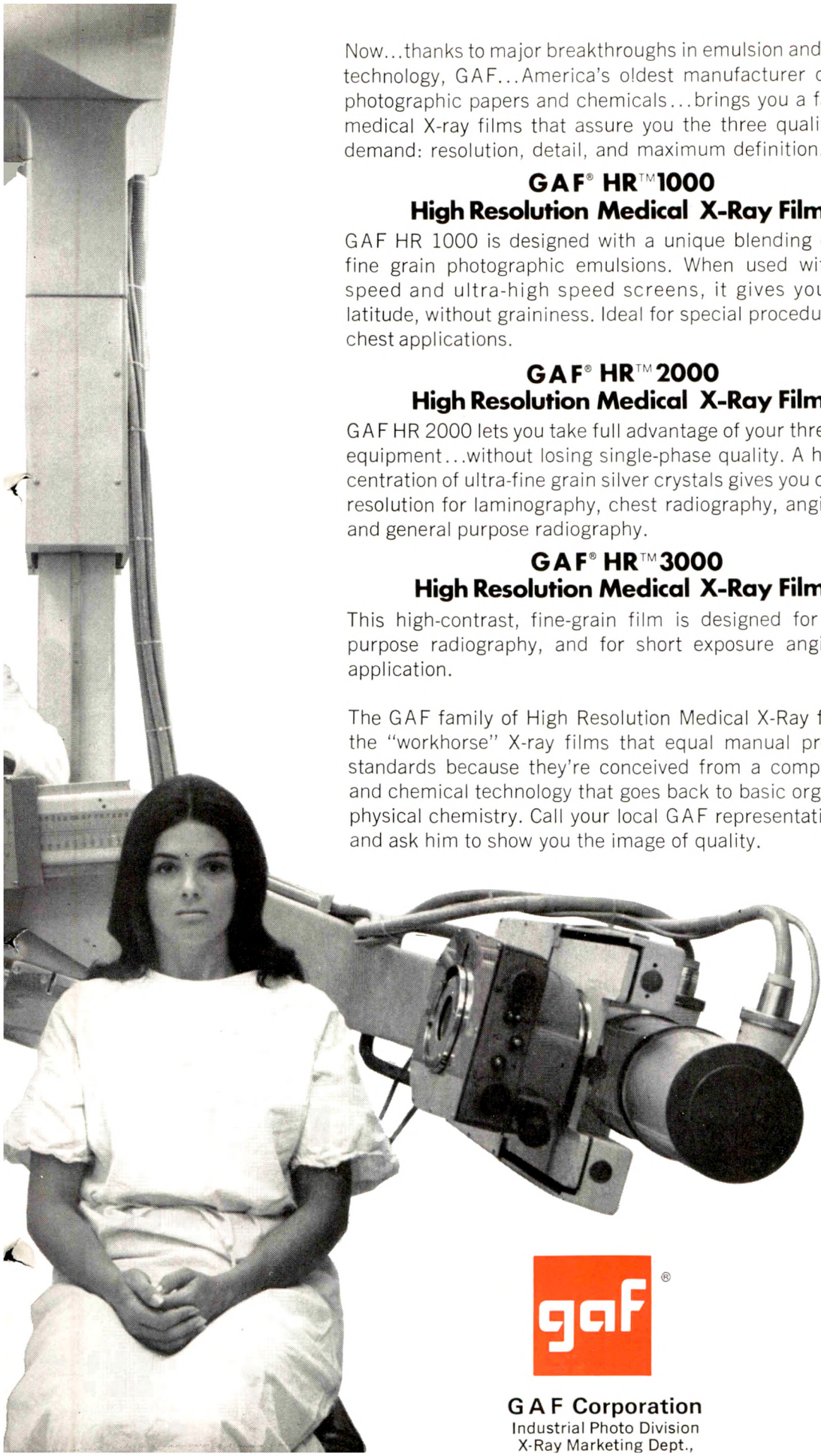
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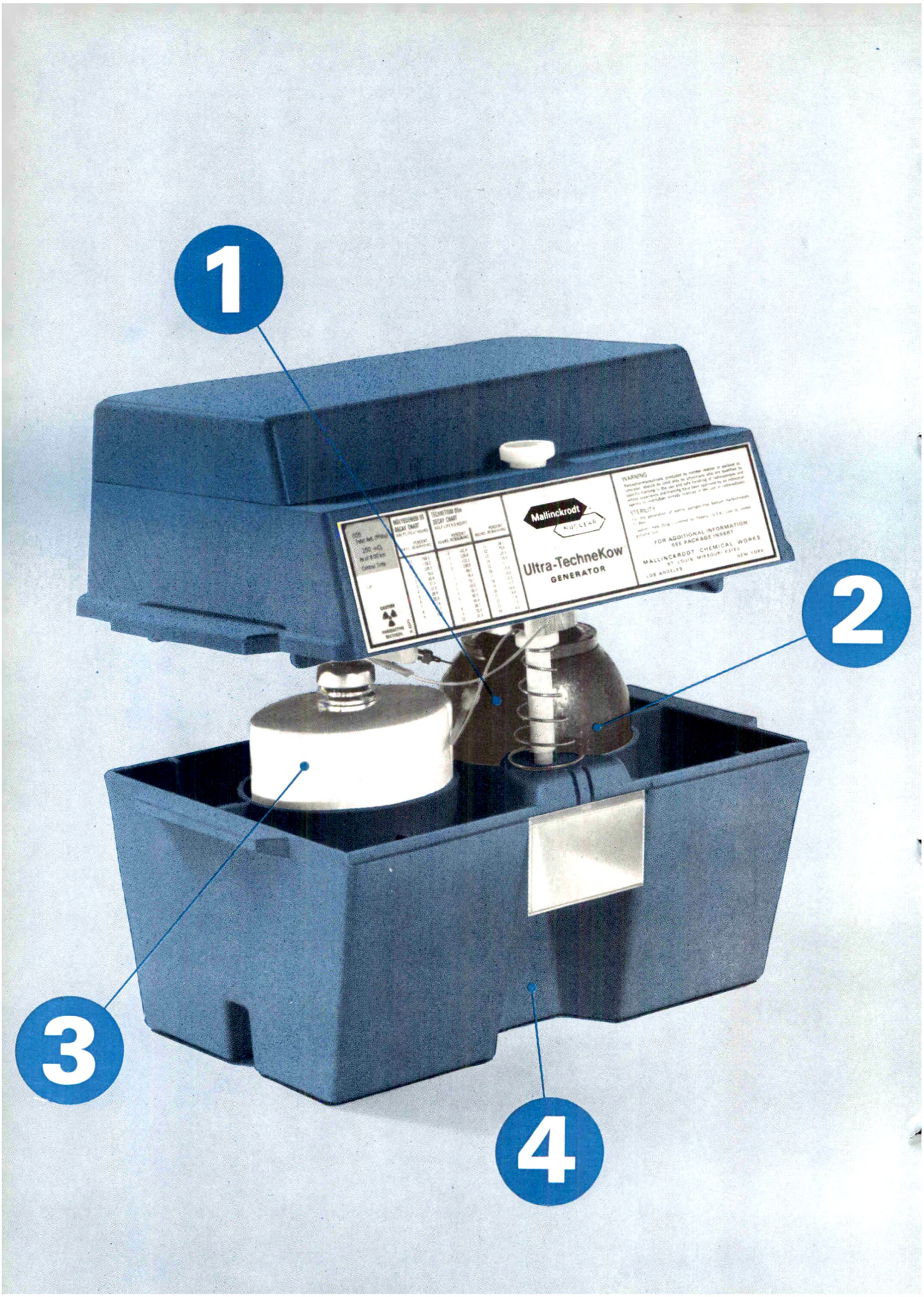
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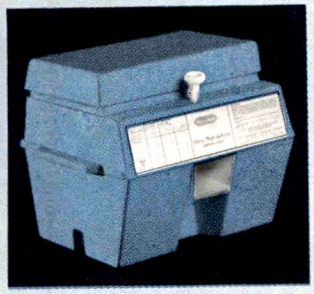
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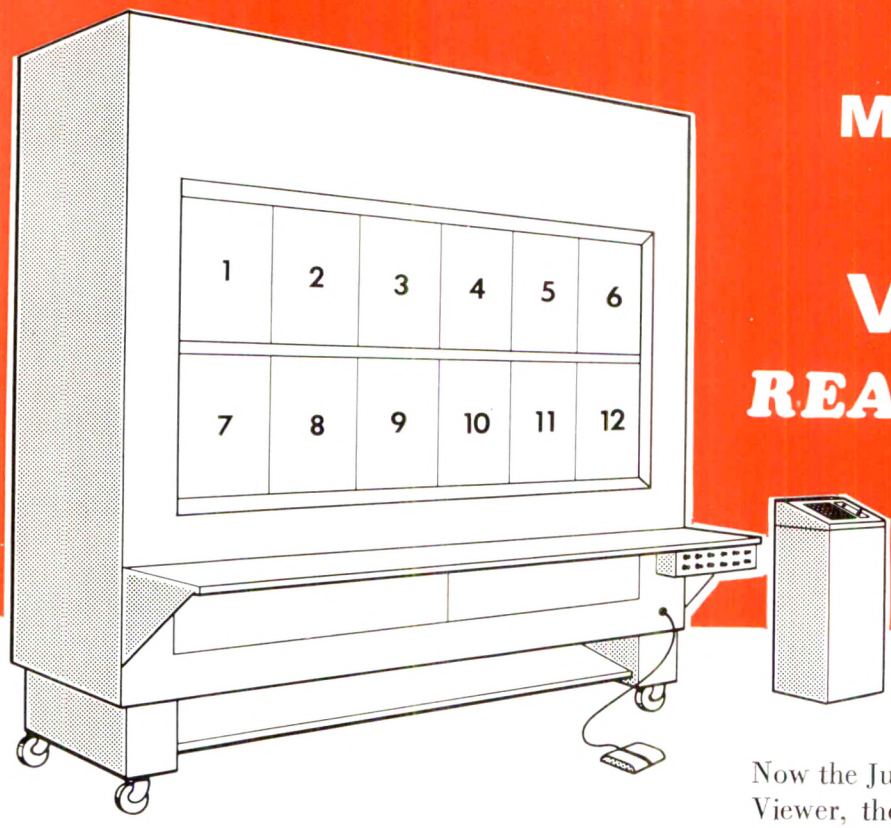
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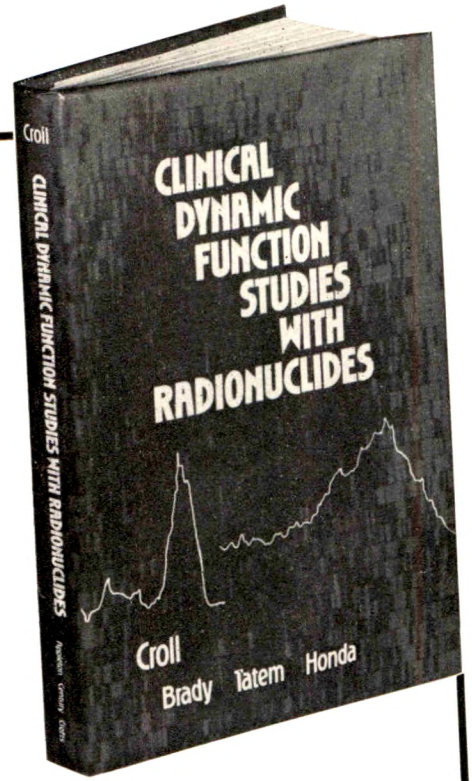
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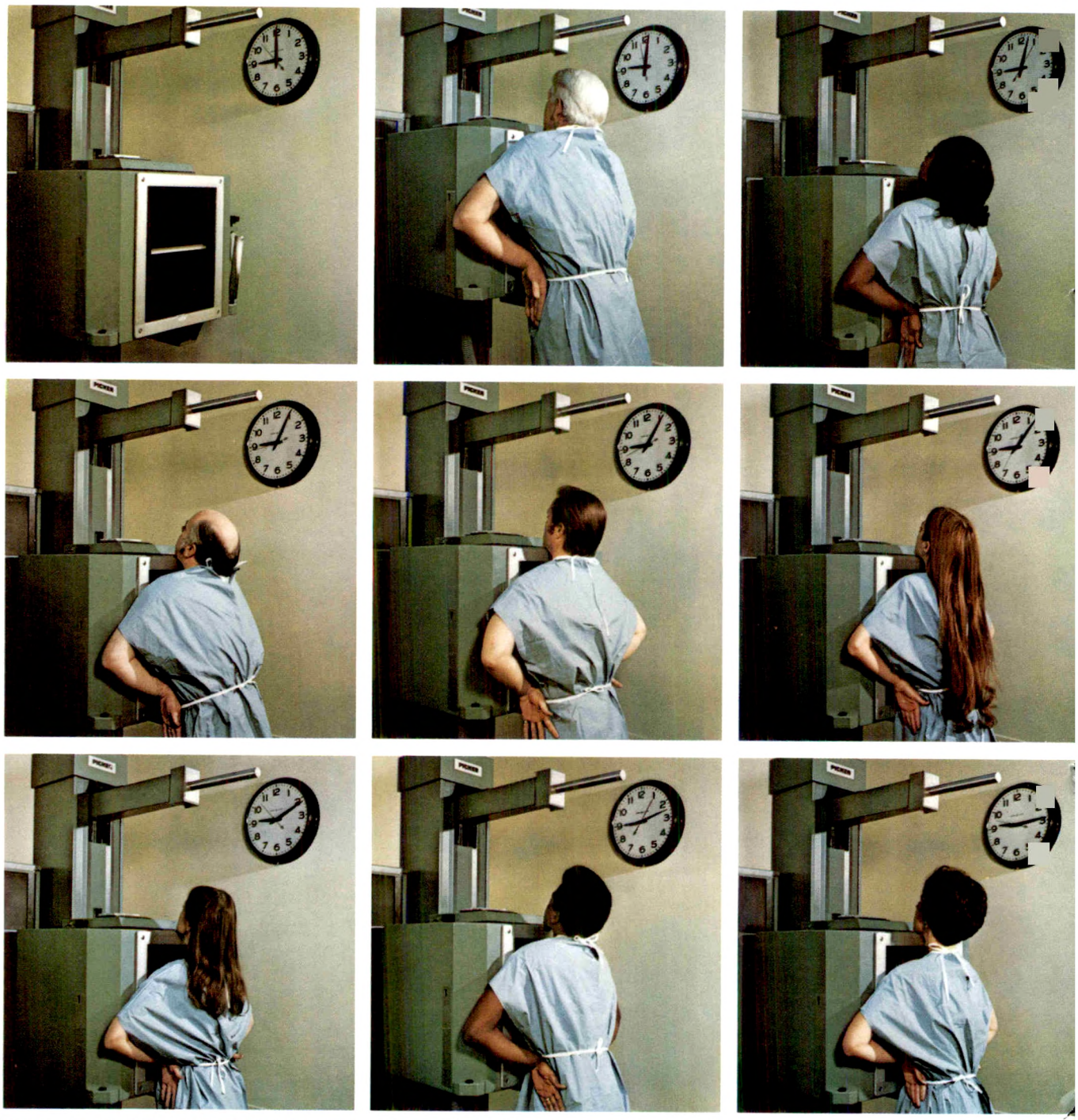
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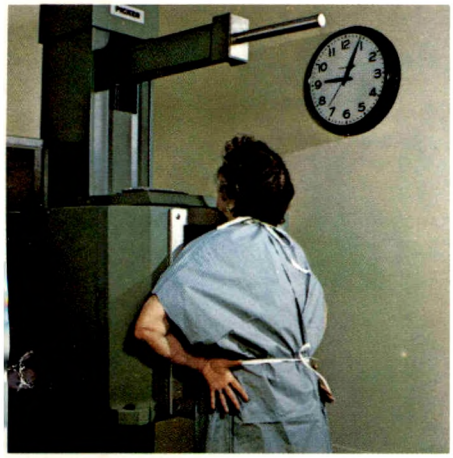
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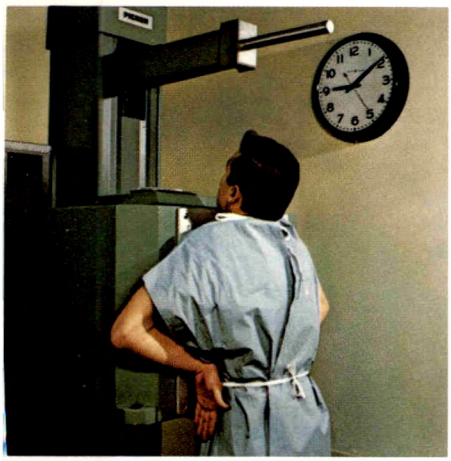
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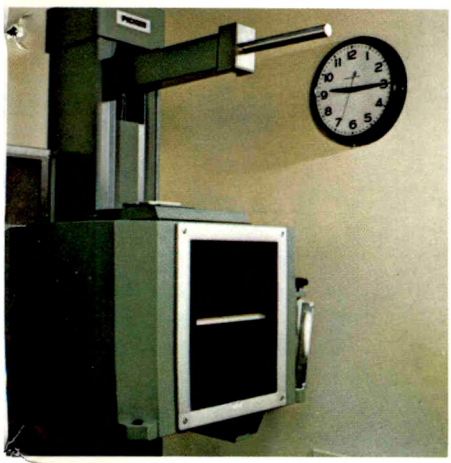
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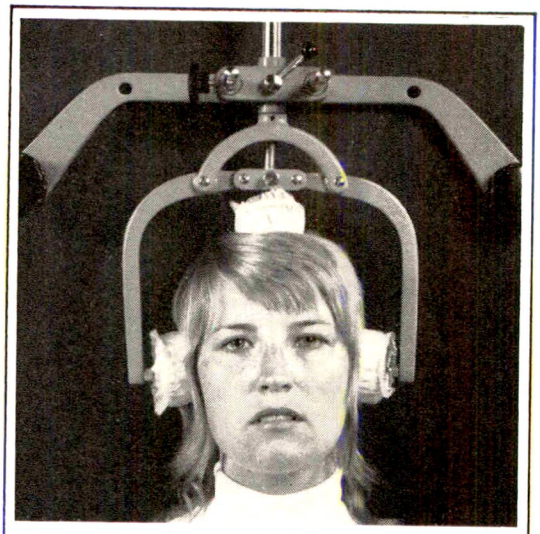
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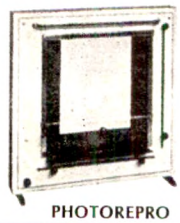
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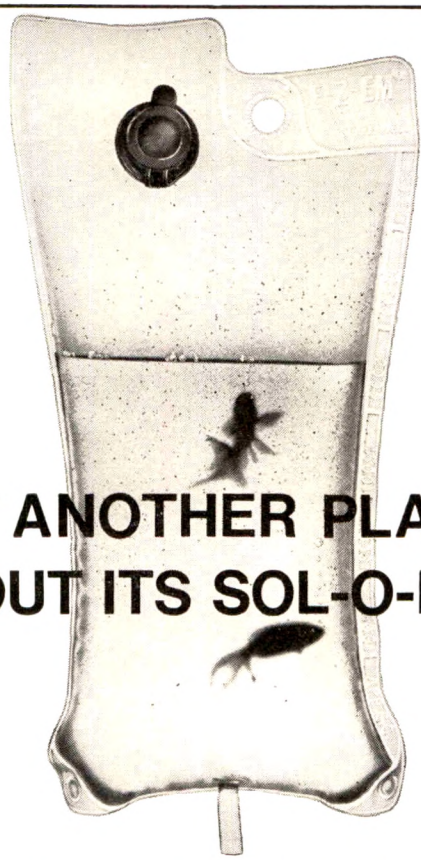
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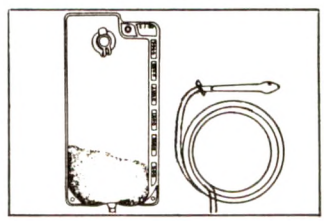
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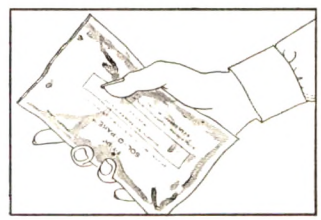
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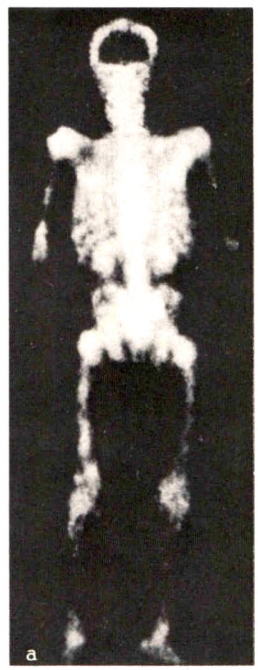


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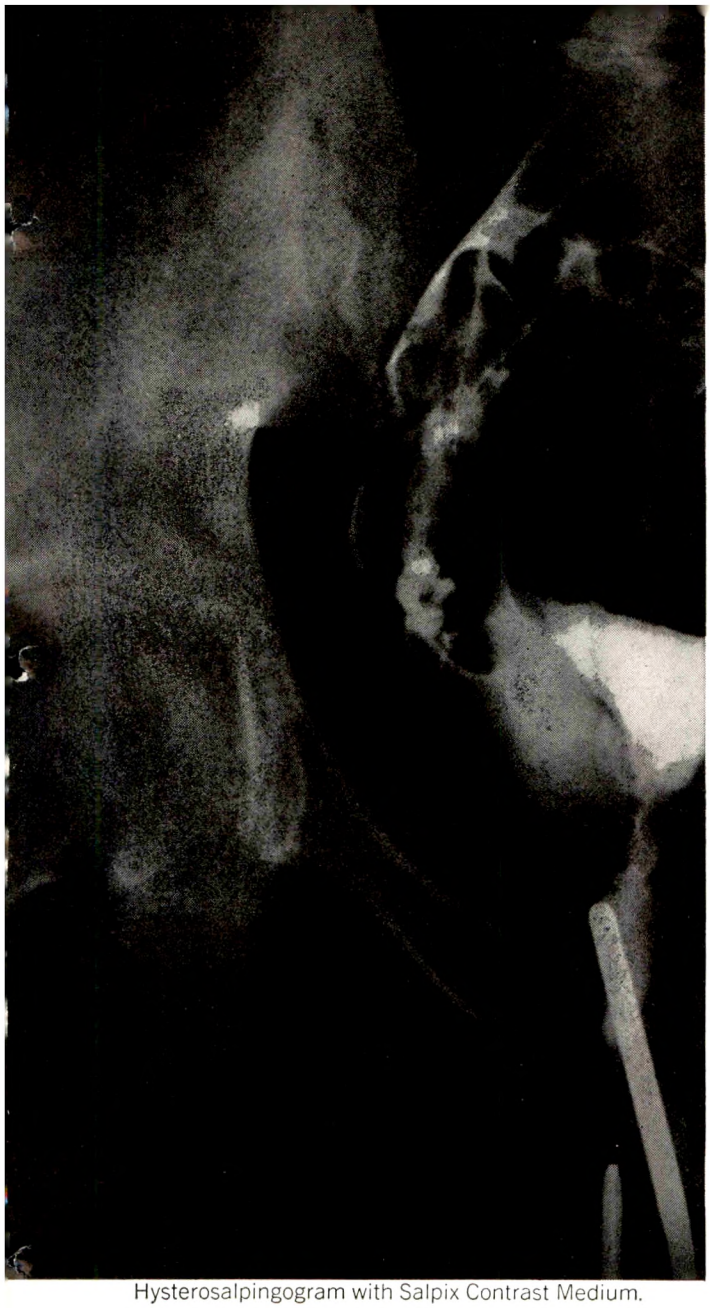
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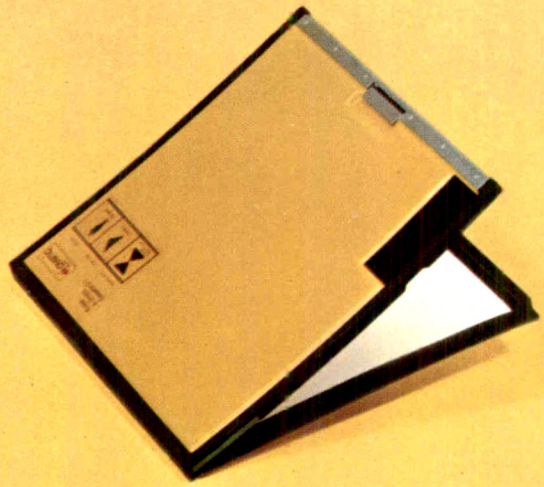
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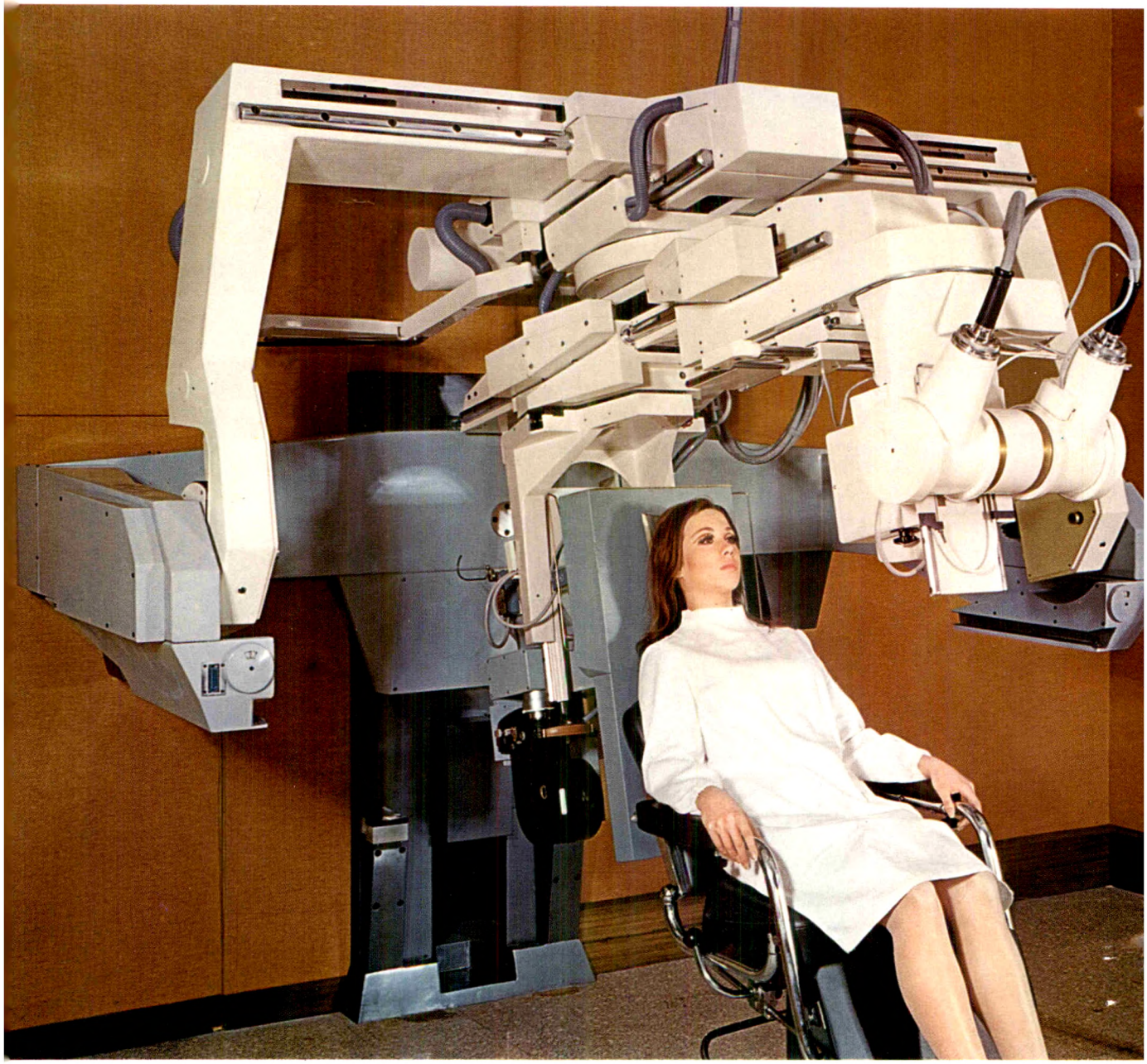
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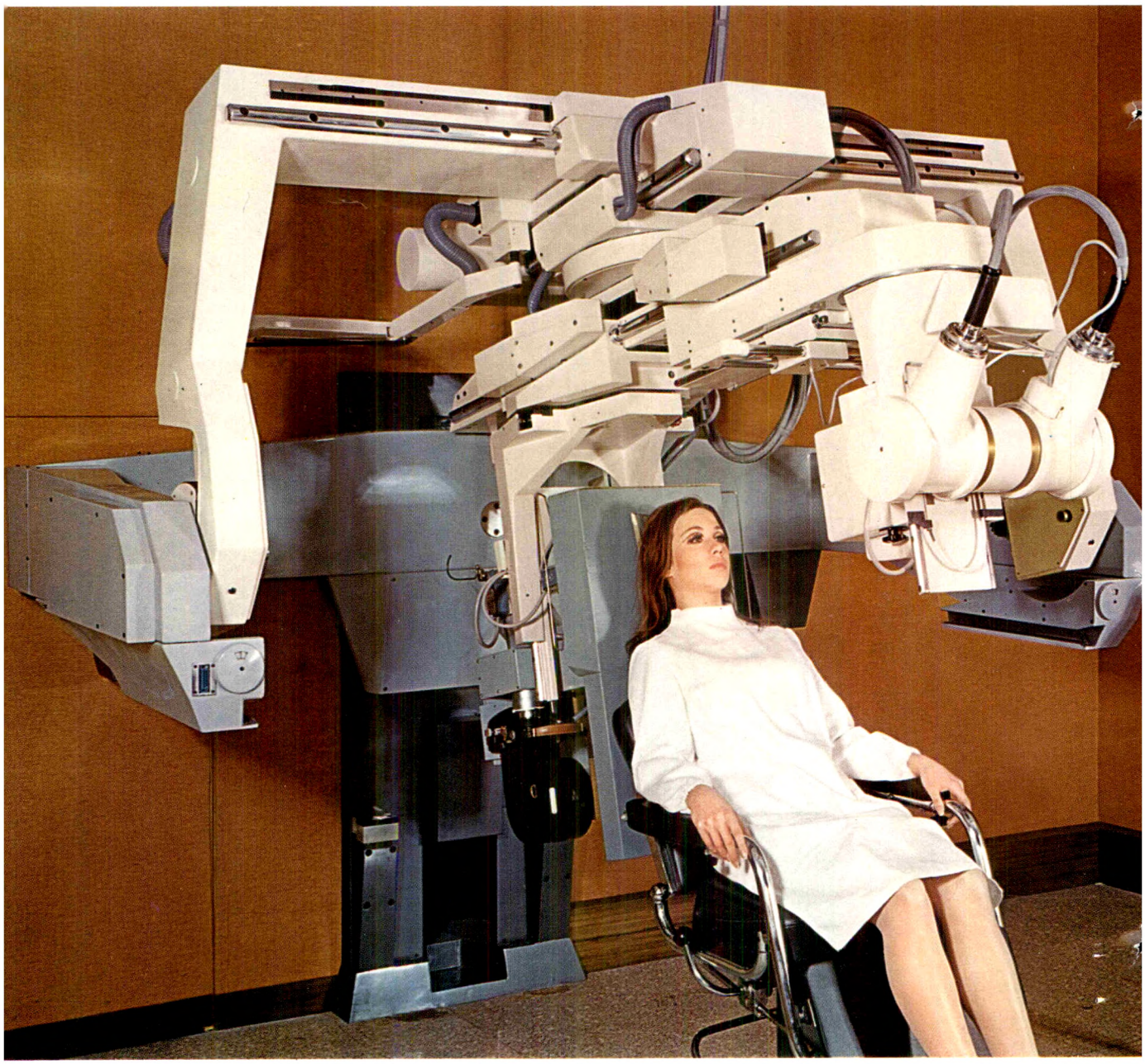
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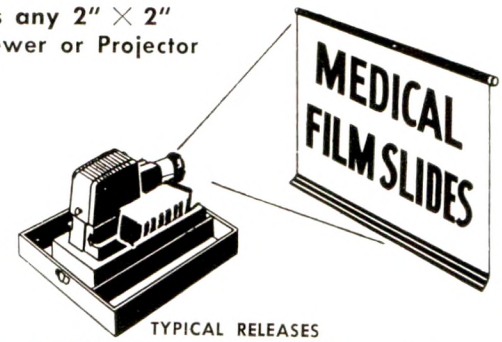
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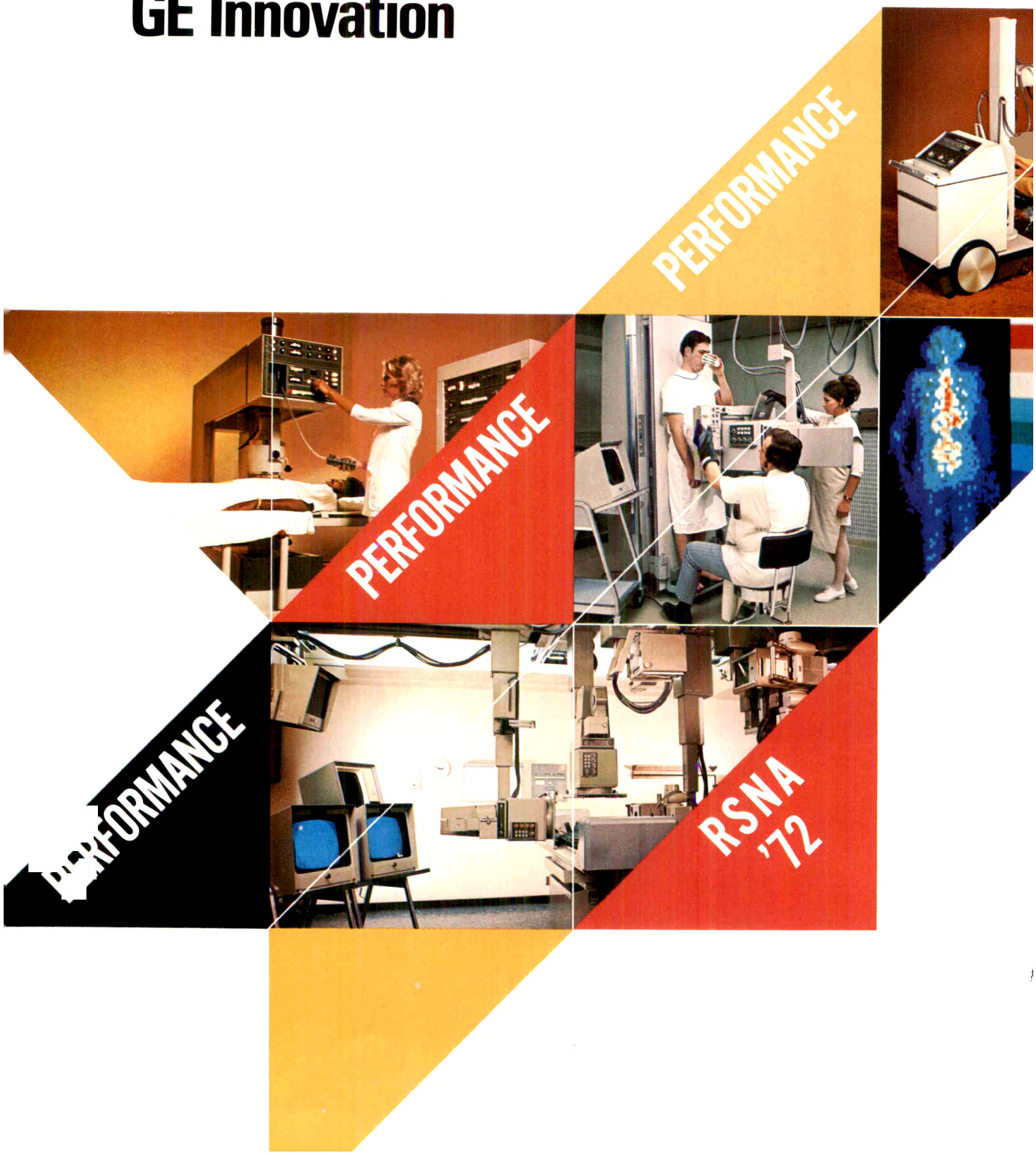
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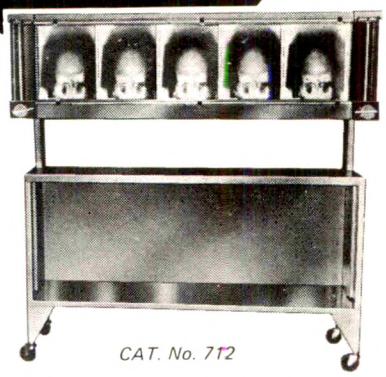
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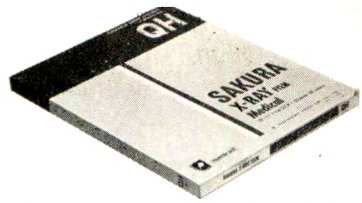
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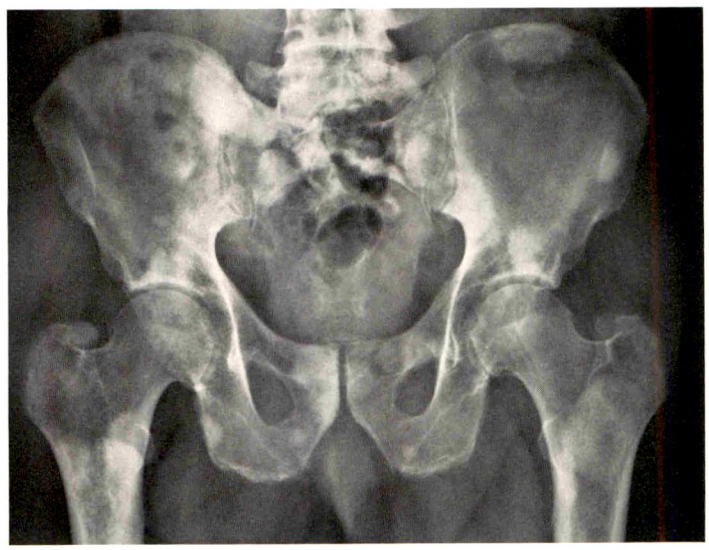
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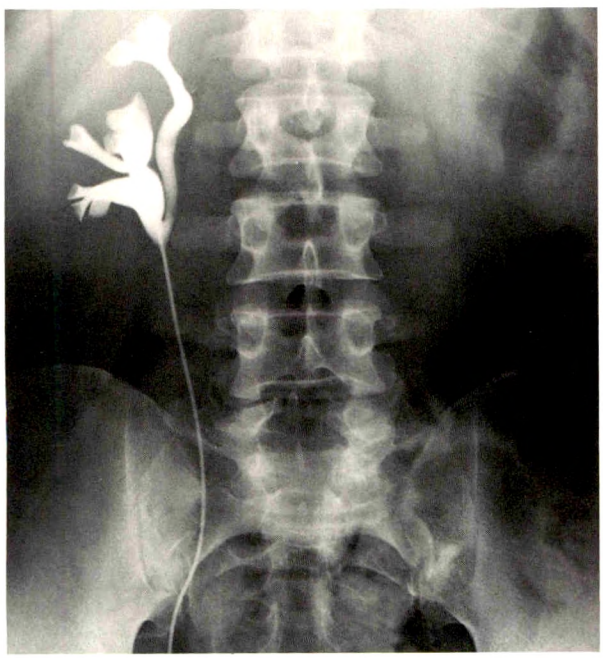
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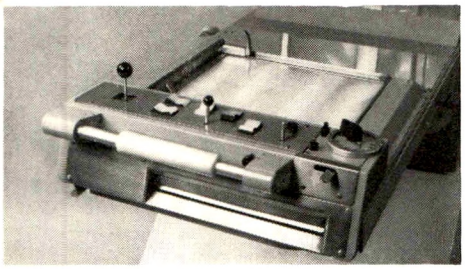
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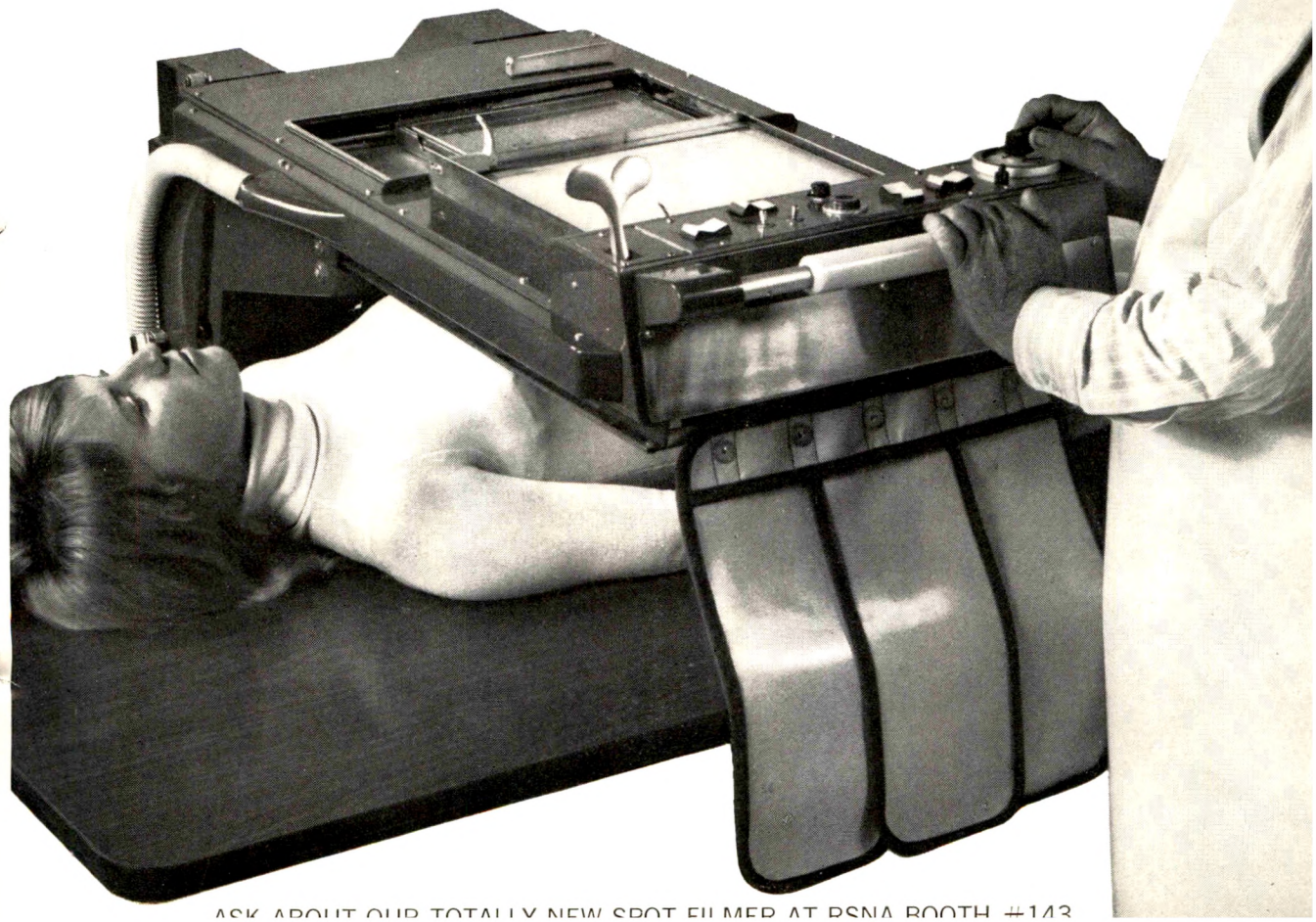
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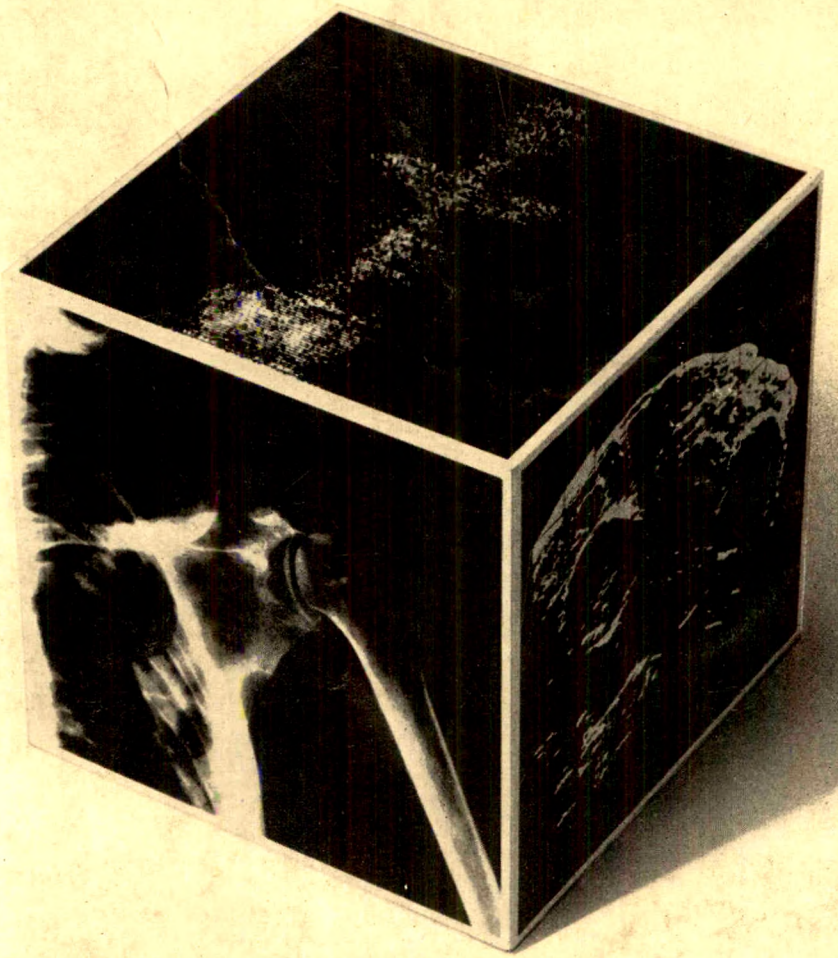


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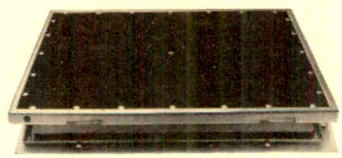
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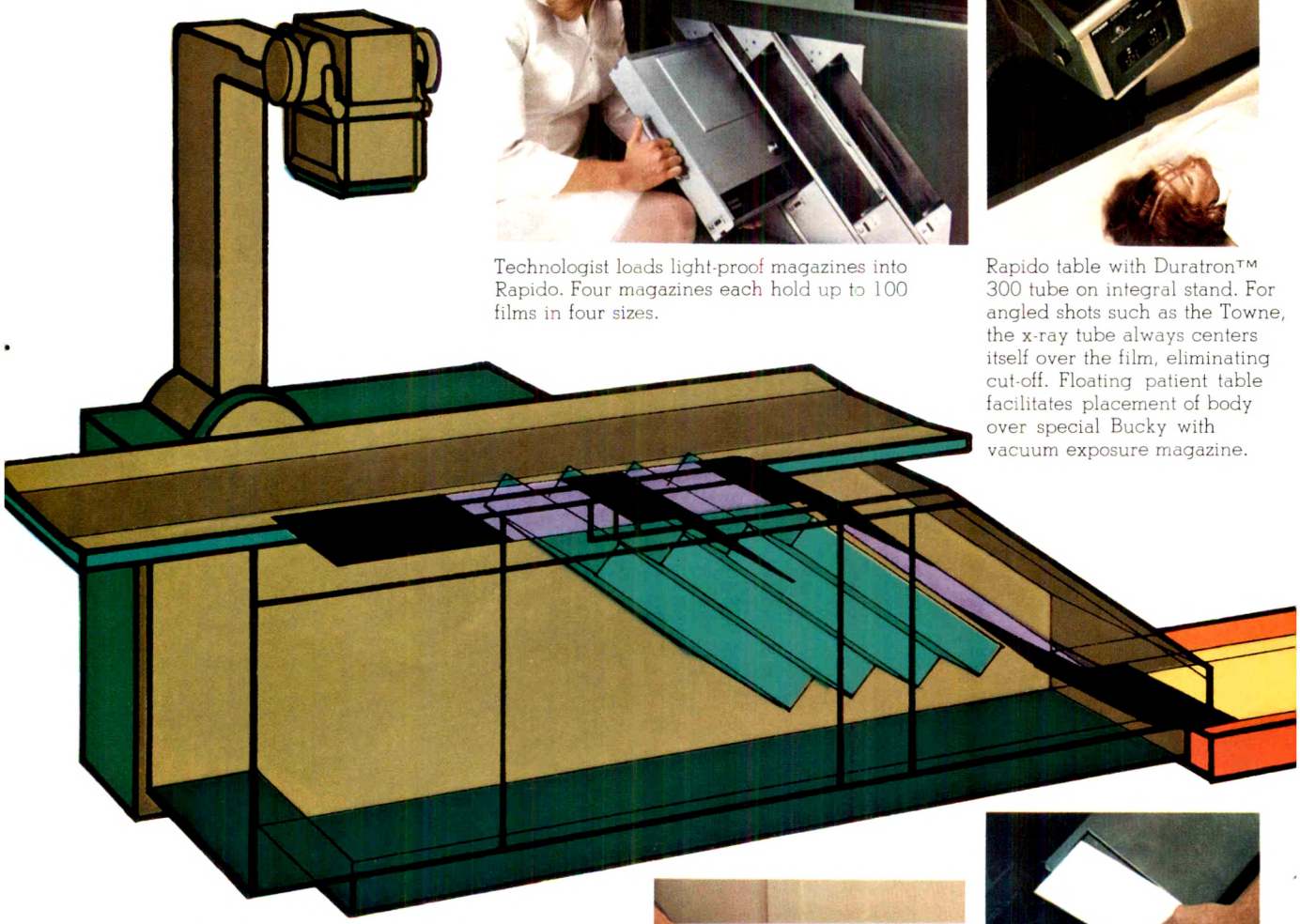
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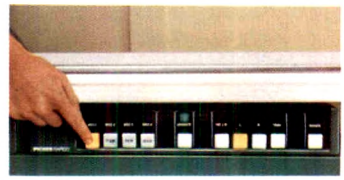
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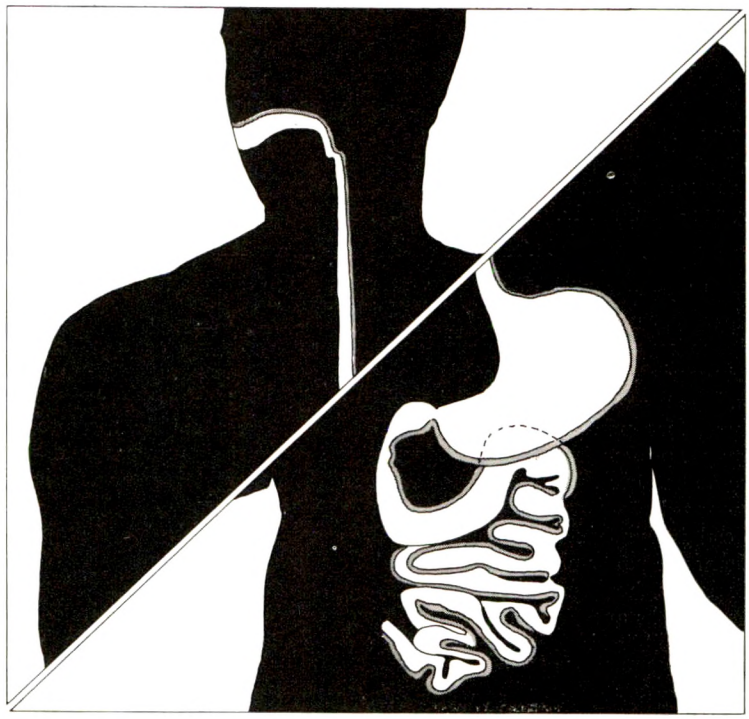
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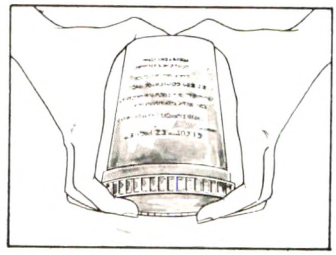
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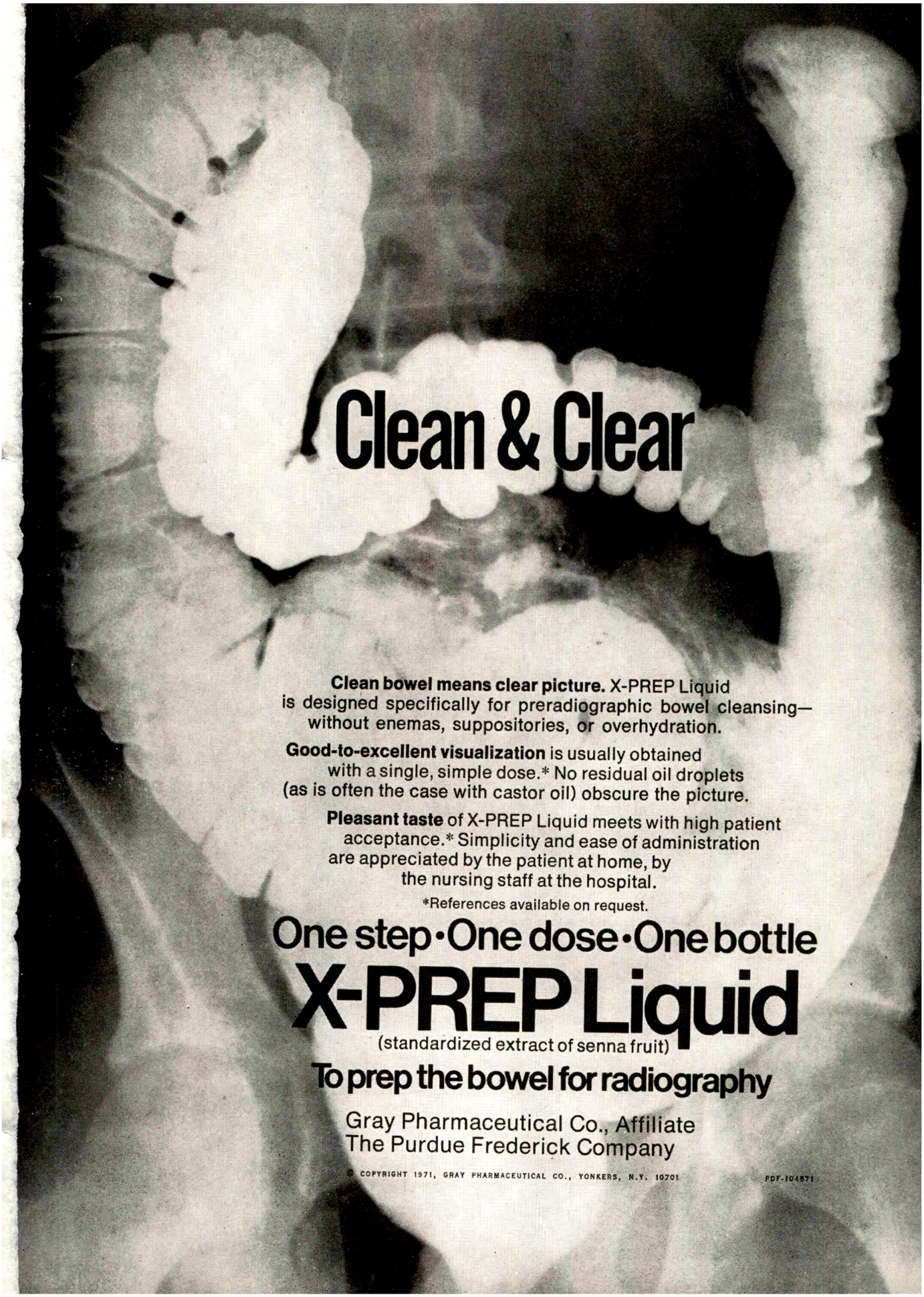
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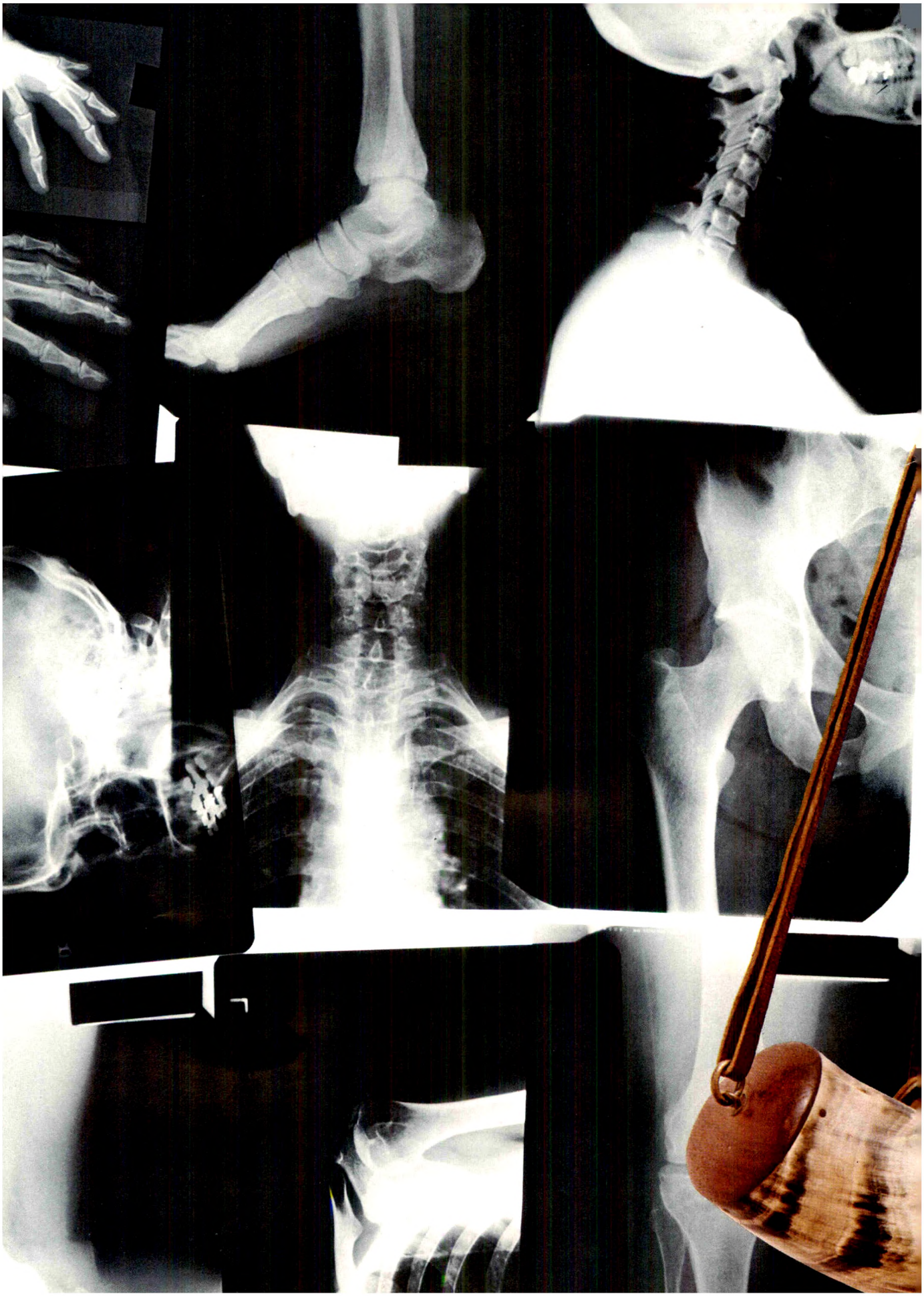
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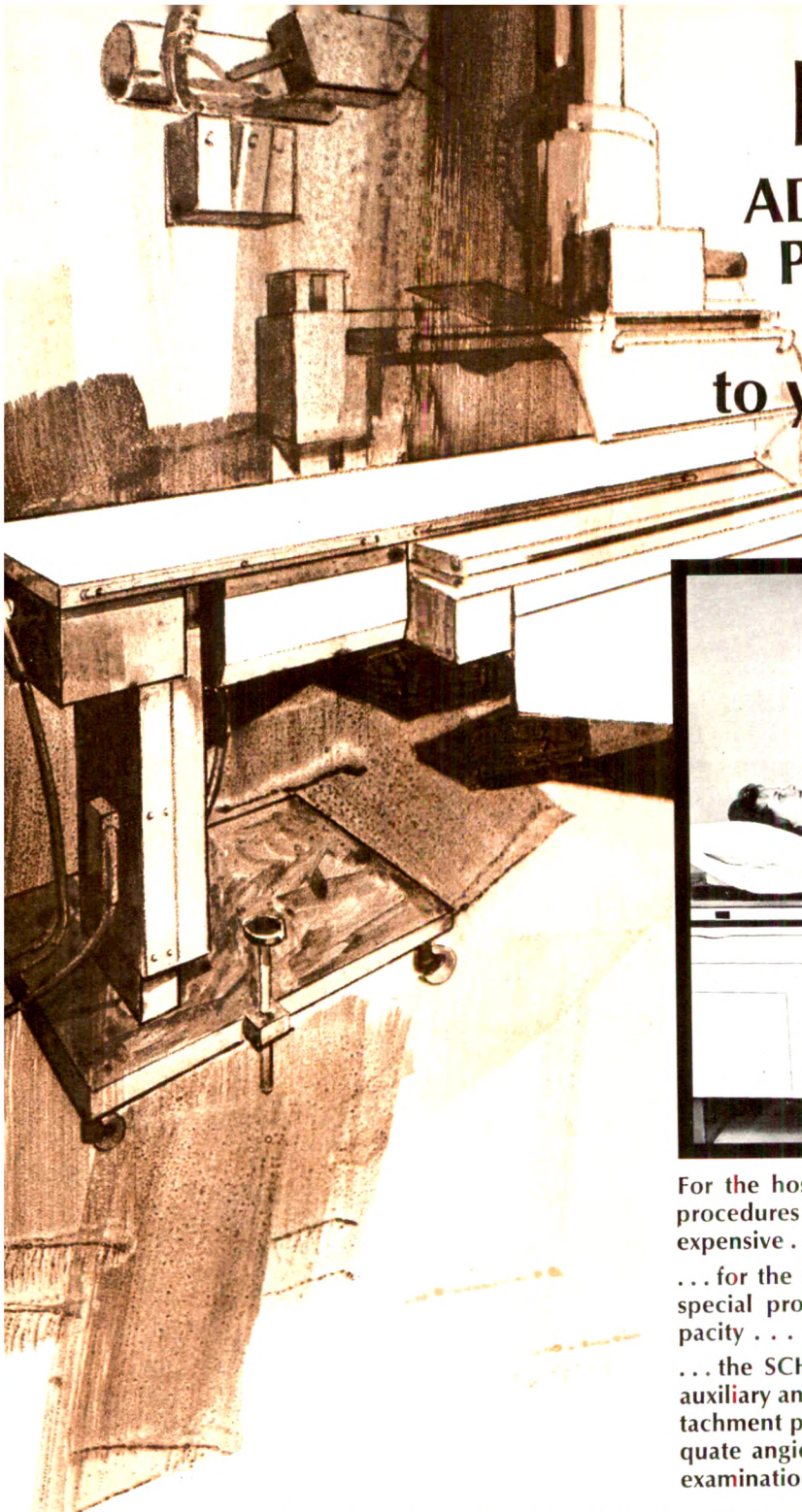
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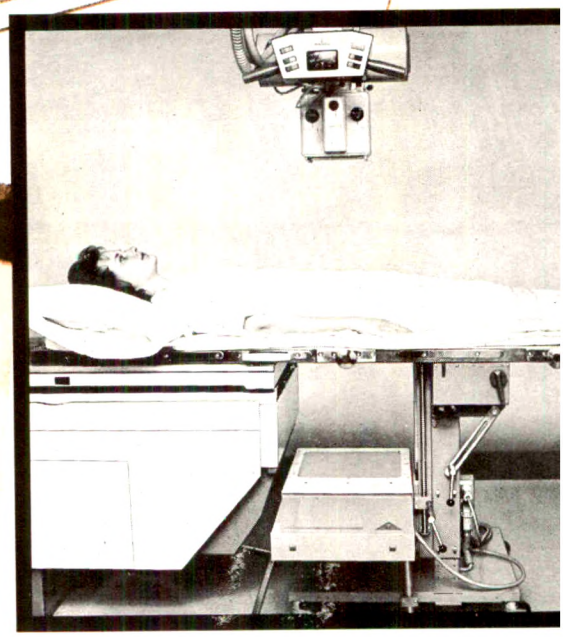
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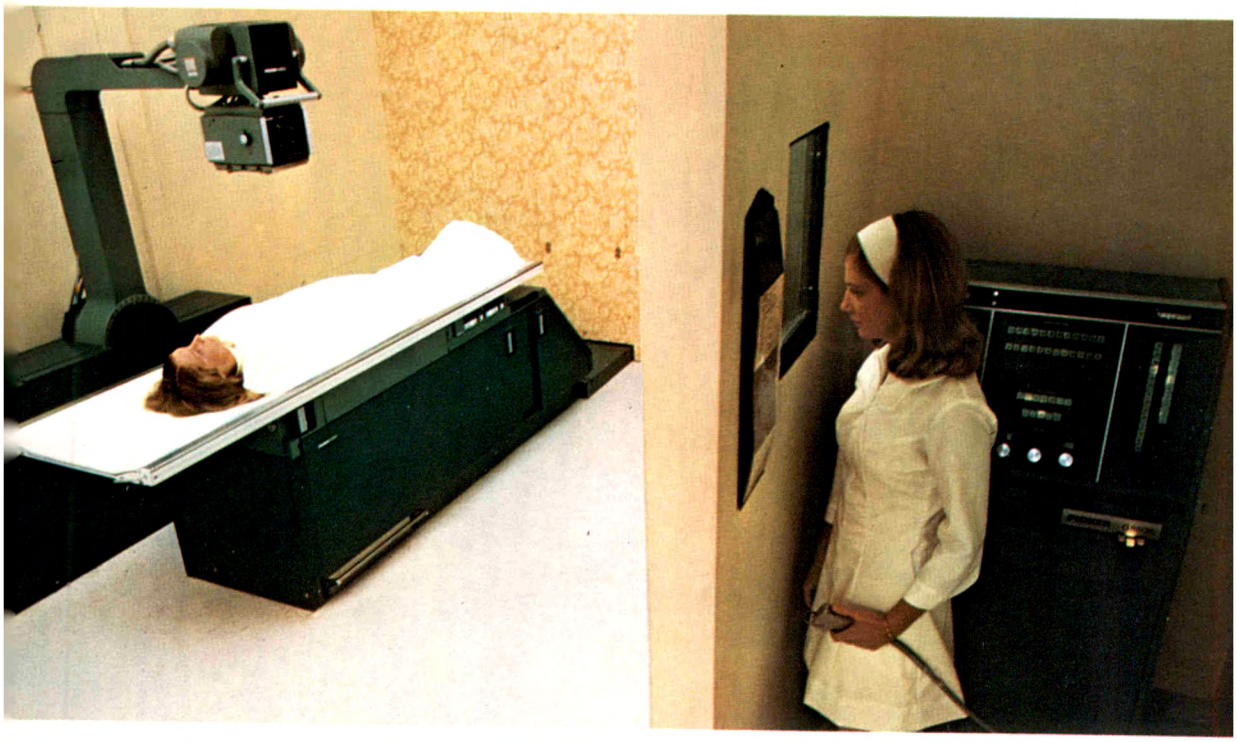
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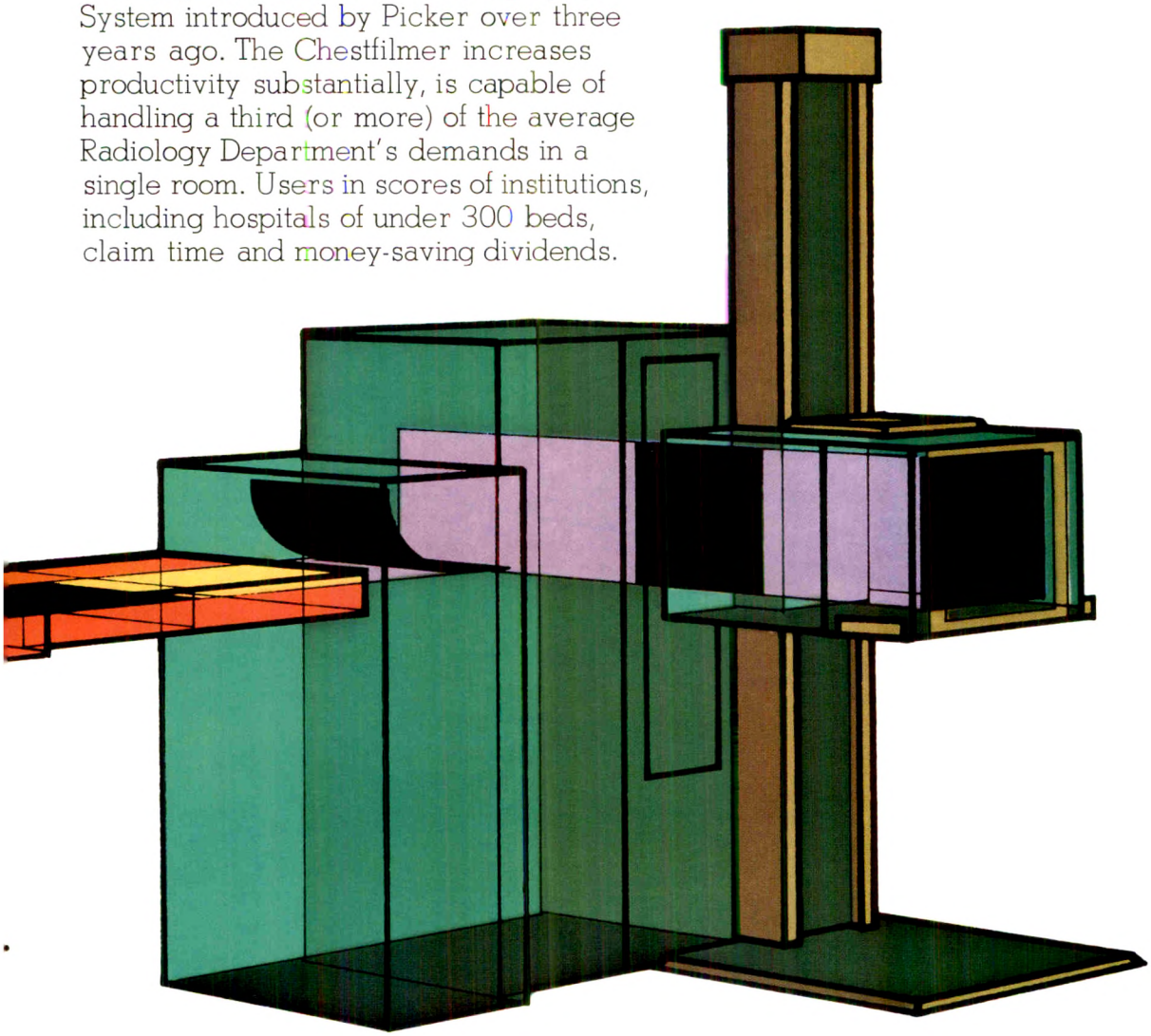
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Radiologist retrieves film from Diplomat processor for viewing within an average time, from exposure, of about 120 seconds, depending on length of conveying system. Improved diagnostic image quality with Cassetteless

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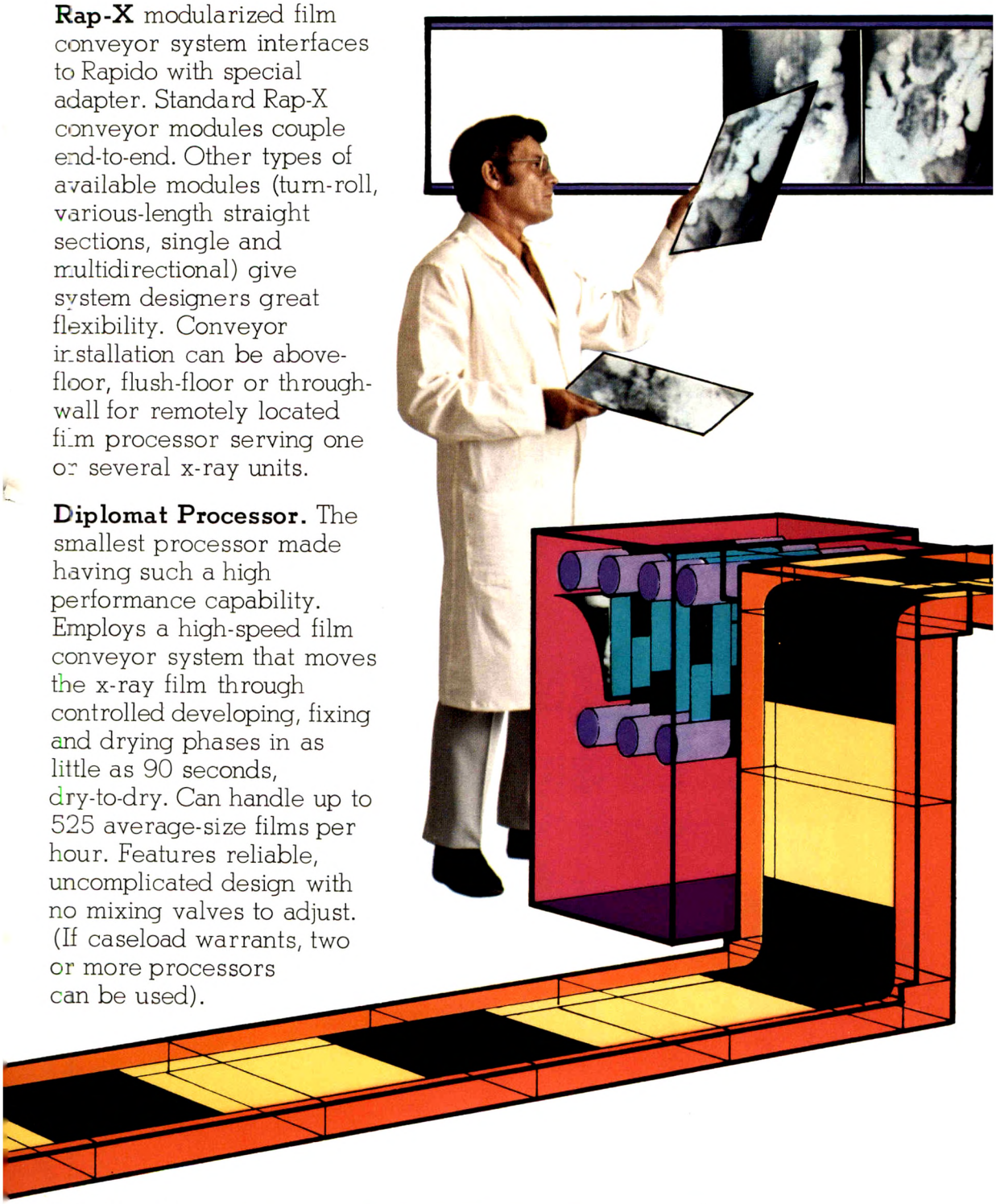
A picture of productivity. Ten patients can be x-rayed within a 15-minute time span by the Chestfilmer System, or in 20 minutes with Rapido.



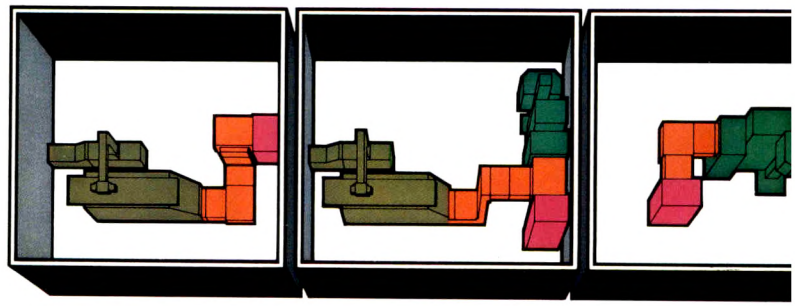


Rap-X modularized film conveyor system interfaces to Rapido with special adapter. Standard Rap-X conveyor modules couple end-to-end. Other types of available modules (turn-roll, various-length straight sections, single and multidirectional) give system designers great flexibility. Conveyor installation can be above-floor, flush-floor or through-wall for remotely located film processor serving one or several x-ray units.

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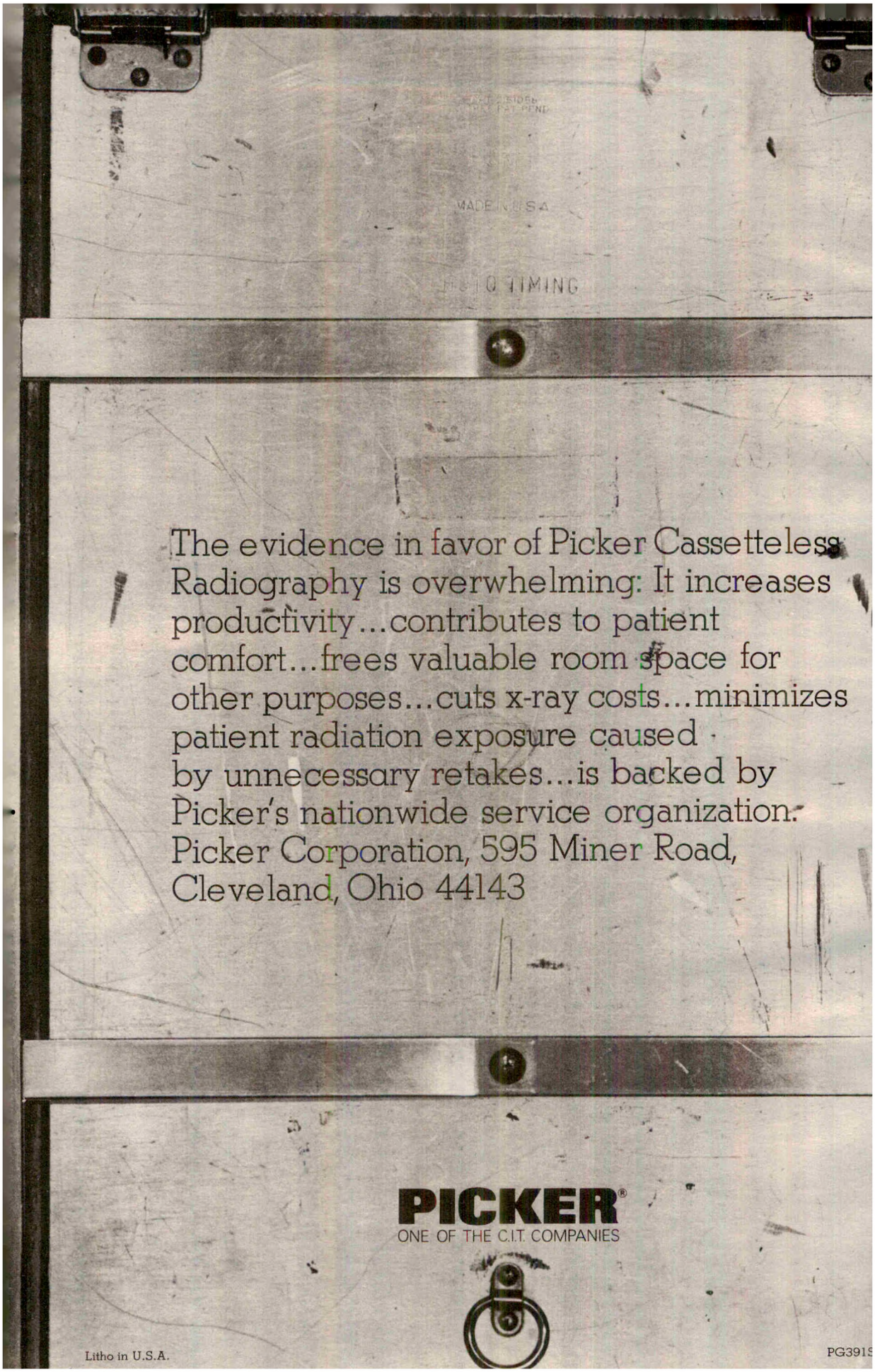


Typical cassetteless installation employing Rapido, Rap-X film conveying system and a single Diplomat processor.



Cassetteless system of Rapido and Automatic Chestfilmer with Rap-X conveyor feeding into a Diplomat processor.

Automatic Chestfilmer System with Rap-X film conveyor system and Diplomat automatic processor.



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VOL. 116 DECEMBER, 1972 NO. 4

THE PATIENT AS A PERSON

THE PRESIDENT'S ADDRESS, 1972

By J. SCOTT DUNBAR, M.D.
VANCOUVER, BRITISH COLUMBIA, CANADA

MR. CHAIRMAN, members and guests of the American Roentgen Ray Society. I am deeply conscious of the honor which has been conferred upon me by my election to the office of President of the American Roentgen Ray Society. May I express my sincere gratitude to the many dear friends and colleagues in the Society who have made this proud moment possible for me, and at the same time, may I assure you that I will do my best to be worthy of such a signal honor.

I would like to speak about "The Patient as a Person." There is a real danger that radiologists—and other doctors too—may come to regard the patient as a necessary property to be manipulated in the conduct of medical affairs, or as a package for an interesting and challenging disease, rather than as a sick human being. The reasons for this are multiple.

First, doctors must be accustomed to dealing with disease without becoming emotionally involved; if it were not so, if they were to become preoccupied with the human misery which accompanies illness, they might be unable to function efficiently and to exercise judgement in a necessary and appropriate manner.

Second, radiologists and other physicians become engrossed in the organizational and administrative aspects of their tasks, in "keeping patients moving" and "getting reports out," so that the individual human beings who appear in radiologic departments are in danger of becoming faceless objects in a production line rather than individual persons seeking attention.

Third, the teacher of Medicine, not excluding Radiology, if he is a good teacher, must be enthusiastic, and in his enthusiasm may develop in himself and impart to those he teaches a distorted view of the patient. He becomes so interested in the correct diagnosis and how to arrive at it, that he loses sight of what the disease does to a person. We radiologists frequently speak of good "case material" and of individual examples of the causes of human misery as "a beautiful case," a "good case," a "fascinating case" or even a "cute case." This tendency is accentuated by the film reading panel where experts engage in friendly competition in which diseases and radiographs are manipulated and toyed with like inanimate pieces on a chess board. Indeed, the expert performer and showman can carry his audience with him in this sort of

exercise in gamesmanship, so that to refer to a malignant lesion of bone as "our old friend osteogenic sarcoma" does not strike anyone in the room as strange behavior.

Fourth, it is well to remind ourselves of a rather harsh socio-economic fact which probably contributes to this gradual distortion of the philosophy of the physician. Doctors, including those of us who specialize in Radiology, get superb medical care for themselves and their families. We know how to seek out the most expert and sympathetic physicians and surgeons among our colleagues. We don't have to wait through weeks of anxiety for an appointment. We don't have to sit on hard benches in strange health facilities to be cared for by indifferent or aloof health personnel. It is possible, therefore, that even when a physician is sick himself, he does not gain sufficient insight into what it is like to be a patient, and to have radiologists and other physicians interested in his disease in a detached or academic fashion, but not interested in himself as a person. Perhaps each of us should go through the experience of being a patient in a busy, overcrowded Outpatient Department or stay in a shoddy, understaffed hospital ward once in a while. To put the position more positively and constructively, perhaps we can learn from the thoughtful, prompt and considerate care we get from our colleagues and from nurses and paramedical personnel, how every patient should be treated.

If it be true that there is a tendency for us to become merely academicians collecting diseases, or professionals making a living, is there anything we can do to improve our own conduct or to set a better example for our students, our residents or our paramedical assistants?

I submit that the manner in which we speak of patients, to patients, and in the presence of patients could be improved. I further submit that to be a good diagnostician, and a good teacher, is not incompatible with being a compassionate physician.

How we speak of patients and their diseases in the presence of our colleagues and our students can perhaps be modified just a little. Instead of the inevitable reference to a "beautiful case," perhaps one can occasionally refer to a "good demonstration of a bad disease."

In speaking to patients, the key concepts, I would suggest, are respect and compassion. The good physician must remember to treat each patient as an individual, and in so doing, to try to set an example to his colleagues, students and assistants. It is worth mentioning in this context that in being shown through a Radiology Department or office, one immediately gets an idea of the extent to which the radiologist respects the feelings of the patients entrusted to his care. If I take a colleague through a radiographic room in which there is a patient lying on the table, without pausing for a moment to say something to the patient—if I just ignore him—I convey a feeling that the patient is there as a piece of merchandise going through the departmental system rather than a human being deserving my respect and consideration.

There is an obvious, but often unfulfilled, need to make the patient comfortable and to reassure him while he is undergoing radiologic examination or treatment. The planning of Radiology Departments usually, but not invariably, is attentive to this need. Provision is usually made for cheerful, comfortable and well ventilated surroundings for the waiting areas and the examination areas—but not always. One would not have to look too far in Canada or the United States for health facilities having hard benches for their patients and soft seats for the staff, dirty linoleum floors for the patient examination areas, and clean rugs in the physicians' offices. It is instructive, and sometimes startling, to stand in the doorway of a radiographic room and ask oneself whether one would be willing to register his family at a motel maintained in the condition which has become habitual in this area set aside for the care of sick people.

If a hospital or department of radiology

is told that a Very Important Person is to arrive on a specified date, it is not surprising that a considerable effort is made to make the place look as clean, cheerful, comfortable and as welcoming as possible. One way of changing our attitude about patients and diseases just a little is to think of this "Very Important Person" as the individual patient who comes for radiologic examination or treatment to an office or hospital. He is, or should be, the only really important person in the entire institution. He is, or should be, the focus of our attention, our plans, and our hopes. He is our client, and the raison d'être for our existence as physicians. We are there to be useful to him, to help him, not *vice versa*. The Radiologist, the Resident, the Hospital Administrator, the Hospital Trustee, should think of him as the object of their endeavors, *the* person they are trying to please and to help, not *an* object being carried along the production line.

This patient, this case, this diagnostic challenge, is a person, a human being with a mixture of fears, hopes, misery, trust in the doctors, and worries about himself; is not just a unit or package in a list of "interesting case material."

We are fortunate in our association with clerical staff, technologists, and residents who are, for the most part, thoughtful, gentle and dedicated people. The radiologist himself, however, can have a profound influence on the workings of the entire hospital department or office by the manner in which he speaks of patients, to patients, and in the presence of patients. Efficiency, economy, enthusiasm for learning and teaching, are not incompatible with the provision of prompt, expert and above all, compassionate care for *the patient as a person*.

University of British Columbia
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Canada

"COLUMN OF BERTIN": DIAGNOSIS BY NEPHROTOMOGRAPHY*

By WILLIAM M. GREEN, M.D., BARRY D. PRESSMAN, M.D., BRUCE L. McCLENNAN, M.D., *and* WILLIAM J. CASARELLA, M.D.
NEW YORK, NEW YORK

A CONSIDERABLE literature exists concerning the radiographic diagnosis and distinction of renal masses. The roles of the excretory urogram, nephrotomogram, retrograde pyelogram, arteriogram and ultrasonogram have all been well described in distinguishing normal structures from cyst, tumor or abscess. Particularly important, in view of the obligation to minimize unnecessary nephrectomy, have been recent discussions calling attention to a variety of "pseudotumors."[3,8,9,11–13,16–20]

One such entity which has received scant attention until recently is the mass effect produced by invaginated cortical tissue, the "column of Bertin." As emphasized by Hartman and Hodson,[13] this is probably a misnomer. Bertin in 1744[4] (Fig. 1, *A–C*) used the word "cloison" to describe the normal septum or partition of cortical tissue extending between papillae. This was apparently mistranslated from "cloison" to "columnae" to column which, while perhaps inappropriate to describe the septum originally noted by Bertin, conjures up an image of a more substantial mass of renal tissue. Since it becomes important as a radiographic entity only when it is indeed a mass, and since by virtue of time and usage it is accepted, the term column probably should be retained.

Developmentally the metanephrogenic blastema is induced to form renal cortex by the invasion of the ascending ureteric buds. The cortex both overlies the bases of the pyramids and interdigitates between the pyramids toward the hilum. This normal cortical tissue extending centrally between the papillae of the medullary pyramids is designated the column of Bertin.

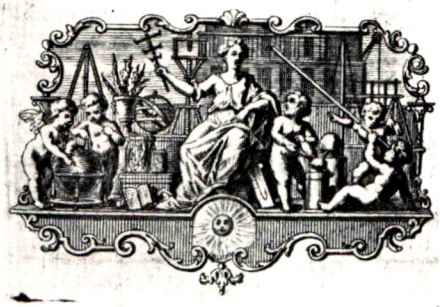
The mechanism for formation of a column of Bertin or enlarged cortical partition has been described by Hartman and Hodson.[13] It is common to find a column when a duplication anomaly exists—partial or complete. The overlapping and junction of an abortive upper pole of one moiety of a duplicated system with the lower pole of the other gives the characteristic pattern. Excessive infolding of normal cortex during development as described by King *et al.*[16] and Lopez[17] is a similar theory which also explains so-called "renal dysmorphism."[9] The column or partition of Bertin is a normal part of the renal architecture. When unusually prominent, for whatever reason, it should be appropriately recognized (Fig. 2).

RADIOGRAPHIC CRITERIA

A column of Bertin becomes radiographically, and thus clinically, significant when it simulates an intrarenal mass on the excretory urogram. Classically, it is of the same or greater density than the surrounding renal cortex and is situated at the junction of the upper and middle thirds of a kidney with some form of duplication anomaly. The calyces and infundibula are often displaced, thus creating a mass effect. The nephrotomographic phase reveals a discernible, circumscribed blush. On arteriography the interlobar arteries are displaced in a curvilinear manner around the mass. The arcuate arteries are normal. There are no "tumor vessels," puddling of contrast material, or rapid venous filling. It has been said, and apparently accepted, that these findings are characteristic and diagnostic. Furthermore, it has been emphasized that arteriography must be employed to make the diagnosis.[12,13,16,17] Ultra-

* Presented at the Seventy-third Annual Meeting of the American Roentgen Ray Society, Washington, D. C., October 3–6, 1972.
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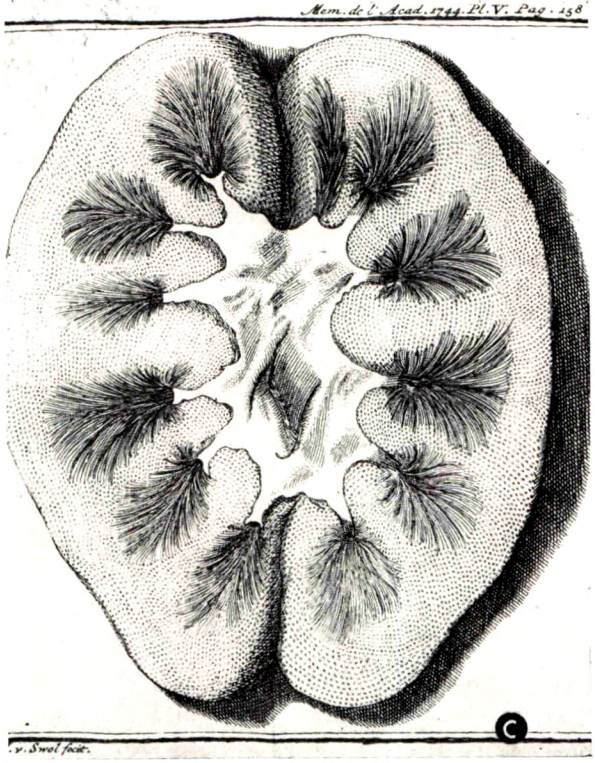
HISTOIRE
DE
L'ACADEMIE
ROYALE
DES SCIENCES.
ANNE'E M. DCCXLIV.
Avec les Mémoires de Mathématique & de Physique, pour la même Année,
Tirés des Regiſtres de cette Académie.



A AMSTERDAM,
Chez PIERRE MORTIER.
M. DCCLI.

Avec Privilège de N. S. les Etats de Hollande & de West-Frise

Mem. de l'Acad. 1744. Pl. V. Pag. 158



108 MEMOIRES DE L'ACADEMIE ROYALE

*Pag. 77. in 4.

* (a) MEMOIRE
POUR SERVIR
A L'HISTOIRE DES REINS.

Par Mr. BERTIN.

4 Juillet 1744.

ON diſtingue ſans l'aide du microſcope & ſans injection deux ſubſtances dans le Rein, une extérieure qui ſe trouve placée ſous la membrane propre, & qui paroit d'abord qu'on enlève cette membrane, & une intérieure qui eſt cachée ſous la prémière; l'extérieure, connue ſous le nom de *corticale* ou *glanduleuſe*, enveloppe exactement l'intérieure qu'on nomme *tubuleuſe*, celle-ci ſe termine dans la ſinuoſité du rein par pluſieurs éminences qui reſſemblent à des mamelons, & qu'on appelle *papilles*. On aperçoit facilement ces deux ſubſtances, mais il n'en eſt pas de même quand il s'agit de déterminer le rapport ou la différence de l'une à l'autre. Pour y réuſſir il eſt néceſſaire de donner une hiſtoire exacte de l'une & de l'autre.

La ſubſtance corticale n'enveloppe pas ſeulement les dehors du rein (a), elle ſe plonge dans la ſinuoſité, elle s'avance entre les papilles, les

(a) Planche I.

(a) Nota Il ſera bon d'examiner les Planches, & de lire l'explication des Figures avant que de commencer la lecture du Mémoire.

FIG. 1. (*A*) Faceplate and (*B*) initial page from Bertin's treatise in 1744. (*C*) A hand drawn illustration from Bertin's article depicts normal septa of invaginating cortical tissue.

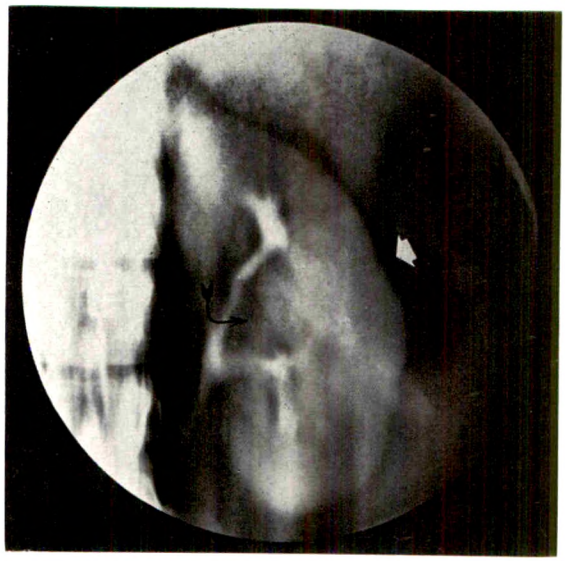


FIG. 2. Nephrotomogram demonstrates the classical appearance of a column of Bertin (black arrow) (see text), including a cortical dimple opposite the invaginated cortical tissue (white arrowhead).

sonography will show no transonic areas in these kidneys and is of little help in small (2 cm. or less) intrarenal masses. In addition to the classical findings described above, less frequently seen is a short or truncated calyx coming directly off the upper or middle pole infundibulum and ending abruptly at the medial border of the column (Fig. 3; and 5, *A–C*). A dimple on the cortical surface margin opposite the column is occasionally seen and represents the normal raphe on the surface of the kidney as described by Hodson.[15]

MATERIAL

During the period of the past year we have seen 10 cases demonstrating the characteristic findings of a column of Bertin. The first 4 were confirmed arteriographically. However, we were impressed with the fact that the findings on excretory urography and nephrotomography were so consistent and convincing in most cases that arteriography was unnecessary.

Table 1 summarizes the pertinent data in our 10 cases. Of these, 4 are presented in greater detail in the following report of cases.

REPORT OF CASES

CASE 1. (Fig. 4, *A–C*.) C.K. 135 74 59. This 53 year old female was referred for intravenous urography as part of an evaluation for right-sided back pain.

Routine urography demonstrated partial duplication of the left renal pelvis and a mass distorting the upper pole infundibulum. The question of tuberculosis *vs.* tumor was raised and nephrotomography was performed. The mass splaying the collecting systems was noted to be homogeneous functioning renal tissue somewhat denser than the contiguous cortical tissue.

Ultrasonography was normal, there being no evidence of solid or cystic mass.

Arteriography revealed 2 left renal arteries, a blush in the region of the "mass" and no abnormal vessels.

The "mass" was considered to be an invaginated column of Bertin.

Comment. Both the tomogram obtained during routine urography and the nephrotomogram demonstrated the "mass" to be contiguous with



FIG. 3. Tomogram obtained during intravenous urography demonstrates a shortened calyx arising from the middle pole infundibulum and terminating abruptly next to an invaginating column of Bertin (arrow).

TABLE I

SUMMARY OF FINDINGS IN 10 CASES

Case Age Sex	Reason for Urogram	Routine Urogram	Nephrotomogram	Arteriogram
C.M. 60 M.	Dysuria	Duplication anomaly on the right with mass effect	Homogeneous cortical blush in "mass," contiguous with cortical tissue	Cortical blush, no abnormal vessels
C.K. 52 F.	Back pain for 6 months and old Pott's disease	Duplication anomaly on the left with mass distorting upper pole calyces	Homogeneous cortical blush in "mass," contiguous with cortical tissue	Cortical blush, no abnormal vessels
B.W. 38 M.	Hesitancy	Duplication anomaly on the left with mass effect; truncated calyx arising from middle pole infundibulum	Homogeneous cortical blush in "mass," contiguous with cortical tissue	Cortical blush, no abnormal vessels
H.A. 65 M.	Pyuria	Duplication anomaly on the right with suggestion of mass	Homogeneous cortical blush in "mass," contiguous with cortical tissue	Cortical blush, no abnormal vessels
A.R. 59 M.	Preoperative evaluation for left groin neoplasm	Duplication anomaly on the right with suggestion of mass	Homogeneous cortical blush in "mass," contiguous with cortical tissue	Not performed
E.L. 73 M.	Right flank pain	Duplication anomaly on the right with mass effect	Homogeneous cortical blush in "mass," contiguous with cortical tissue	Not performed
S.S. 68 M.	Proteinuria and hematuria	Duplication anomaly on the right with mass effect	Homogeneous cortical blush in "mass," contiguous with cortical tissue	Not performed
U.W. 58 F.	Low back and left leg pain	Duplication anomaly on the left with mass displacing upper and lower pole calyces	Homogeneous cortical blush in "mass," contiguous with cortical tissue	Not performed
A.A. 51 F.	Pyelonephritis	Duplication anomaly on the left with mass effect on upper infundibulum; truncated calyx arising from middle infundibulum	Homogeneous cortical blush in "mass," contiguous with cortical tissue; cortical dimple opposite mass	Not performed
J.C. 31 F.	Hypertension	Duplication anomaly with mass effect	Homogeneous cortical blush in "mass," contiguous with cortical tissue; cortical dimple opposite mass	Not performed

cortical tissue. On the nephrotomogram a blush was seen and homogeneity of the region was well shown. These findings, along with the location of the "mass" at the junction of the middle and upper thirds of the kidney with partial or complete duplication, are classical for an "aberrant" column of Bertin. We now feel that these findings are sufficient to make the correct diagnosis without resorting to arteriography.

CASE II. (Fig. 5, *A–C.*) B.W. 149 22 31. In-

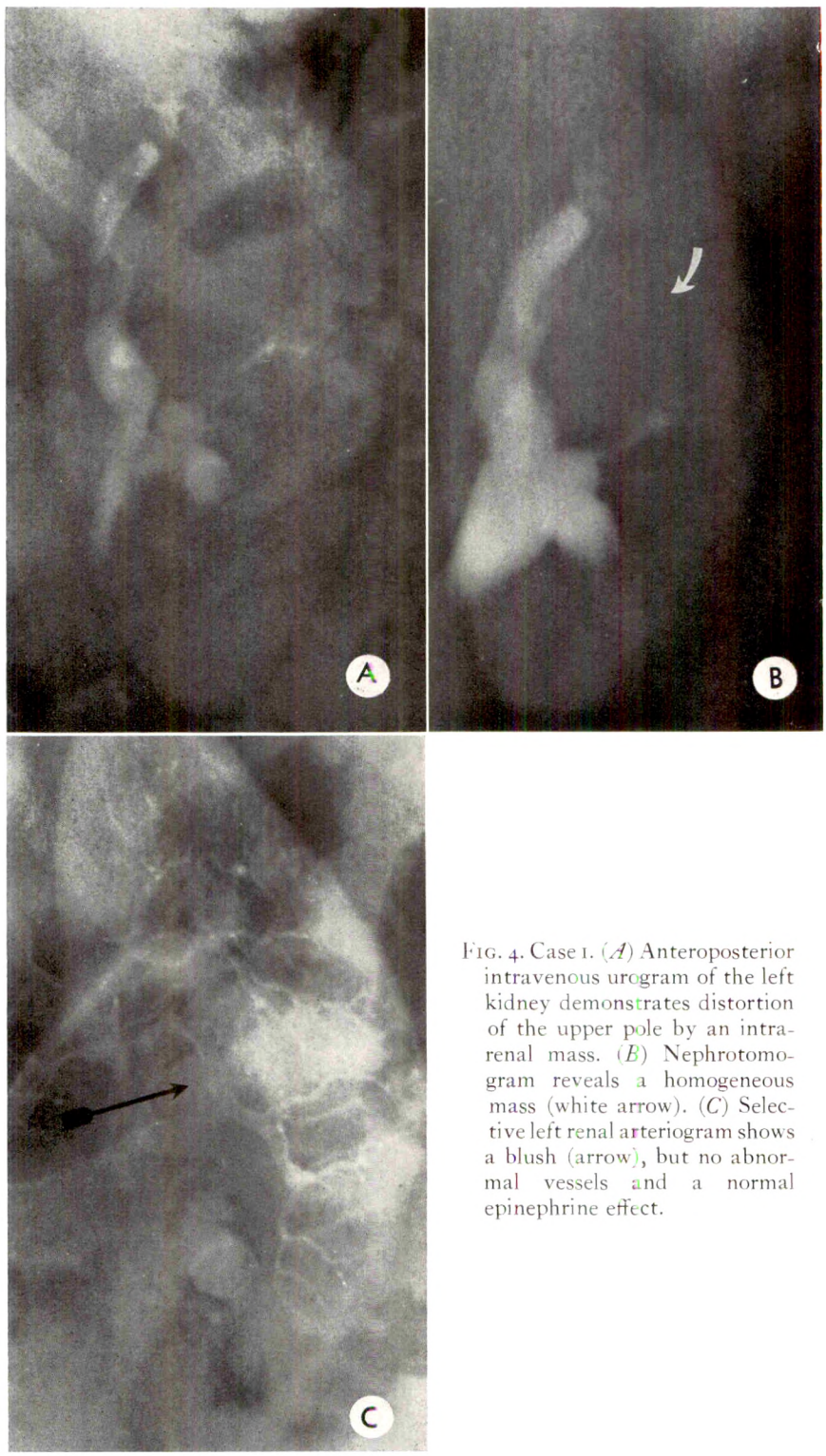


FIG. 4. Case 1. (*A*) Anteroposterior intravenous urogram of the left kidney demonstrates distortion of the upper pole by an intrarenal mass. (*B*) Nephrotomogram reveals a homogeneous mass (white arrow). (*C*) Selective left renal arteriogram shows a blush (arrow), but no abnormal vessels and a normal epinephrine effect.

travenous urography was performed in this 37 year old male who was experiencing difficulty in initiating urination. The routine examination demonstrated duplication of the left collecting system and suggested a renal mass.

Accordingly, nephrotomography was under-

taken. This demonstrated a cortical blush in the region of the mass.

Arteriography, including epinephrine studies, was diagnostic of invaginated column of Bertin in that there was splaying of the interlobar vessels, a homogeneous cortical blush, and no neovascularity.

Comment. This case demonstrates another feature sometimes seen in the presence of an invaginated column of Bertin; *i.e.*, a truncated calyx arising directly from the middle pole infundibulum. As in Case 1, arteriography was performed to clarify the diagnosis, but in retrospect this was unnecessary. The mass was at the junction of the middle and upper thirds of a kidney with a duplicated collecting system;

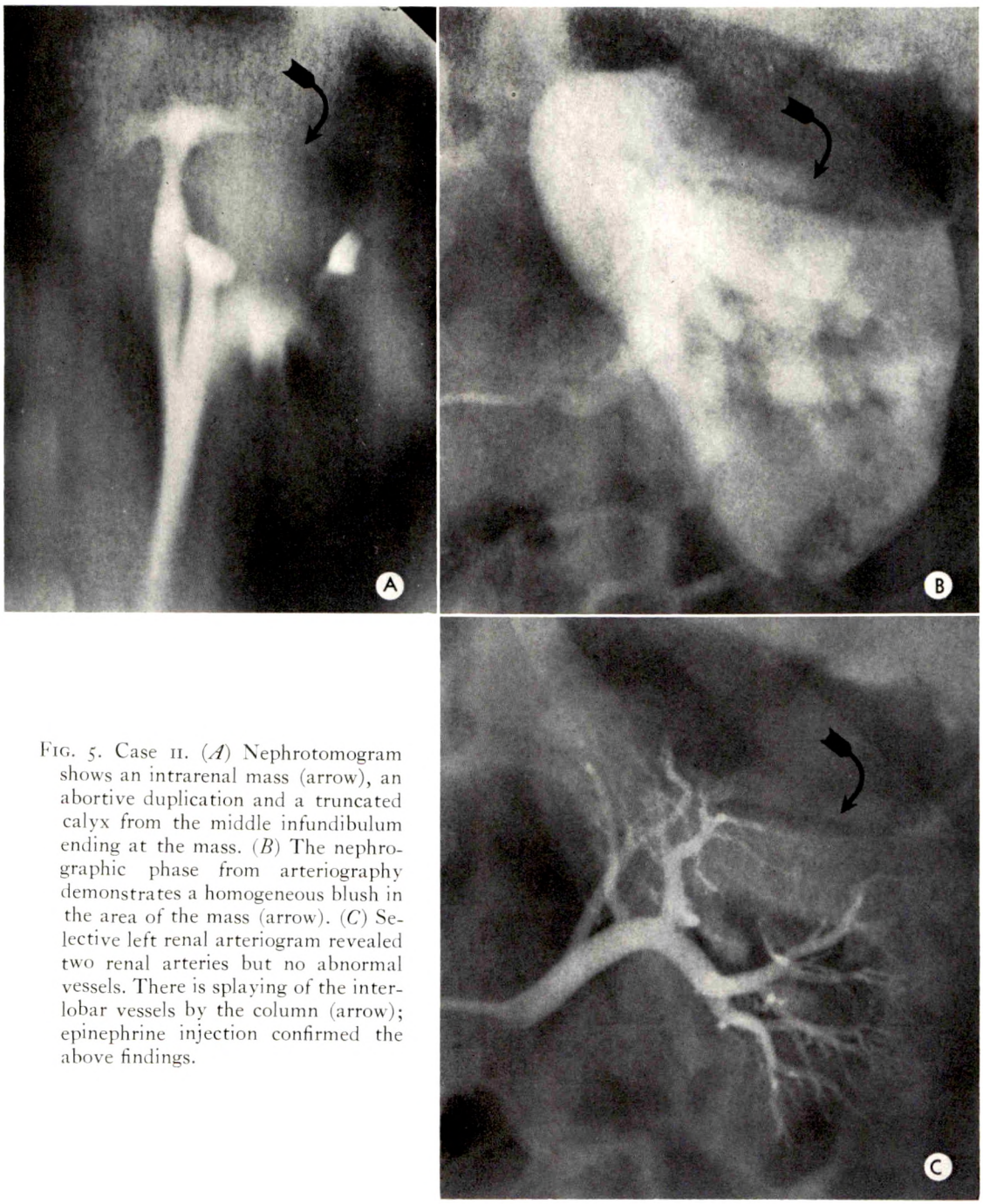


Fig. 5. Case 11. (*A*) Nephrotomogram shows an intrarenal mass (arrow), an abortive duplication and a truncated calyx from the middle infundibulum ending at the mass. (*B*) The nephrographic phase from arteriography demonstrates a homogeneous blush in the area of the mass (arrow). (*C*) Selective left renal arteriogram revealed two renal arteries but no abnormal vessels. There is splaying of the interlobar vessels by the column (arrow); epinephrine injection confirmed the above findings.

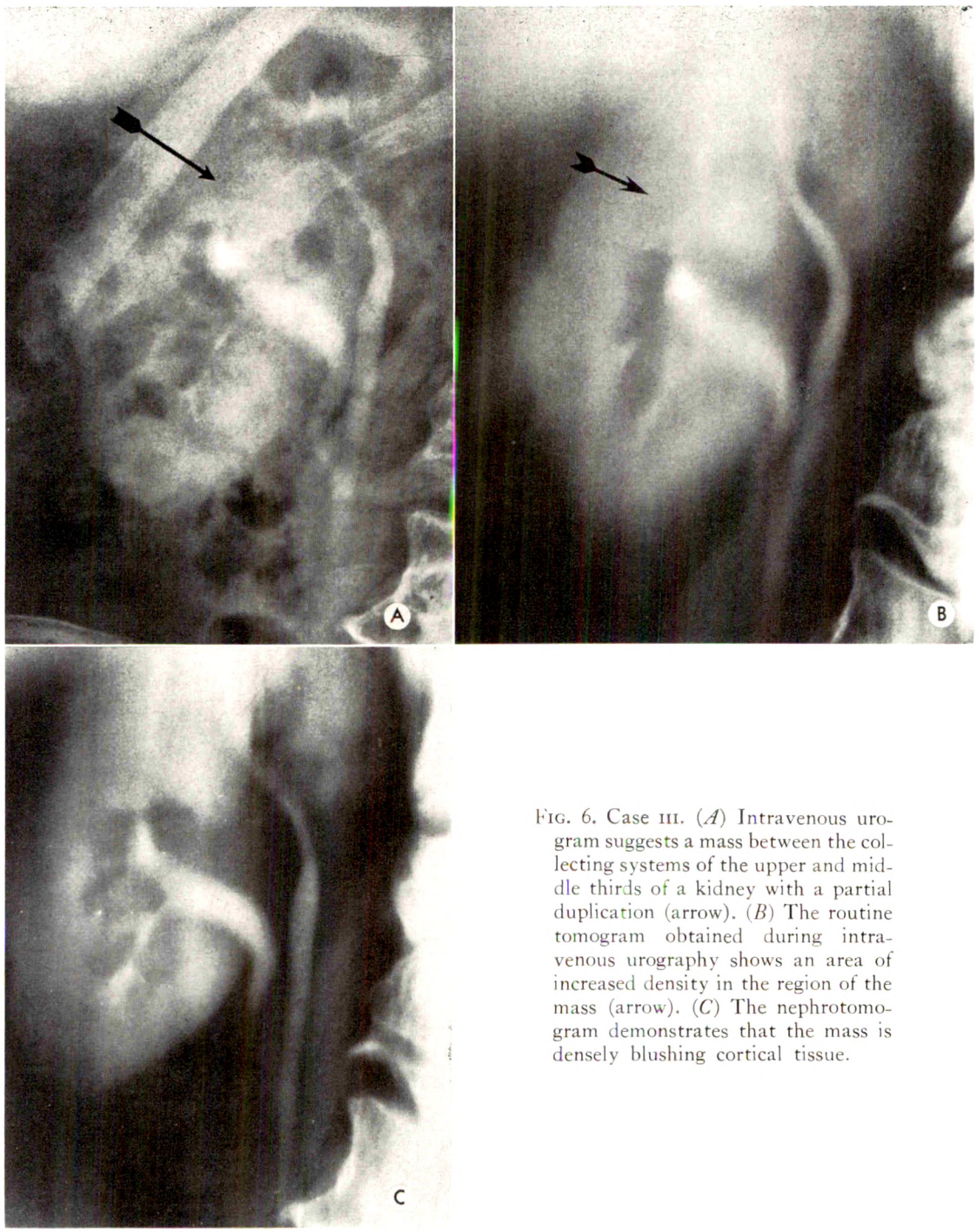


FIG. 6. Case III. (*A*) Intravenous urogram suggests a mass between the collecting systems of the upper and middle thirds of a kidney with a partial duplication (arrow). (*B*) The routine tomogram obtained during intravenous urography shows an area of increased density in the region of the mass (arrow). (*C*) The nephrotomogram demonstrates that the mass is densely blushing cortical tissue.

a cortical blush was present; the tissue was homogeneous and contiguous with cortical tissue; and finally a truncated calyx emanated from the middle pole infundibulum.

CASE III. (Fig. 6, *A–C.*) E.L. 077 81 95. Recurrent right lower quadrant and low back pain was the indication for urography in this 74 year old male. There was duplication of the right collecting system and the upper pole infundibulum was displaced slightly medially raising the question of a mass.

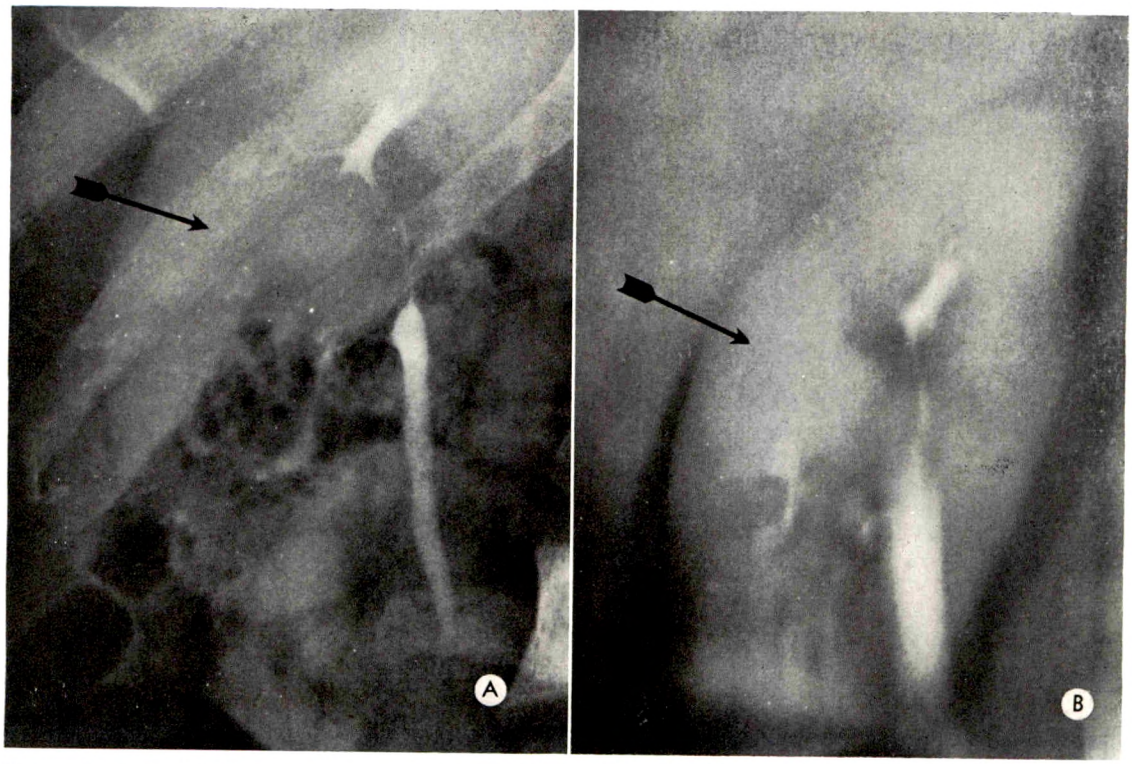


FIG. 7. Case IV. (*A*) Intravenous urogram shows distortion of the upper and middle collecting systems on the right by a mass (arrow). (*B*) Nephrotomogram demonstrates this to be a column of Bertin (arrow).

On nephrotomography this "mass" was seen to be contiguous with the overlying cortex, to have a dense blush and to be homogeneous. Accordingly, the diagnosis of invaginated column of Bertin was made and no further evaluation was undertaken in this regard.

Comment. Because of our previous experience as outlined above and in Table I, we felt that the diagnosis was secure on the basis of the urographic and nephrotomographic findings. Urinalysis was normal as were the laboratory studies and hematocrit. The "mass" was in the classical location in a kidney with a duplicated collecting system, was homogeneous with a dense blush on nephrotomography, and was contiguous with the cortex.

CASE IV. (Fig. 7, *A* and *B*.) A.R. 210 08 50. This 59 year old male was admitted for evaluation of an enlarging mass in the left groin. Preoperatively, intravenous urography was performed to determine if there was any pelvic extension of tumor. There was abortive duplication of the right pelvis with a mass effect between the upper and middle pole infundibula.

Nephrotomography clearly demonstrated a homogeneous cortical blush, and an invaginated column of Bertin was diagnosed without further renal evaluation.

Comment. The classical criteria of an invaginated column of Bertin were present in this case as demonstrated on intravenous urography and nephrotomography. Accordingly, no further renal work-up was deemed necessary, particularly in the presence of a normal urinalysis and no urinary tract symptoms.

DISCUSSION

Our routine excretory urogram includes tomographic sections of both kidneys during the initial 10 minute period, after abdominal compression is applied. This markedly enhances the renal outlines, highlights differences in homogeneity of cortical tissue and clarifies distortion of normal architecture. Should the excretory urogram suggest an abnormality in cortical mantle, nephrotomography is undertaken on a subsequent day.

Nephrotomography is performed by the

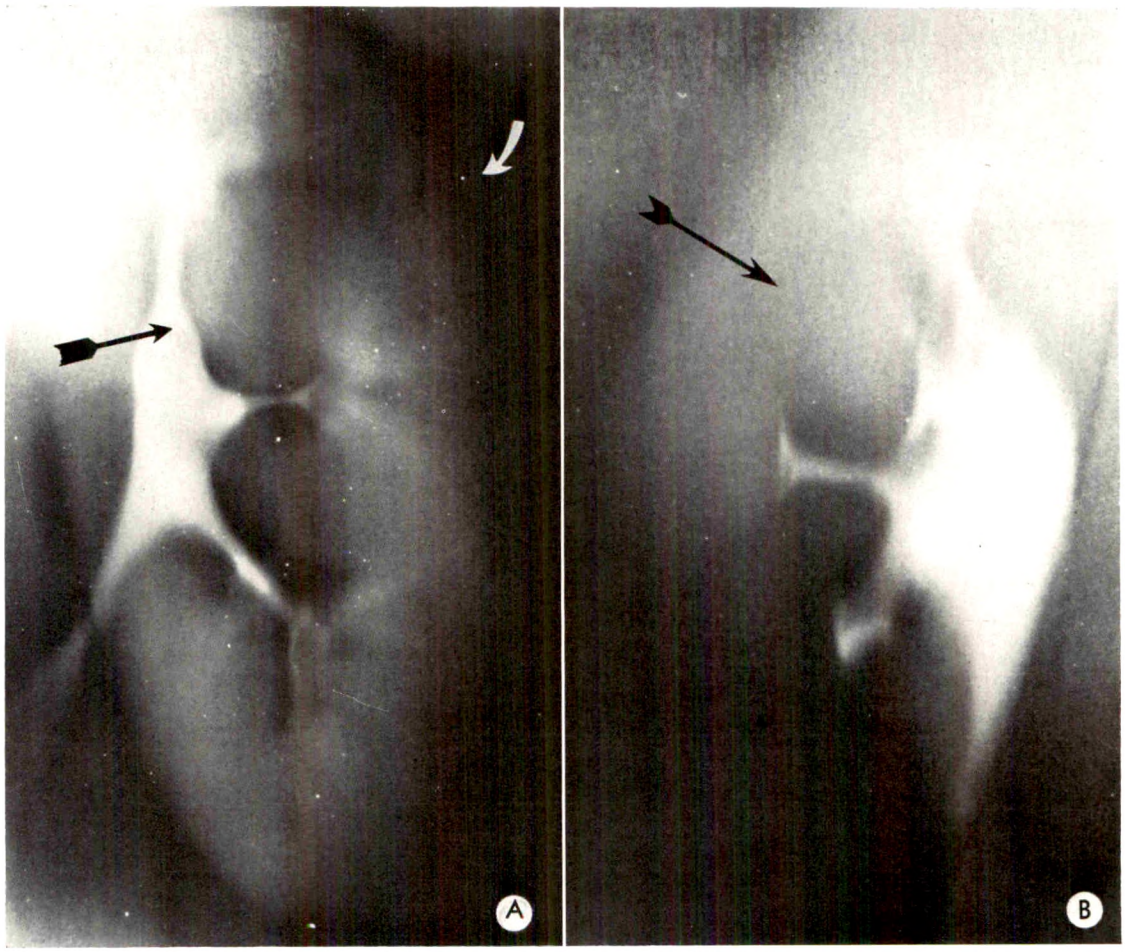


FIG. 8. (*A* and *B*) Nephrotomograms in 2 patients without duplication, each of which demonstrates a homogeneous area of tissue contiguous with the cortex which has the appearance of an upper pole mass (black arrows). A cortical dimple is present opposite the mass in *A* (white arrow). A column of Bertin is causing this appearance and should be recognized as such.

drip infusion technique using 150 cc. of hypaque M-90* mixed with an equal amount of dextrose and water. This is infused over approximately 10 minutes and tomographic sections are obtained in 2 projections. Iodine concentrations of this magnitude are necessary for adequate nephrotomographic diagnosis. We have had a high degree of success with this procedure since 1963.

On occasion, excretory urography with routine tomography may demonstrate all the classical criteria for a column of Bertin. However secure the diagnosis may seem, it is recommended that nephrotomography be performed to substantiate the findings.

* Winthrop Laboratories.

As in all radiologic and clinical entities there are cases which do not demonstrate every one of the characteristic criteria of the entity, but which so much resemble it, that other diagnoses seem unlikely. For example, there are cases without renal duplication which meet all the other criteria (Fig. 8, *A* and *B*). In these cases a column is strongly suggested. Should these localized increases in renal cortex occur at sites other than the junction of the upper and middle pole of a partially duplicated kidney, further evaluation including arteriography is warranted.

We are aware that it may be possible for a renal malignancy to present with a similar appearance. However, in a duplicated

kidney with all other criteria satisfied, this not only is rather remote, but we have been unable to locate such a case in the literature.

All previous work, while noting the features of the column of Bertin on excretory urography and nephrotomography, stressed that the diagnosis could only be made arteriographically. In view of the findings we consider characteristic on excretory urography and nephrotomography, the not uncommon nature of the entity, and the cost of radiologic overkill in the work-up of a benign process, our conviction is that in most cases the diagnosis can be made on excretory urography and nephrotomography alone.

SUMMARY

The column of Bertin originally described in 1744 is now a common uroradiologic finding which becomes radiographically and thus clinically significant when it simulates an intrarenal mass on the excretory urogram.

Nephrotomography is a definitive diagnostic study when the following features are seen:

1. Partial or complete renal duplication
2. A mass of homogeneous density contiguous with the renal cortex between the upper and middle pole infundibula
3. The mass density is equal to or greater than the surrounding parenchyma.

Supporting signs are:

1. A dimple on the renal margin opposite the column
2. A short or truncated calyx coming directly off the upper or middle pole infundibulum, ending at the mass.

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We would like to thank Dr. Walter E. Berdon and Dr. Kent Ellis for stimulating our interest in this entity.

REFERENCES

1. Allen, A. C. The Kidney: Medical and Surgical Diseases. Grune & Stratton, Inc., New York, 1962, pp. 11.
2. Arey, L. B. Developmental Anatomy. W. B. Saunders Company, Philadelphia, 1954, pp. 301.
3. Becker, J. A., Fleming, R., Kanter, I., and Melicow, M. M. Misleading appearances in renal angiography. *Radiology*, 1967, *88*, 691–700.
4. Bertin, R. Mémoire pour servir à l'histoire des reins. In: Histoire de L'Académie Royale des Sciences, July 1744, 79, 108–159.
5. Boijsen, E. Angiographic studies of anatomy of single and multiple renal arteries. *Acta radiol.*, 1959, Suppl. 183, 1–135.
6. Campbell, M. Clinical Pediatric Urology. W. B. Saunders Company, Philadelphia, 1951, pp. 165.
7. Campbell, M., and Harrison, J. H. Urology. W. B. Saunders Company, Philadelphia, 1970, pp. 1393–1395.
8. Caplan, L. H., Siegelman, S. S., and Bosniak, M. A. Angiography in inflammatory space-occupying lesions of kidney. *Radiology*, 1967, *88*, 14–23.
9. Charghi, A., Dessureault, P., Drouin, G., Gauthier, G. E., Perras, P., Roy, P., and Charbonneau, J. Malposition of renal lobe (lobar dysmorphism): condition simulating renal tumor. *J. Urol.*, 1971, *105*, 326–329.
10. Dees, J. E. Clinical importance of congenital anomalies of upper urinary tract. *J. Urol.*, 1941, *46*, 659–666.
11. Felson, B., and Moskowitz, M. Renal pseudotumors: the regenerated nodule and other lumps, bumps and dromedary humps. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *107*, 720–729.
12. Gooding, C. A. Childhood renal pseudotumor. *Radiology*, 1971, *98*, 79–80.
13. Hartman, G. W., and Hodson, C. J. Duplex kidney and related abnormalities. *Clin. Radiol.*, 1969, *20*, 387–400.
14. Herbut, P. A. Urologic Pathology. Lea & Febiger, Philadelphia, 1952, pp. 464.
15. Hodson, C. J. Personal communication.
16. King, M. C., Friedenberg, R. M., and Tena, L. B. Normal renal parenchyma simulating tumor. *Radiology*, 1958, *91*, 217–222.
17. Lopez, F. A. Renal pseudotumors. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *109*, 172–184.
18. Salmon, R. B., and Koehler, P. R. Angiography in renal and perirenal inflammatory masses: report of three cases. *Radiology*, 1967, *88*, 9–13.
19. Simon, H. Renal pseudotumor: case report. *J. Urol.*, 1970, *104*, 45.
20. Sukthomya, C., and Levin, B. Pseudotumors of kidney secondary to anticoagulant therapy. *Radiology*, 1967, *88*, 701–703.

ROENTGENOGRAPHIC CHANGES AFTER PYELOPLASTY*

By HOWARD J. MINDELL, M.D.
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URETEROPELVIC junction (UPJ) obstruction has been considered by far the commonest congenital anomaly of the urinary tract.[7] Although pyeloplasty is the usual procedure employed in the surgical approach to this problem, there is little material in the radiologic literature[7] regarding urographic changes after pyeloplastic surgery.

Consideration of the roentgen changes after pyeloplasty is of further interest in view of Shopfner's work which casts doubt on the validity of organic UPJ obstruction as a concept. Shopfner[14] has stated that the pathogenesis is the same in the nonobstructive and secondary obstructive types of renal pelvic dilatation, and that primary obstructive ureteropelvic dilatation is rare. Evaluation of the problem is also complicated by the large number of pyeloplastic procedures devised since the original operations of Kuster[11] and Fenger.[8] Culp and DeWeerd[4] noted that obstruction at the UPJ has been too variable, complex and elusive to permit any one type of plastic repair.

Assessment of the surgical success has been difficult, and the role of postoperative urography has been controversial. Eberhart and Reiser[6] stated that the postoperative pyelogram may be "pictorial and dramatic" but not the "entire story," and often of minor importance. They concluded that the final evaluation must be made on a less spectacular and less tangible basis, citing good pyeloplastic results in their own hands regardless of the findings of postoperative pyelography. Similarly, Williams[16] cautioned to not expect "perfect radiographic results." Burns *et al.*,[2] on the other hand, noted that although return to normal is seldom anticipated, in most instances the pyelogram will show some improvement, and in fact all cases showing good pyelographic results were symptomatically improved. Gibson[9] insisted that no one is justified in citing end results (pyeloplasty) as successful without urographic evidence. Sargent[13] further noted that even with very severe hydronephrosis, one may be "startled beyond belief" by the degree of postoperative involution.

Despite conflicting views on technique, postoperative evaluation, and the concept of organic UPJ obstruction itself, a few approaches to pyeloplasty have gained wide urologic acceptance.[15] Three commonly used pyeloplastic procedures are diagrammatically outlined in Figures 1, 2, and 3.

After analysis of the experiences at the Medical Center Hospital of Vermont, Burlington, and review of the literature, some aspects of the urographic changes after pyeloplasty are assessed. An attempt is made to correlate the postoperative clinical and roentgenographic course; note frequency, degree, and speed of postoperative urographic resolution or change; and search for appearances which might be typical of the several operations in use.

MATERIAL

In the past 5 years 24 patients at the Medical Center Hospital of Vermont have required 29 operations for UPJ obstruction, exclusive of complex problems such as horseshoe kidney.

Patient age ranged from 2 to 74 years, including 14 males and 10 females, 2 repeat operations and 3 bilateral procedures.

The etiology of the UPJ obstruction was believed at surgery to be due in 18 patients

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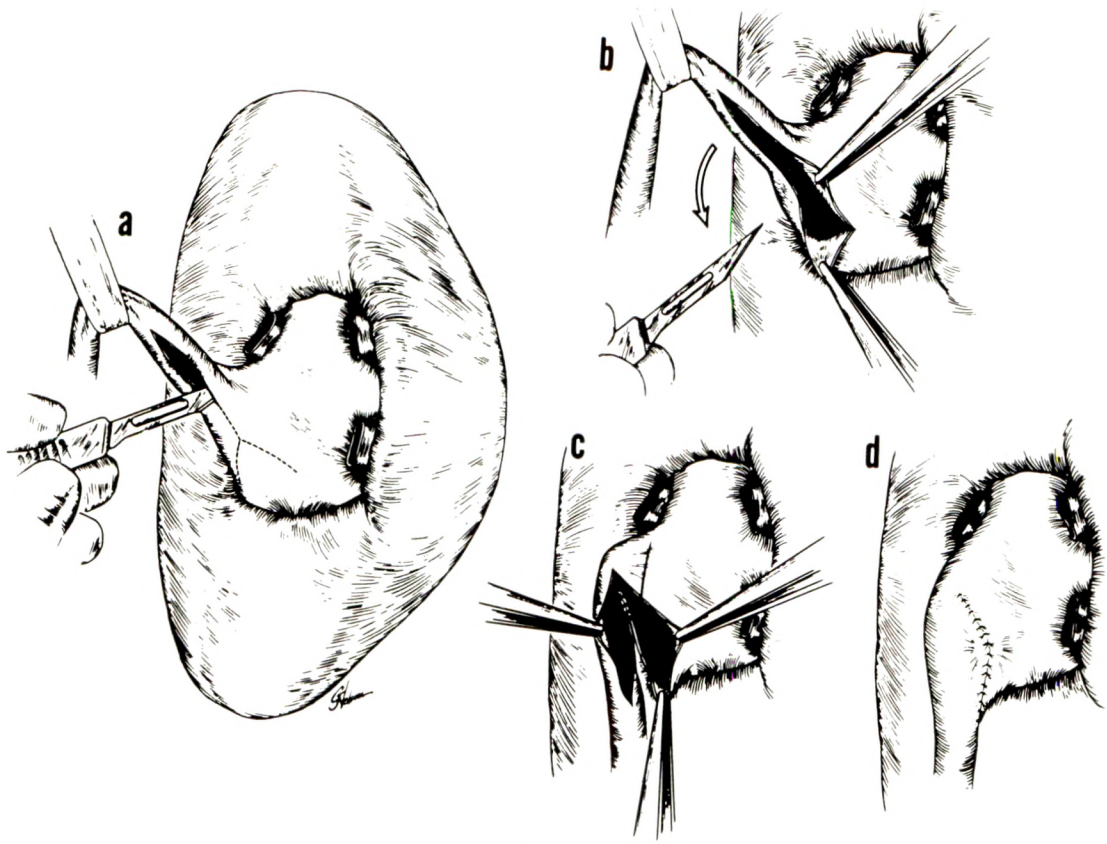


Fig. 1. (*a–d*) Foley Y–V pyeloplasty.

to intrinsic stenosis, and in 6 patients to vascular obstruction. Whether the aberrant vessels in these 6 patients crossed or gravitated over a pre-existing narrowed segment appears to be a moot point, but of significance in this regard is that simple ligation or re-orientation of the crossing vessels was never used alone as primary correctional surgery.

The operations included: intubated ureterotomy, 1; vertical flap of Culp, 2; Foley Y-V pyeloplasty, 7; and dismembered pyeloplasty of Anderson-Hynes, 18.

In the following, illustrative case reports are briefly summarized:

REPORT OF CASES

Case I. G.C., a 17 year old male, was well until onset of right flank pain 3 days before admission. Initial examination showed right flank tenderness, urine with slight protein, 4+RBC/HPF (red blood cells/high power field), but negative for culture.

Preoperative right retrograde pyelogram (Fig. 4*a*) showed UPJ stricture, and moderately severe hydronephrosis.

Right Foley Y-V pyeloplasty was performed on the 6th hospital day.

Nephrostogram 10 days postoperatively (Fig. 4*b*) showed persistent UPJ obstruction with decreased pelvic size and minimal decrease in caliectasis. By 7 weeks the UPJ opened (Fig. 4*c*) and at 6 months (Fig. 4*d*) intravenous pyelography (IVP) showed a small pelvis with a flat medial border (frequent in Foley Y-V pyeloplasty), slight decrease in caliectasis, and improved ureteral opacification.

No recurrence of disease was present at 4 years follow-up.

Case II. J.P., a 13 year old male with acute onset of left flank pain, fever, urine with 4+RBC, 4+WBC/HPF (white blood cells/high power field), and 2+ protein.

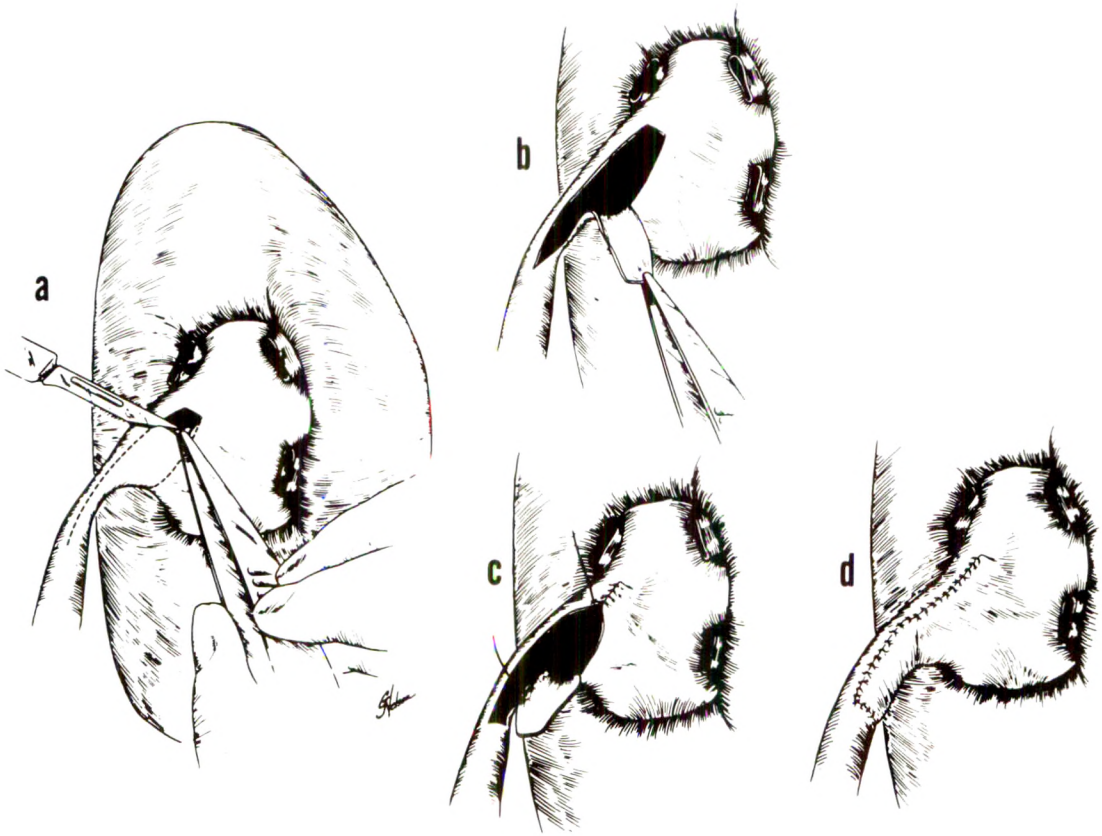


Fig. 2. (*a–d*) Culp (Scardino) vertical flap pyeloplasty.

IVP (Fig. 5*a*) showed very severe left hydronephrosis due to UPJ obstruction.

Left Foley Y-V pyeloplasty was performed.

Nephrostogram at 8 days showed less calyceal distention (Fig. 5*b*). At 6 weeks IVP showed continued improvement (Fig. 5*c*).

Patient remained well with negative urine on follow-up at 2 years postoperatively.

Case III. E.R., a 30 year old female with a 4 year history of left UPJ obstruction. Urine examination and culture were negative.

IVP (Fig. 6*a*) showed very severe left hydronephrosis due to UPJ obstruction.

Left Culp vertical flap pyeloplasty was performed.

Nephrostogram at 9 days postoperative (Fig. 6*b*) showed marked improvement with decrease in pelvic size and striking resolution of caliectasis.

Follow-up showed patient well, urine normal and no evidence of recurrent disease 3 years after surgery.

Case IV. R.G., a 17 year old male with 3 months of chills and fever. Physical examination showed a right flank mass, although urinalysis was normal.

Right retrograde pyelogram showed UPJ obstruction (Fig. 7*a*).

Dismembered Anderson-Hynes pyeloplasty was performed. Although at surgery the obstruction was thought to be due to aberrant polar vessels crossing the inferior surface of the dilated pelvis, the pathologic examination of the resected specimen showed a stricture with proximal ureteral hypertrophy (could not here and in similar cases dilated pelves herniate over vessels secondarily?).

IVP (Fig. 7*b*) 7 days postoperative still showed UPJ obstruction, but at 3 months (Fig. 7*c*) there was marked improvement, with lateral displacement of the small pelvis, and medial tilting of the cephalad portion of the pelvis (tilting due to sewing of lower pole of the kidney to psoas muscle, as done at this hospital).

Clinically the patient became well, and was discharged at 6 months.

Case v. J.R., an 11 year old male with a 7 year history of recurrent flank pain. Preoperative urine examination and culture were normal.

Pyelography (Fig. 8, *a* and *b*) showed right UPJ obstruction.

Dismembered pyeloplasty was performed with mobilization of lower pole "obstructing vessels."

Nephrostogram at 10 days showed dramatic improvement (Fig. 8*c*). IVP 4 months after surgery (Fig. 8*d*) showed a small pelvis, delicate calyces and improved ureteral opacification.

Patient is well, with normal urine at 2 years after surgery.

Case vi. T.P., a 43 year old female with acute onset of left flank pain, urine with 5–10 WBC, occasional RBC/HPF, and culture positive for gram positive cocci.

Left retrograde pyelogram (Fig. 9*a*) showed UPJ obstruction.

Dismembered pyeloplasty was done.

IVP 3 weeks after surgery (Fig. 9*b*) showed marked improvement, and characteristic changes for dismembered pyeloplasty as in Case iv.

At 2 months after operation the patient was well, and the urine was negative; she was lost to follow-up thereafter.

Case vii. M.M., a 74 year old male, developed right flank pain while in the hospital for prostatism. Urinalysis revealed 2–3 WBC, and 15–20 RBC/HPF.

IVP (Fig. 10) demonstrated right UPJ obstruction.

Dismembered pyeloplasty was done.

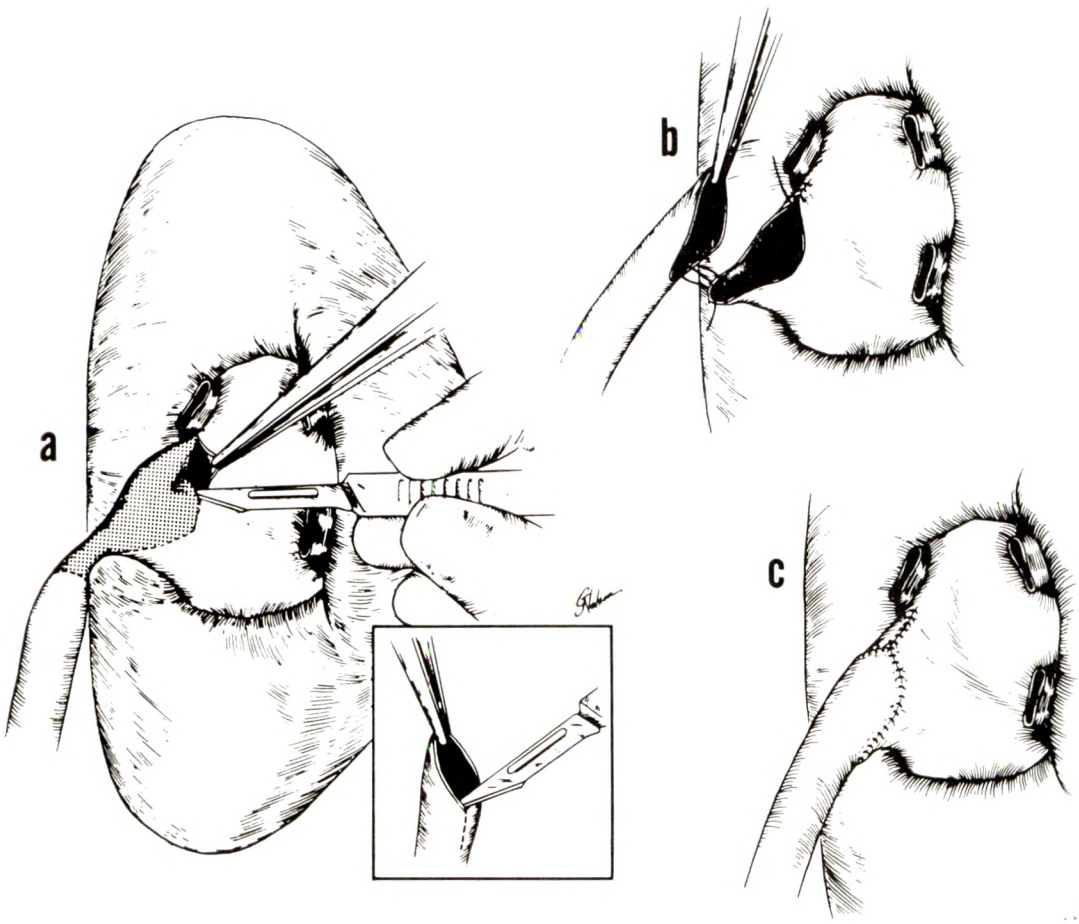


Fig. 3. (*a–c*) Dismembered (Anderson-Hynes) pyeloplasty.

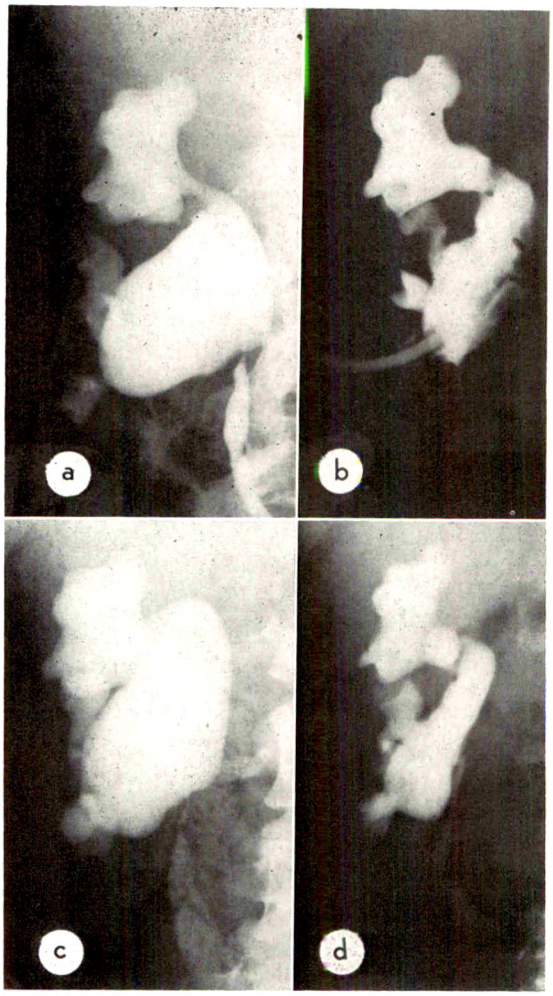


FIG. 4. Case I. (*a*) Preoperative right retrograde pyelogram showing ureteropelvic junction (UPJ) obstruction due to short stenotic segment. (*b*) Nephrostogram 10 days after Foley Y-V pyeloplasty showing slightly less caliectasis but complete obstruction at UPJ. (*c*) Intravenous pyelogram (IVP) 7 weeks postoperative showing some ureteral opacification. (*d*) IVP at 6 months showing slightly less caliectasis than in *a* and ureteral opacification. Note decrease in pelvic size and flat medial pelvic border.

At 7 days, postoperative nephrostogram (Fig. 11*a*) already showed marked calyceal decompression. Figure 11*b* shows IVP at 2 weeks after surgery. At 4 months, IVP (Fig. 11*c*) showed findings similar to those of Cases IV and VI (laterally displaced UPJ with flat medial pelvic wall and medial tilt to cephalad pelvic contour).

Patient remains well with negative urine at 2 years after surgery.

CASE VIII. L.M., a 7 year old female with a 4 year history of left flank pain. Urinalysis showed 4+WBC and occasional RBC/HPF.

Retrograde pyelography revealed left UPJ obstruction (Fig. 12*a*).

Dismembered pyeloplasty was performed with slow opening at operative site.

Nephrostogram 3 weeks after surgery (Fig. 12*b*) showed no improvement. IVP at 3 months also showed no significant improvement (Fig. 12*c*).

Despite little roentgenographic improvement the patient is clinically well 1 year after surgery.

CASE IX. L.R., a $2\frac{1}{2}$ year old female with a 1 year history of urinary tract infection and right UPJ obstruction (Fig. 13*a*). Urine examination and culture were normal.

Dismembered pyeloplasty was done.

IVP 5 days postoperatively (Fig. 13*b*) showed slight improvement.

At $2\frac{1}{2}$ months severe right hydronephrosis was seen (Fig. 13, *c* and *d*) due to restenosis at UPJ. Urinalysis now showed 10–15 WBC/HPF and urine cultures were positive for *E. coli*.

Repeat dismembered pyeloplasty was done.

Slow but gradual improvement occurred, as shown by a nephrostogram at 7 days postoperatively (Fig. 13*e*), IVP 2 weeks after the second surgery (Fig. 13*f*), IVP at 6 weeks (Fig. 13*g*), IVP at 10 months (Fig. 13*h*), and IVP at 16 months (Fig. 13*i*).

One bout of urinary tract infection occurred at 16 months after the second operation; the patient otherwise is well.

RESULTS

In 21 of 29 operations there was some urographic evidence of improvement, with diminished caliectasis and improved ureteral opacification (2 or 21 improved were re-operations). Of 8 operations yielding no roentgenographic improvement, 2 developed fibrosis re-stenosing the UPJ, 1 a renal pelvis-cutaneous fistula, and 1 a calculus formation. It proved difficult, however, as in the literature, to closely correlate the clinical and urographic courses, while it was true that 2 cases requiring re-operation deteriorated by both clinical and roentgenographic parameters (Case IX). The 6 other

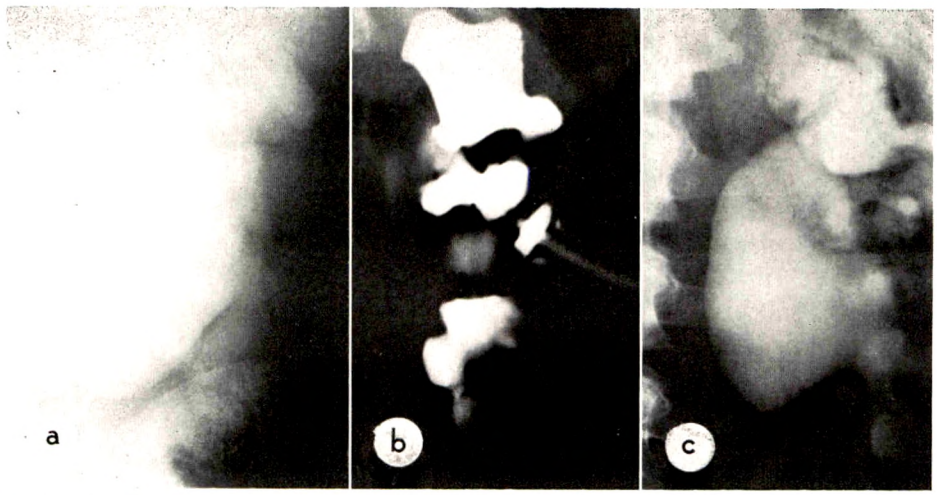


Fig. 5. Case II. (*a*) Preoperative IVP showing left UPJ obstruction and severe left hydronephrosis. (*b*) Nephrostogram 8 days after Foley Y–V pyeloplasty showing considerable calyceal decompression. (*c*) IVP 6 weeks after surgery showing some improvement over preoperative examination. Note that the pelvis, while smaller than in *a*, is larger than typically seen with more resective procedures (dismembered pyeloplasty).

roentgenographic failures all showed some degree of clinical improvement (Case VIII) or were asymptomatic to begin with. Judging the clinical course in our material was of additional difficulty, since the UPJ obstruction was found incidentally in 6 of the operated patients (during study for enuresis, failure to thrive, hypertension etc.) and

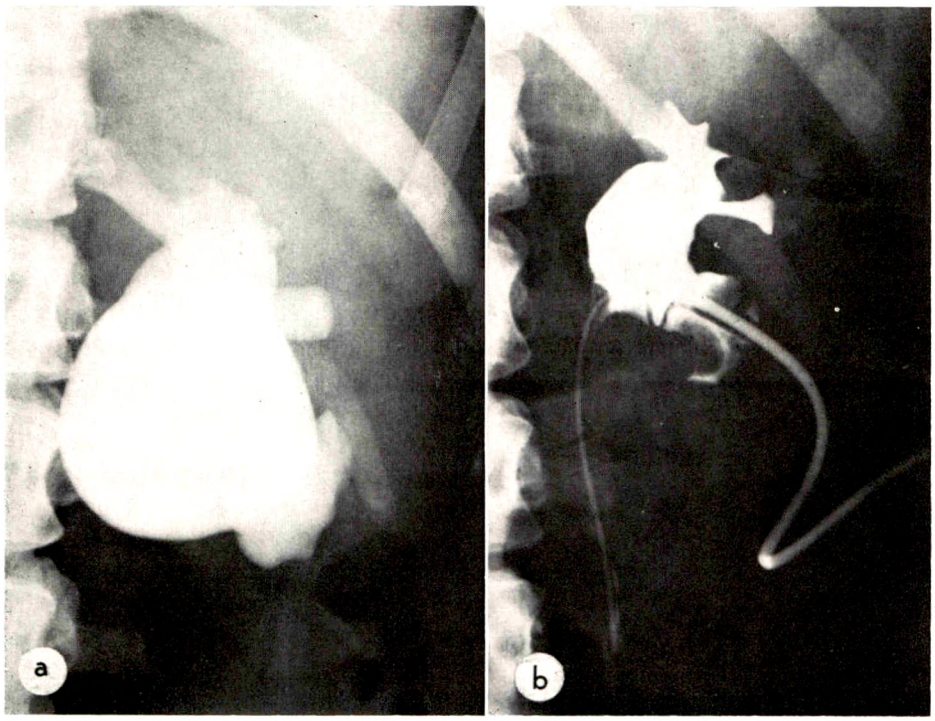


Fig. 6. Case III. (*a*) Preoperative IVP showing left UPJ obstruction, and severe left hydronephrosis. (*b*) Nephrostogram 9 days after Culp operation showing calyceal and pelvic decompression.

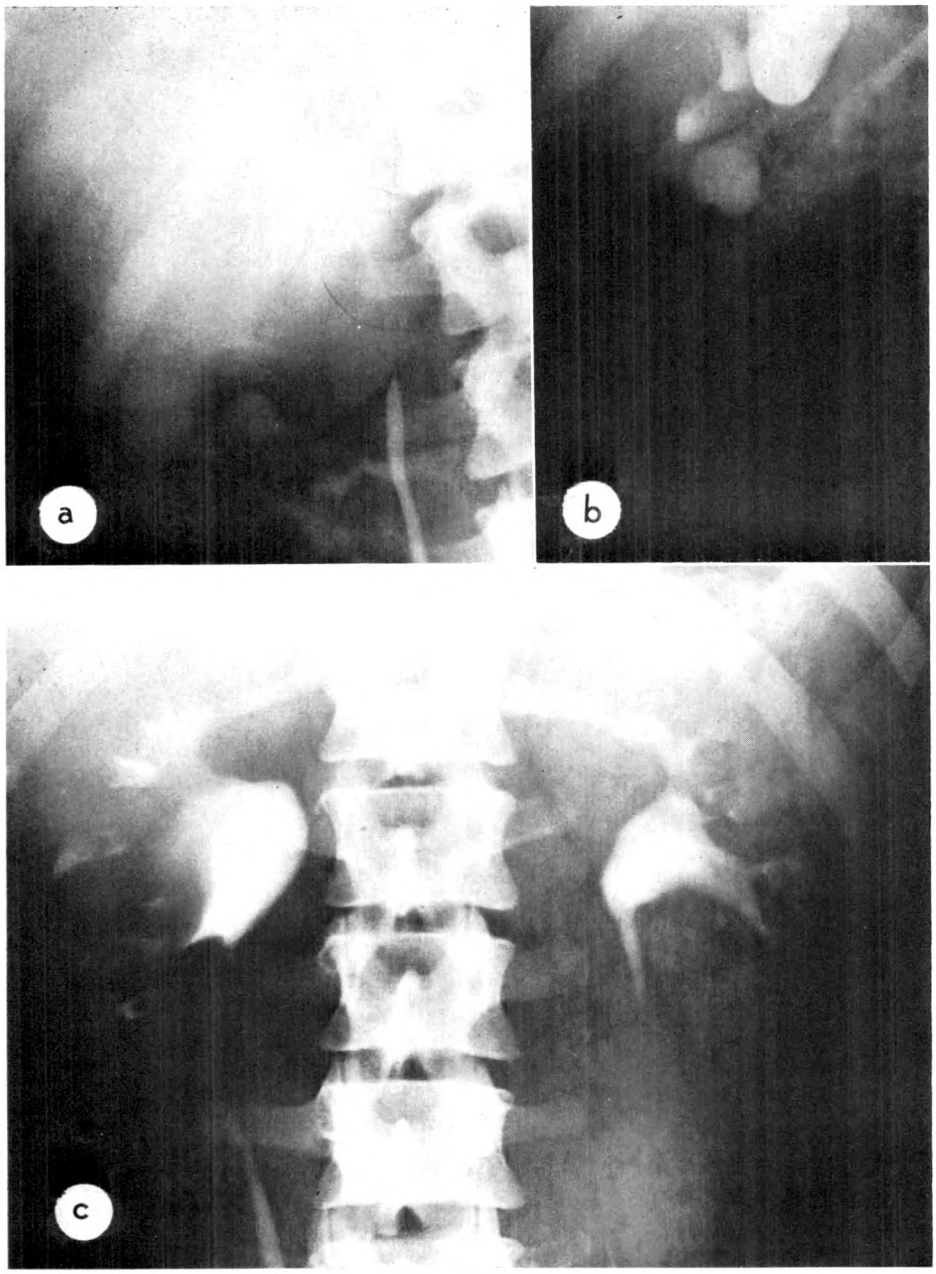


FIG. 7. Case IV. (*a*) Preoperative right retrograde pyelogram showed UPJ obstruction thought to be due to short segmental stricture, and severe right hydronephrosis. (*b*) IVP 7 days after right dismembered Anderson-Hynes pyeloplasty showed persistent right UPJ obstruction. (*c*) IVP 3 months after surgery showed marked improvement. Note the more lateral position of the UPJ, some flattening of the medial border of the relatively smaller pelvis, medial tilting of the cephalad portion of the pelvis, and ureteral opacification.

the urine studies (examination, culture, or both) were negative initially in 13 cases (not surprising, perhaps, in view of obstruction to diseased kidney). The blood urea nitrogen or creatinine levels have not been useful with unilateral disease. It has been further noted[12] that renal dissection for pyeloplasty may itself result in effective

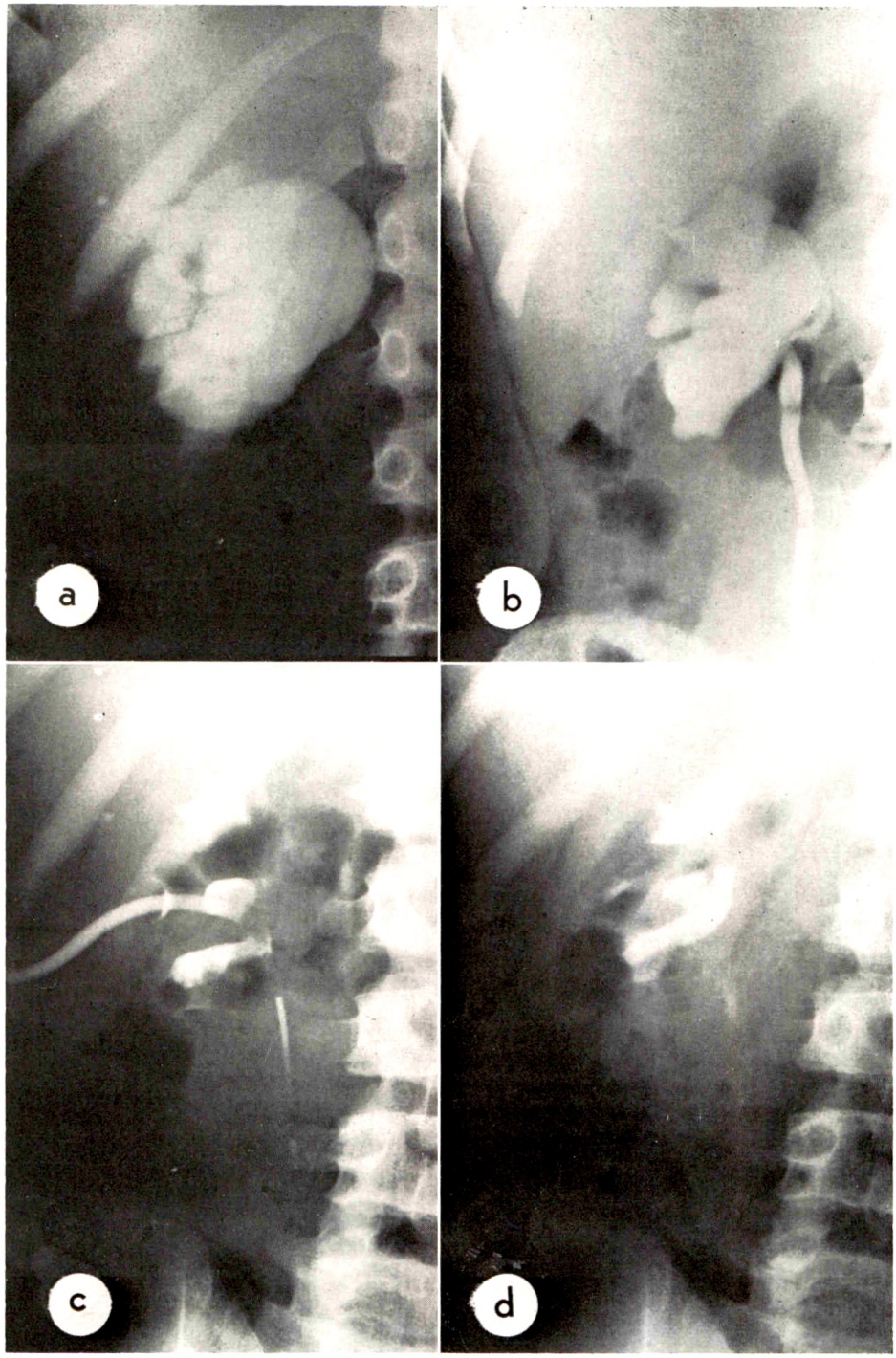


Fig. 8. Case v. (*a*) Preoperative IVP, 2½ hour film, showing right UPJ obstruction. (*b*) Right retrograde pyelogram showing apparent stricture just beyond UPJ. (*c*) Nephrostogram 10 days after dismembered pyeloplasty showing marked pelvicalyceal decompression. (*d*) IVP 4 months after surgery showing nearly complete resolution of hydronephrosis, and ureteral opacification.

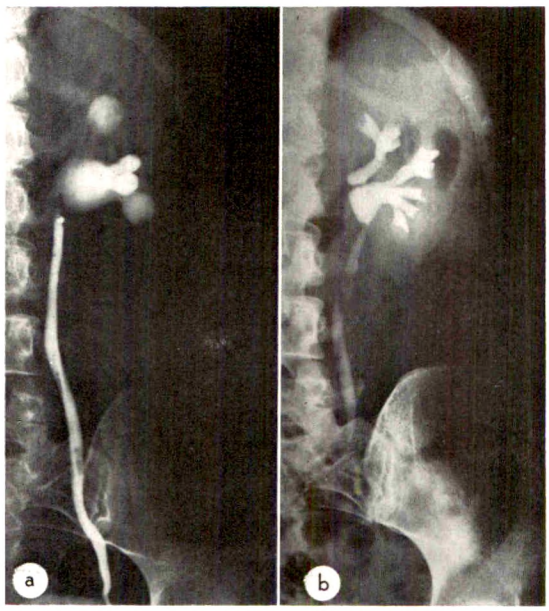


FIG. 9. Case VI. (*a*) Preoperative left retrograde pyelogram demonstrated UPJ obstruction. (*b*) IVP 3 weeks after dismembered pyeloplasty, showing marked improvement, and changes similar to those of Case IV (laterally displaced UPJ with flat medial pelvic wall and slight medial tilt of cephalad portion of pelvis).

renal denervation and give pain relief—a fact which must be considered when evaluating the 94 per cent pain relief figure of Anderson.[1] Operative success or improvement has been variable in the literature, with improved rates reported as 87 per cent,[15] 66 per cent,[1] 62.5 per cent,[12] and 83.7 per cent.[3] "Normal" IVPs were obtained by Prince and Scardino[12] in 32 per cent of cases. Davies *et al.*[5] cited clinical improvement of 95 per cent, but improved drainage via radioactive renography only in 50 per cent of cases.

Reported common causes[3,9] of failure have included: calculus formation; anastomosis leak; infection; buckling anastomosis; new stricture; urinary fistulae; and impaired motor mechanism. Our own material included examples of: postoperative fibrosis at the UPJ; UPJ-cutaneous fistula; and calculus formation. Simple failure of resolution of caliectasis, cause indeterminate, predominated among the unimproved cases (Case VIII).

Postoperative roentgenographic resolution has been characterized in the urologic literature as slow to occur. Eberhart and Reiser[6] felt that 1 month postoperative was too early to predict results, and that at 3 months a more accurate assessment could be made. Williams[16] noted that the "immediate radiographic results are not so good as late postoperative studies." Of 6 cases showing best improvement in our series, in 2 instances (Cases IV and IX) there was slow initial opening at the operative site, with optimal results evident several months later. However, the other 4 examples of marked roentgenographic resolution became manifest quite early: at 7 days (Case VII, Fig. 10*a*); at 9 days (Case III, Fig. 6*b*); and 10 days (Case V, Fig. 8*c*) after surgery. It may well be then that failure of resolution in the first weeks postoperatively may be a worse prognostic sign than previously believed.

Successful cases, from published material and as described above, show diminished

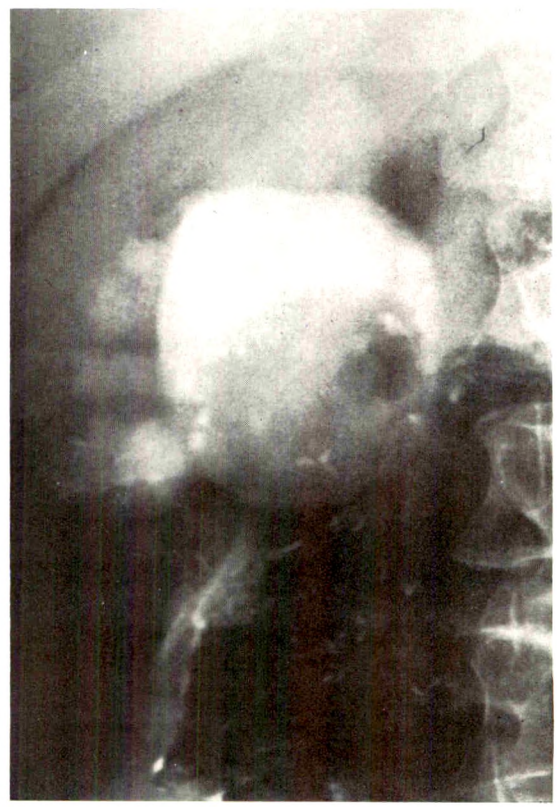


FIG. 10. Case VII. Preoperative IVP, right UPJ obstruction.

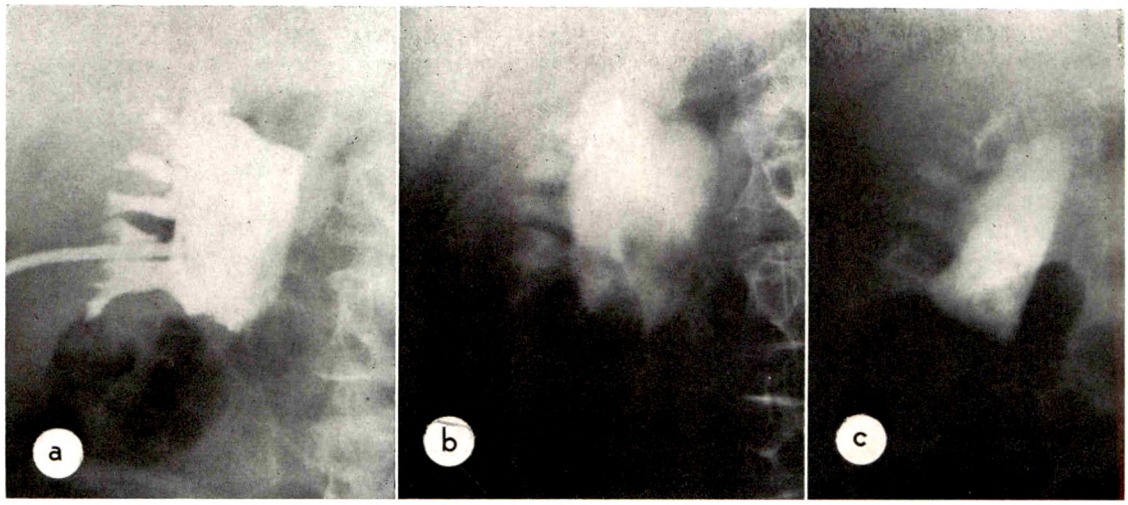


Fig. 11. Case VII. (*a*) Nephrostogram 7 days after dismembered pyeloplasty. Calyceal decompression. (*b*) IVP 2 weeks after surgery. (*c*) IVP 4 months after surgery showing calyceal decompression as compared with Figure 10.

caliectasis, variable decrease in pelvic size, and improved UPJ and ureteral opacification. Decreased pelvic size was a less helpful indicator of relief of obstruction with dismembered pyeloplasty because of the frank resection of pelvis in this approach. It is difficult to closely relate the various approaches with specific roentgen changes, since minor UPJ and pelvic contour defects appear that are largely unpredictable and of little significance in themselves. However, the dismembered pyeloplasty, of considerable current interest and comprising the bulk of our case material, did typically cause, in addition to decreased caliectasis where successful, lateral displacement of the UPJ and marked flattening of the medial pelvic border. Slight medial tilt of the cephalad portion of the pelvis (Cases IV–IX) is believed to be related to associated sewing down of the kidney to the psoas muscle, at this institution. Also noted with the dismembered procedure is a generally smaller pelvis than the flap or intubated procedure (in keeping with pelvic resection) (Fig. 3).

CONCLUSIONS

1. Satisfactory urographic evidence of drainage is required before judging a pyeloplasty successful.[2,9] The difficulties in evaluating the clinical changes are outlined: it is difficult to envision good urine flow, but poor contrast medium flow.

2. Resolution of hydronephrosis may occur quite early in the successful case (Cases III, V, and VII), assuming that occasionally optimal results are seen only after several months.

3. Minor postoperative UPJ contour defects are largely unpredictable and of little consequence. It would be difficult to specifically reconstruct the surgical approach from urography alone, although resective operations such as dismembered pyeloplasty result in greater decrease in pelvic size.

4. While it may well be that nonobstructive hydronephrosis is commoner than previously believed, from analysis of this series and a review of the published material in the literature, it appears that some patients with true organic UPJ obstruction are amenable to surgical correction.

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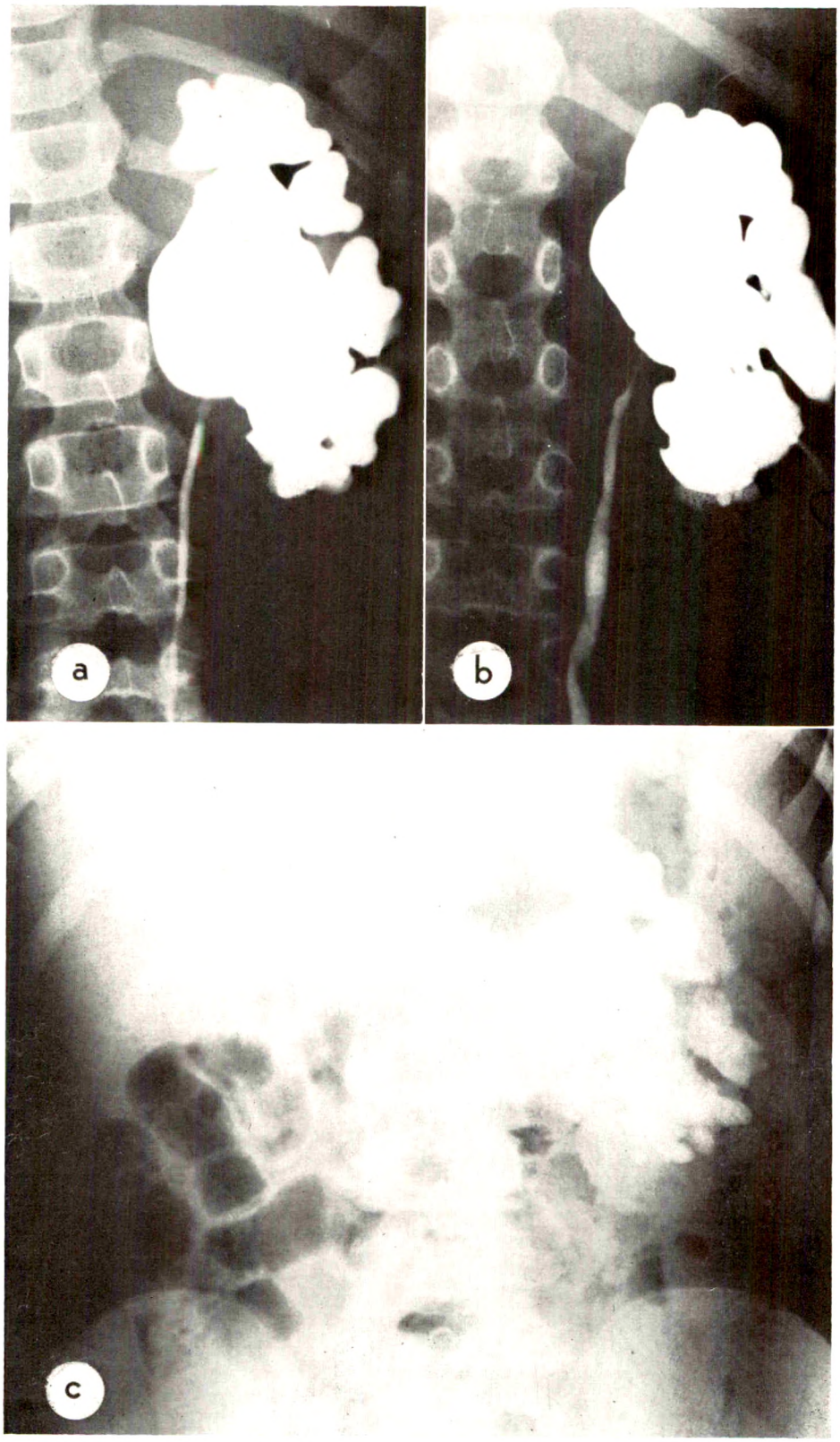


FIG. 12. Case VIII. (*a*) Preoperative left retrograde pyelogram showing left hydronephrosis. (*b*) Nephrostogram 3 weeks after left dismembered pyeloplasty. No improvement. (*c*) IVP 3 months after surgery. No significant improvement.

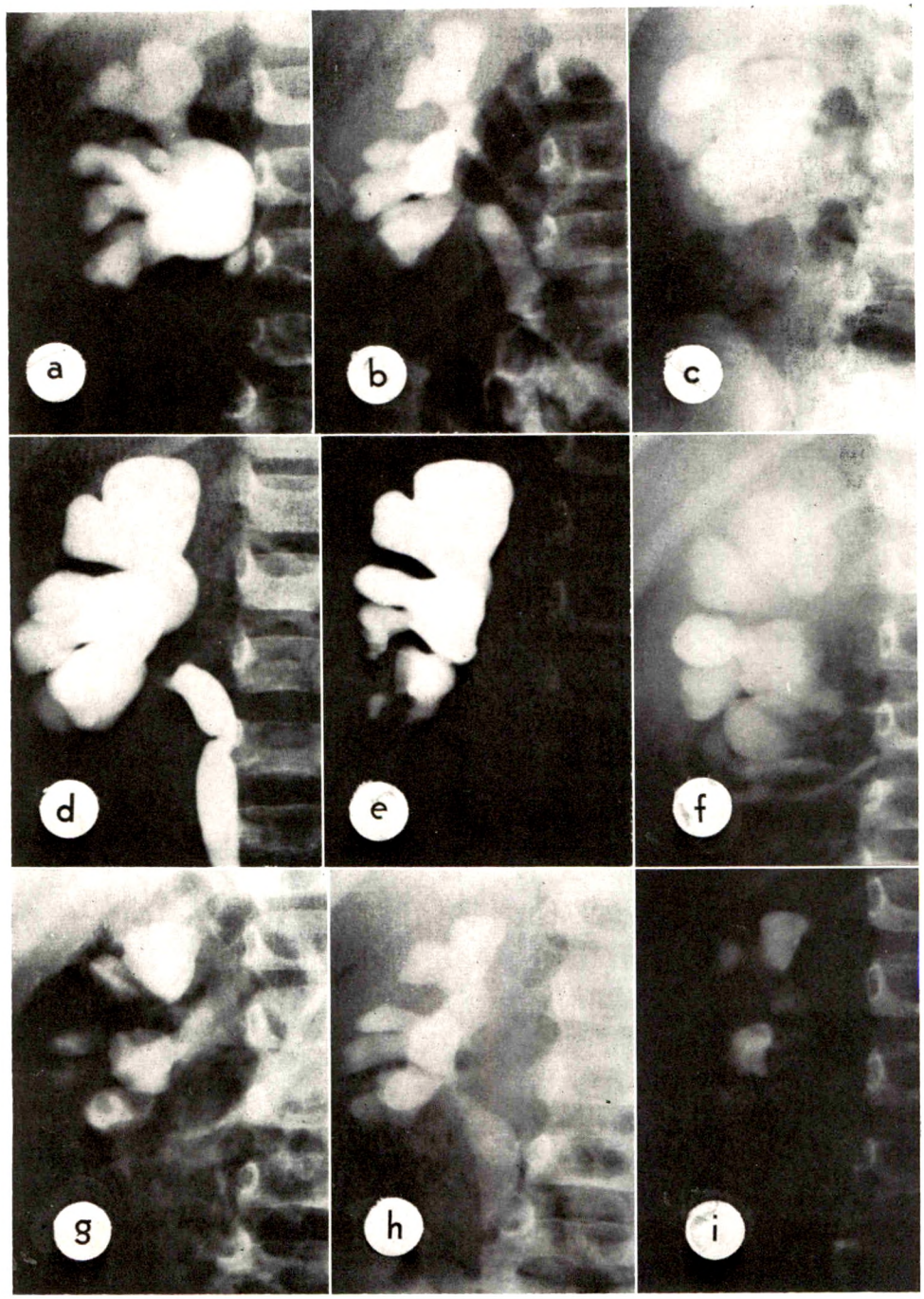


Fig. 13. Case IX. (*a*) Preoperative right retrograde pyelogram, UPJ obstruction. (*b*) IVP 5 days after dismembered pyeloplasty. Slight improvement. (*c*) IVP 2½ months after surgery. (*d*) Right retrograde pyelogram 2½ months after surgery, recurrent UPJ obstruction. (*e*) Right nephrostogram 7 days after second operation, little change. (*f*) IVP 2 weeks after second operation. (*g*) IVP 6 weeks after second operation. (*h*) IVP 10 months after second operation, some improvement. (*i*) IVP 16 months after second operation. Further improvement.

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REFERENCES

1. ANDERSON, J. C. Hydronephrosis: fourteen years survey of results. *Proc. Roy. Soc. Med.*, 1962, *55*, 93–99.
2. BURNS, C. N., DREW, J. E., and DEAN, A. L. Ureteropelvic obstruction with hydronephrosis: treatment by pyeloplasty in 23 cases. *J. Urol.*, 1953, *70*, 845–855.
3. CREEVY, C. D., and HELENBOLT. Results of Y plasty after 5 to 24 years: review of 73 operations. *J. Urol.*, 1957, *77*, 149–167.
4. CULP, O. S., and DEWEERD, J. H. Pelvic flap operation for certain types of ureteropelvic junction obstruction. *J. Urol.*, 1954, *81*, 523–529.
5. DAVIES, R. J., JONES, D. J., CROFT, D. N., and SHUTTLEWORTH, K. E. Assessment of Anderson-Hynes pyeloplasty by radioisotope renography. *Proc. Roy. Soc. Med.*, 1969, *62*, 1123–1124.
6. EBERHART, C., and REISER, C. Experiences with pyeloplasty. *J. Urol.*, 1953, *69*, 208–216.
7. EMMETT, J. L., and WITTEN, D. A. Urography. Third edition. Volume 1. W. B. Saunders Company, Philadelphia, 1971, pp. 408–431.
8. FENGER, C. A. Operation for relief of valve formation and stricture of ureter in hydro or pyonephrosis. *J.A.M.A.*, 1894, *22*, 335–343.
9. GIBSON, T. E. Hydronephrosis: classification and plastic repaire of ureteropelvic obstruction. *Surg., Gynec. & Obst.*, 1945, *80*, 485–496.
10. HENLINE, R. B., and MENNING, J. H. Management of hydronephrosis due to ureteropelvic obstruction: preliminary report. *J. Urol.*, 1943, *50*, 1–23.
11. KUSTER, Ein Fall von Resection des Ureter. *Arch. f Klin. Clin.*, 1892, xliv, H.*4*, 850.
12. PRINCE, C. L., and SCARDINO, P. L. Results of various procedures used for correction of congenital ureteropelvic obstruction. *J. Urol.*, 1962, *87*, 315–318.
13. SARGENT, J. C. Basic principles governing conservative surgery in hydronephrosis. *J. Urol.*, 1942, *47*, 323–343.
14. SHOPFNER, C. E. Ureteropelvic junction obstruction. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1966, *98*, 148–159.
15. WEKER, C. H., and GLENN, J. F. Efficacy of pyeloplastic surgery. *Am. Surgeon*, 1970, *36*, 69–73.
16. WILLIAMS, E. C. Ureteropyeloplasty in treatment of advanced hydronephrosis. *South. M. J.*, 1952, *45*, 287–292.

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THE ROENTGENOGRAPHIC DETERMINATION OF NORMAL ADULT KIDNEY SIZE AS RELATED TO VERTEBRAL HEIGHT*

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THE determination of normal standards of measurement by the use of ratio relationships between body parts of an individual obviates to great degree the problem of individual variation and lends for easy applicability. This technique applied to the determination of normal size would have considerable merit as a screening method.

Normal kidney size in pediatric patients has been evaluated with regard to correlating the height of the renal shadows with the number of equivalent lumbar vertebrae.[1]

This investigation was undertaken to test the validity of broadening this concept to the establishment of a normal standard ratio measurement of renal size on roentgenograms in adult subjects. Rather than the assemblage of a compendium of proportionality tables, the major goal has been the attainment of a readily useful standard for the radiologist in evaluating renal size as an indication of pathology.

METHOD

Two hundred intravenous urograms were randomly selected. Inclusion in the study was dependent upon normal roentgenologic appearance, normal urinalysis, absence of subsequent renal symptomatology in follow-up clinic visits, and normal renal arteriograms, in some cases. The roentgenologic examinations were performed for diagnostic evaluation of suspected specific renal disease (renal calculi, acute glomerulonephritis), hypertension, metastatic surveys, or nonspecific symptomatology of possible renal origin. Patients with "normal" urograms, but with a past history of renal dysfunction, were excluded. Urograms included were from studies done from 1969 through 1971. The patient population of 200 consisted of 112 males and 88 females ranging in age from 15 to 77 years, 150 of whom were from 20 to 60 years of age at the time of examination.

Roentgenograms were taken at a uniform distance (40 inches), with the patient in the supine position, to minimize forward renal displacement and/or ptosis, which might cause distortion in the prone and upright positions.

Measurements were made using a standard millimeter rule. These included: (1) right and left renal shadows from upper to lower pole; (2) lumbar vertebrae L_1 through L_3; (3) L_1 through L_3 plus the third intervertebral disk; and (4) L_1 through L_4 (Fig. 1). Where anterior and posterior shadow projections of the vertebral body borders did not superimpose, a median interpolation was used for measurement (Fig. 2).

RESULTS

Analysis of the renal measurements of the 200 patients (400 measurements) or 389 kidneys (97.3 per cent) corresponded to the range defined by the top of L_1 to the bottom of L_3 and the top of L_1 to the bottom of L_4, inclusive of intervertebral disks: 283 measurements (70.8 per cent) were within the range of L_1 through L_3 and L_1 through $\frac{1}{2}$

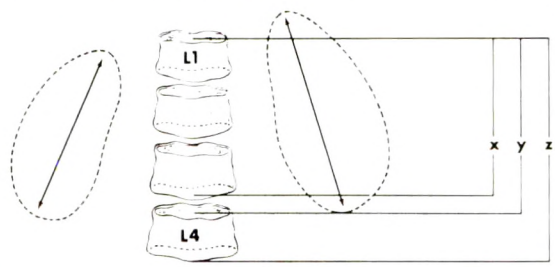


FIG. 1. Drawing of the lumbar vertebrae and kidneys showing the points of measurement.

* From the Department of Radiology, University of Virginia School of Medicine, Charlottesville, Virginia.

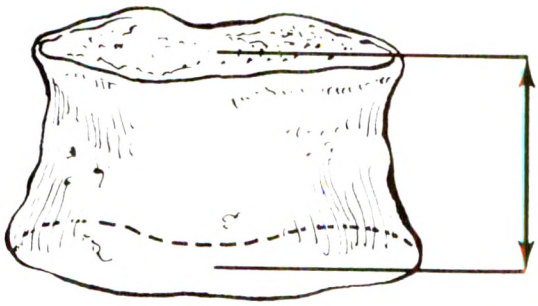


FIG. 2. Drawing of the lumbar vertebra showing median interpolation used for measurement.

L_4, inclusive of intervertebral disks. There was no significant difference between the sexes or among the age groups by decade.

Ratios relating kidney height to various lumbar segments are given in Table 1. Inspection reveals higher ratios of the left kidney *versus* the right kidney. Comparative renal shadow heights in the same patients showed the right equal to the left kidney in 60 patients, the left > right kidney in 120 patients, and left < right kidney in 20 patients. Again, there were no significant differences in comparison of sex and age groups.

The range of the absolute size taken directly from the roentgenograms was 10.0–15.4 cm. In those cases at the extremes of this range, there was no appreciable disproportion in size of the right *versus* the left kindey, as found in unilateral compensatory hypertrophy.

DISCUSSION

As is evident from this study, a roentgenographic determination of normal renal morphology is dependent upon apparent excretory function, structural characteristics, and general size. If the former 2 determinants appear normal, evaluation of the height of the renal shadows can provide a reliable parameter when compared with the lumbar dimensions in the same patient.

The range of normal is substantial in terms of absolute measurements (10.0–15.4 cm.), is less when reviewed in terms of lumbar segments, and is narrowed further when ratios of these measurements are employed.

From this study a normal size probability of 97 per cent is possible by utilizing a comparative range of L_1 through L_3 and L_1 through L_4. In clinical evaluation the use of *relative* renal sizes is more widely applicable than usage of absolute values, *e.g.*,

TABLE I

MEASUREMENT OF VARIOUS PARAMETERS

Age	Sex	$\frac{R}{X}$	$\frac{R}{Y}$	$\frac{R}{Z}$	$\frac{L}{X}$	$\frac{L}{Y}$	$\frac{L}{Z}$	$L_{1-3} \rightarrow \frac{1}{2}L_4$		$L_{1-3} \rightarrow L_4$	
								R	L	R	L
15–19	Male 9	1.01 ± .06	1.10 ± .08	0.80 ± .04	1.08 ± .08	1.19 ± .10	0.86 ± .07	8	–	8	9
	Female 7	1.07 ± .03	1.16 ± .04	0.84 ± .03	1.10 ± .07	1.18 ± .06	0.85 ± .04	7	6	7	7
20–29	Male 19	1.09 ± .07	1.19 ± .09	0.86 ± .06	1.11 ± .08	1.22 ± .08	0.88 ± .06	14	12	18	19
	Female 17	1.08 ± .08	1.18 ± .09	0.85 ± .06	1.12 ± .10	1.22 ± .11	0.88 ± .08	14	8	17	17
30–39	Male 17	1.05 ± .05	1.15 ± .07	0.83 ± .05	1.09 ± .06	1.19 ± .07	0.86 ± .05	16	13	17	16
	Female 15	1.08 ± .08	1.18 ± .12	0.84 ± .08	1.11 ± .10	1.21 ± .12	0.87 ± .08	12	9	14	12
40–49	Male 17	1.07 ± .07	1.19 ± .07	0.86 ± .05	1.09 ± .07	1.21 ± .08	0.87 ± .06	13	12	17	17
	Female 24	1.10 ± .07	1.20 ± .07	0.87 ± .05	1.12 ± .08	1.23 ± .09	0.89 ± .09	16	14	24	24
50–59	Male 28	1.04 ± .08	1.14 ± .09	0.83 ± .06	1.04 ± .10	1.16 ± .10	0.83 ± .08	22	20	26	26
	Female 13	1.11 ± .08	1.21 ± .08	0.88 ± .06	1.13 ± .06	1.23 ± .06	0.90 ± .05	8	7	13	13
60–77	Male 22	1.07 ± .11	1.18 ± .11	0.81 ± .10	1.08 ± .10	1.19 ± .10	0.86 ± .03	16	13	22	22
	Female 12	1.07 ± .10	1.18 ± .11	0.85 ± .08	1.09 ± .12	1.20 ± .13	0.86 ± .10	8	8	12	12
	Male 112	1.06	1.16	.83	1.08	1.19	.86				
	Female 88	1.09	1.19	.86	1.11	1.22	.88				
	All 200	1.07	1.17	.84	1.09	1.20	.87				

$X = L_1 \rightarrow L_3$; $Y = L_1 \rightarrow L_3$ + disk; $Z = L_1 \rightarrow L_4$; R = Right Kidney; L = Left Kidney.

12.0–14.0 cm. In this regard, the determination for pediatric patients would perhaps be better expressed as a particular ratio rather than a combined value of L_1 through L_4 +1.0 cm.[1] The limitations of applying these criteria are seen in the occurrence of significant renal pathology in a limited number of patients with normally appearing roentgenograms, despite clinically documented renal dysfunction. These exceptional cases may be roentgenologically "silent" or cause only transient alterations in roentgenologic appearance.

Analyses similar to the one presented here might be valuable in obtaining standards for roentgenologic changes in specific renal disease states with comparative studies prior to the inception of disease, as well as post recovery.

SUMMARY

A determination of normal adult renal size in roentgenographic examinations has been performed with data presented to aid in evaluating this parameter in routine examinations.

The limits of confidence are prescribed in terms of absolute correlation to lumbar segments and as proportionalities.

Ninety-seven per cent of normal adult kidneys are found within the range defined by the distance L_1 through L_3 and the distance of L_1 through L_4.

Factors of age and sex do not significantly affect these determinations, in the absence of intruding pathology.

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REFERENCES

1. Currarino, G. Roentgenographic estimate of kidney size in normal individuals with emphasis on children. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1965, *93*, 464–466.
2. Moell, H. Size of normal kidney. *Acta radiol.*, 1956, *46*, 640–645.
3. Moell, H. Kidney size and its deviations from normal in acute renal failure. *Acta radiol.*, 1961, Suppl. 206, *56*, 5–74.
4. Wald, H. Weight of normal adult human kidney and its variability. *Arch. Path.*, 1937, *23*, 493–500.



ACUTE RENAL CARBUNCLE*

THE ROENTGENOGRAPHIC CLARIFICATION OF A MEDICAL ENIGMA

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MOST roentgenographic descriptions of renal carbuncle emphasize the features, depicting mainly the subacute and chronic varieties.[2,5,6] Although the diagnostic criteria for renal carbuncle have improved, the over-all diagnostic accuracy prior to surgery is still poor and rarely extends beyond 20 per cent in most series. An even greater diagnostic challenge is the acute renal carbuncle. This at one time was almost never accurately diagnosed. Nevertheless, this form of renal infection is by no means uncommon or insignificant, and with the problem of increasing drug addiction by "mainlining" is becoming more frequent. The diagnosis may be clinically suspected when symptoms of fever, chills, and pain localized to the flank, or the signs of sepsis are present. More often, however, symptoms related to the urinary tract are absent, and the urinalysis is practically always normal or nondiagnostic. In addition, if one waits for gross urographic changes to occur before making the diagnosis, much valuable time will be lost.[4] Evans *et al.*,[5] and Himmelfarb *et al.*,[6] in their descriptions of renal carbuncle, alluded to many roentgenographic findings observed in the acute phase. Precise analysis of these findings and their relation to an exact diagnosis were not stressed.

The purpose of this paper is to further analyze the major roentgenographic findings of acute renal carbuncle, so that accurate and definitive criteria can be established.

CASE MATERIAL

A total of 6 patients are presented in this paper (Table 1). Their ages range from 14 to 60 years. Surprisingly, there appears to be an equal distribution of sex, with 3 females and 3 males. Two of the men were in the older age group and the primary disease was related to the prostate gland. In both, the involved organism was *E. coli*. In the remaining 4 cases, the causative agent was *Staphylococcus aureus*. The 3 females were in their twenties and all were drug addicts. Furunculosis was the initial focus of infection in the remaining male patient.

PATHOLOGY

Since the roentgenographic pattern is a reflection of the underlying pathologic changes, a brief review is in order.

Acute infection of the kidney, in general, arises either on a hematogenous basis or *via* a cysto-ureteric route. *Staphylococcus aureus* is the most common bloodborn organism and *E. coli* the causative agent in ascending infections.

The definition of an acute carbuncle is an area of severe suppurative pyelonephritis containing many micro-abscesses coalescing into larger confluent abscesses with as yet not well defined walls. Originally, multiple small abscesses of varying size are formed within the cortex from either septic emboli lodged within the smaller vessels or by lymphatic extension from a suppurative pyelonephritis arising within the medulla. Cortical involvement stimulates a pronounced inflammatory response primarily because of the large blood supply feeding the cortex, and secondarily because of the lower levels of interstitial pressure. The latter enables the cells to move easily into the inflammatory area.[8] The process may be diffuse, virtually involving the entire

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TABLE I

SUMMARY OF FINDINGS IN 6 CASES

Case No.	Sex	Drug Addict	Age (yr.)	Plain Roentgenograms	Intravenous Pyelograms	Retrograde Pyelograms	Arteriograms a. No displacement b. Sparse branching c. Loss of corticomedullary junction	Comments	Results
I	M	no	14	questionable changes	1. partial visualization 2. mass effect	—	—	furunculosis	1. drainage and antibiotics 2. returned to normal
II	F	yes	24	mass of left kidney	nonvisualization	1. displacement of calyces 2. mass	a., b., c.		1. surgical removal 2. carbuncle of upper pole
III	F	yes	28	mass of right kidney	poor visualization	—	refused		1. lost to follow-up
IV	F	yes	20s ?	mass of left kidney	partial to poor visualization	—	a., b., c.		1. drainage 2. clinically improved 3. intravenous pyelogram—normal
V	M	no	55	no mass	nonvisualization	—	questionable a., b., c.	prostatic carcinoma	1. surgical removal 2. diffuse involvement
VI	M	no	62	not available	nonvisualization	1. dilated calyces 2. no displacement 3. filling defect in ureter	a., b., c.	septic course following prostatectomy	1. surgical removal 2. diffuse involvement

kidney, or remain limited to only a portion or portions of the kidney. It is basically parenchymal in location and does not communicate with the collecting system in its early form. Extension through the capsule into the perirenal tissues is nevertheless almost always present, and at one time was thought to be the primary site of involvement. It is now believed that perirenal infection arises secondarily from lesions within the renal cortex via the peripheral lymphatics.

ROENTGENOGRAPHIC FEATURES

The roentgenographic features described represent the composite findings of the 6 patients presented and some personal observations noted in other patients. In general, the entire spectrum of the roentgenographic features prior to angiographic studies consists of a combination of changes indicating both the presence of interstitial or parenchymal disease with associated perirenal involvement.

A. FLAT FILM ROENTGENOGRAPHY OF THE ABDOMEN

With the kidney either diffusely or partially involved by the inflammatory process, a supine roentgenogram of the abdomen demonstrates either diffuse or local enlargement of the renal shadow. Simultaneously occurring perirenal disease is common and, when present, obscures the renal and the psoas muscle shadows (Fig. 1). In 2 of our cases, pronounced perirenal involvement was present, causing an increased density within the retroperitoneum as well as associated scoliosis. In 1 patient, the diseased kidney was also fixed during respiration (Fig. 2). Further extension of the pro-

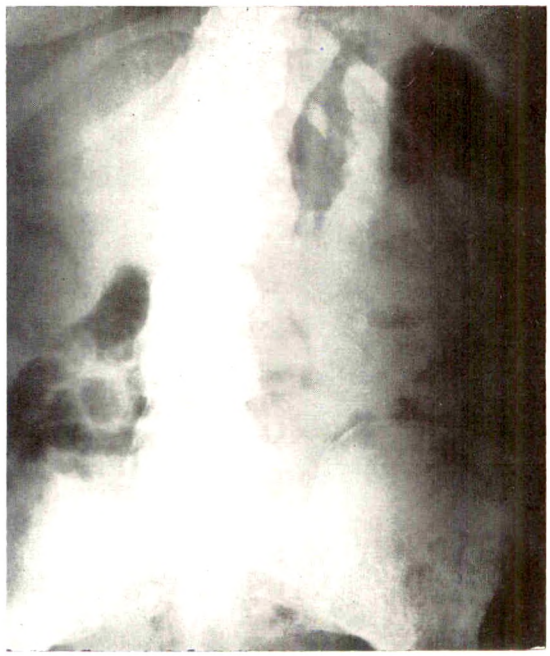


Fig. 1. Plain film roentgenogram of the abdomen reveals a vague density involving the left abdomen. Both the renal and the psoas muscle shadows are difficult to evaluate. Scoliosis with deviation away from the involved area is noted.

cess into the surrounding area, displacing the kidney and the ureter can also occur.[5]

B. Intravenous Pyelography

The findings encountered on intravenous pyelography vary from partial to poor, to complete nonvisualization of the collecting systems (Fig. 3, *A–C*). Partial visualization was observed in 50 per cent of patients. Specific calyceal changes can be noted in the locally infected area. The calyces were either poorly opacified or amputated in contrast to the remaining fairly adequately visualized collecting systems. In some cases, the affected calyces were stretched and draped around a suggested localized mass.

C. Retrograde Pyelography

The need for retrograde pyelography in the diagnosis of acute renal carbuncle is limited. Most of the necessary information can be derived from intravenous pyelography and nephrotomography. However, in cases where the collecting systems are completely nonvisualized, this procedure may be necessary to exclude the presence of either ureteral obstruction by calculus or pyonephrosis. Although the clinical presentation of both these latter entities resembles that observed in acute carbuncle, the therapeutic approach is quite different. Differentiation is therefore very important. In 1 patient with a nonvisualized collecting system, no evidence of obstruction was demonstrated by retrograde pyelography. The calyces were well outlined and displaced downward by a large mass in the upper pole (Fig. 4). In another patient, ureteral obstruction caused by tissue slough within the ureter was demonstrated and a specific diagnosis in this case could not be accomplished. Following surgical removal, the kidney demonstrated diffuse parenchymal disease and necrosis.

D. Nephrotomography

Nephrotomography was not performed on any of our patients. However, in our institution, as well as in others, tomography is now being utilized almost routinely in conjunction with high dose pyelography, and is once again becoming an important diagnostic procedure. In patients showing some degree of visualization, it may prove worthwhile to eliminate the need for arteriography, speed the diagnostic time, and

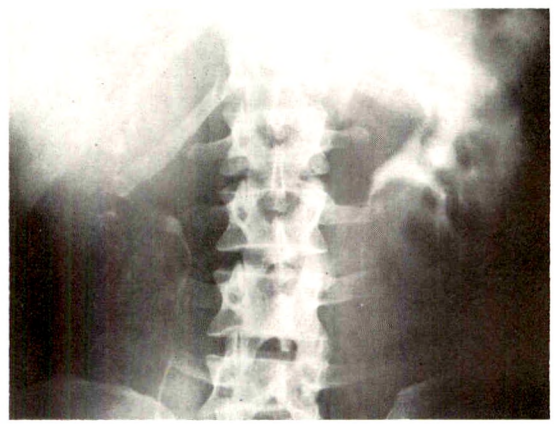


Fig. 2. Roentgenogram taken during respiration shows a poorly opacifying right kidney that is fixed in position. On the left, the collecting systems are well opacified, but mobile.

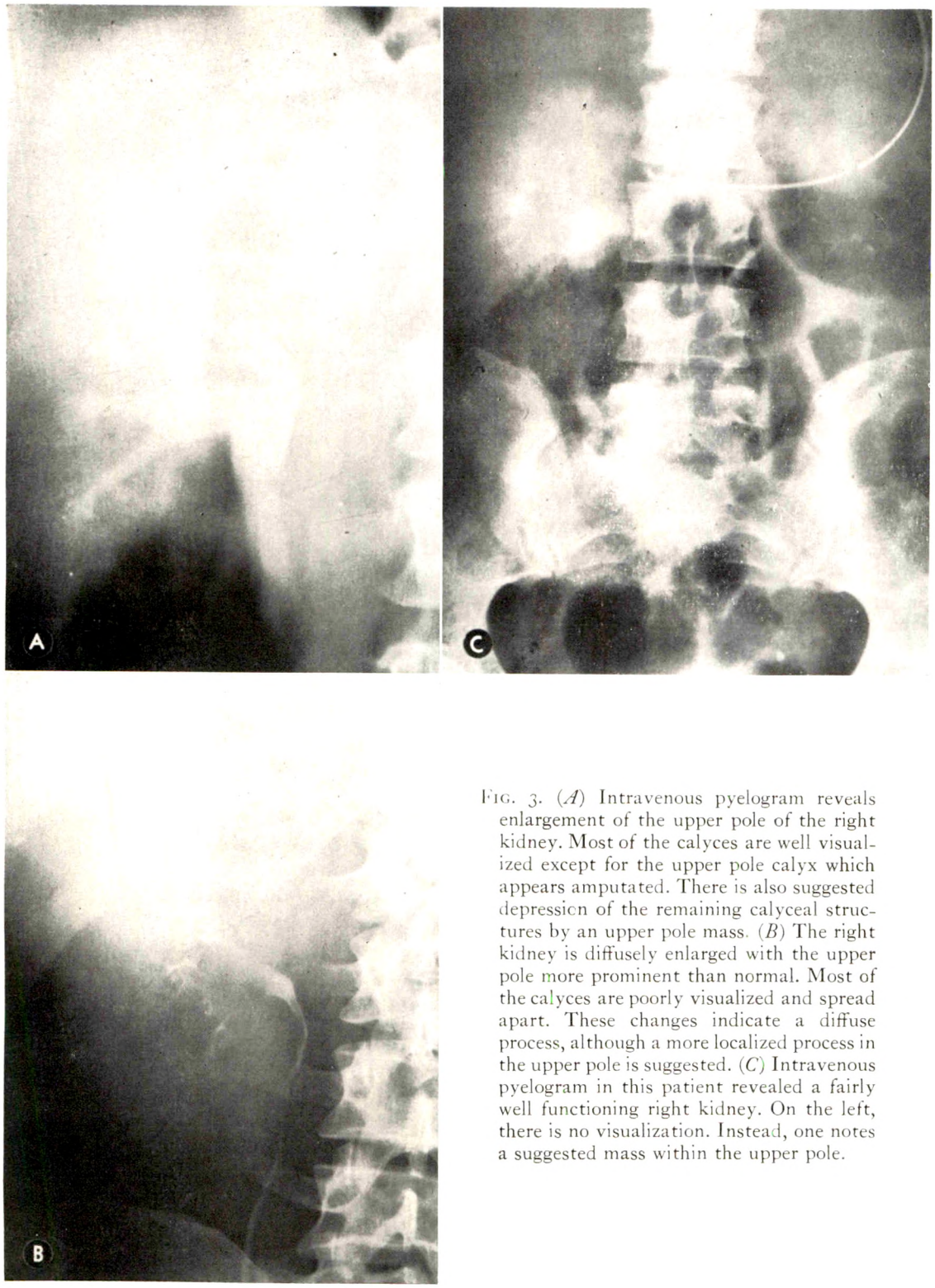


FIG. 3. (*A*) Intravenous pyelogram reveals enlargement of the upper pole of the right kidney. Most of the calyces are well visualized except for the upper pole calyx which appears amputated. There is also suggested depression of the remaining calyceal structures by an upper pole mass. (*B*) The right kidney is diffusely enlarged with the upper pole more prominent than normal. Most of the calyces are poorly visualized and spread apart. These changes indicate a diffuse process, although a more localized process in the upper pole is suggested. (*C*) Intravenous pyelogram in this patient revealed a fairly well functioning right kidney. On the left, there is no visualization. Instead, one notes a suggested mass within the upper pole.



Fig. 4. Retrograde pyelogram in a patient with non-visualization of the collecting system (Fig. 3*C*). The study reveals moderate dilatation of the calyceal structures, which are displaced downward by a mass within the upper pole (arrows).

spare the patient the morbidity of an additional vascular procedure.

The features described during this phase correspond to those obtained during the nephrogram phase of arteriography, and to some cases shown to us by our colleagues. One major feature is the suggestion of a renal mass, when the acute carbuncle is localized, or renal enlargement, when diffuse. The margin of the kidney, due to the surrounding inflammatory process is poorly defined or unsharp.[1] The density of the carbuncle, when localized, varies and may demonstrate either opacification or a vague over-all lucency. Occasionally, multiple lucencies, oval or rounded in configuration, representing multiple abscesses undergoing coalescence, may be apparent. In 1 patient, linear lucencies radiating through the mass were noted (Fig. 5). The surgical specimen revealed these to be dilated collecting tubules distended with pus. This striated pattern may be duplicated by any diffuse interstitial process, and has been described in a case of renal vein thrombosis.[3] (The explanation for these findings presented in the latter situation differs slightly from ours.)

E. ARTERIOGRAPHY

Arteriography was performed in 5 cases. The most characteristic angiographic feature, particularly in the localized acute carbuncle, is the absence of obvious vascular displacement by a mass despite its suggested presence on all the preliminary studies (Fig. 6, *A–C*). Instead, the vessels traverse through the infected area regardless of its location, with minimal, if any, displacement (Fig. 6*A*). They are, nevertheless, slightly spread apart and somewhat less opacified than the vessels within the normal portion of the kidney. In some cases,



Fig. 5. Nephrographic phase, in a patient with an acute carbuncle localized to the left upper pole, reveals diffusely radiating linear lucencies which on the surgical specimen were markedly dilated tubules filled with pus. Note the normal appearing lower pole

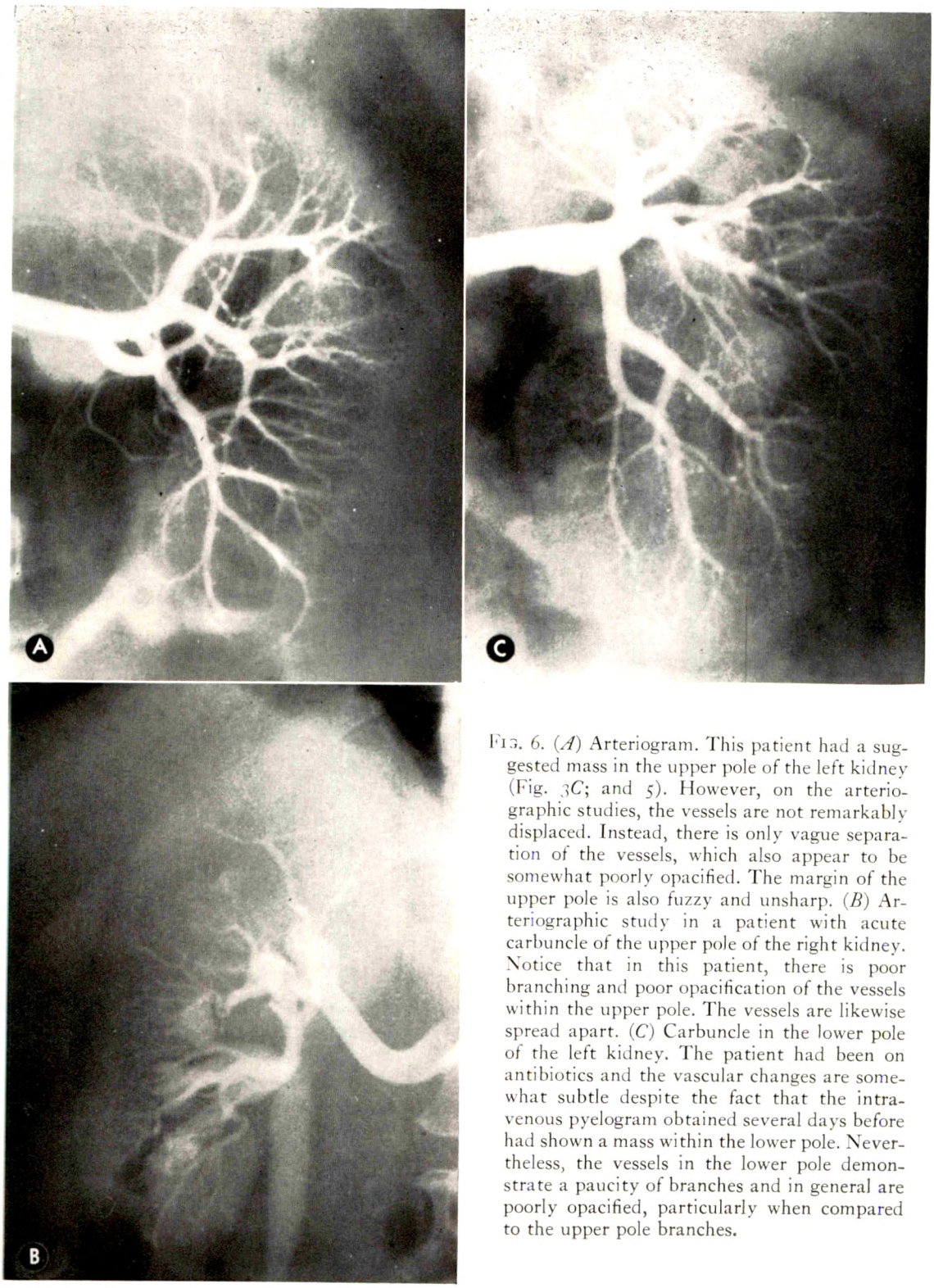


Fig. 6. (*A*) Arteriogram. This patient had a suggested mass in the upper pole of the left kidney (Fig. 3*C*; and 5). However, on the arteriographic studies, the vessels are not remarkably displaced. Instead, there is only vague separation of the vessels, which also appear to be somewhat poorly opacified. The margin of the upper pole is also fuzzy and unsharp. (*B*) Arteriographic study in a patient with acute carbuncle of the upper pole of the right kidney. Notice that in this patient, there is poor branching and poor opacification of the vessels within the upper pole. The vessels are likewise spread apart. (*C*) Carbuncle in the lower pole of the left kidney. The patient had been on antibiotics and the vascular changes are somewhat subtle despite the fact that the intravenous pyelogram obtained several days before had shown a mass within the lower pole. Nevertheless, the vessels in the lower pole demonstrate a paucity of branches and in general are poorly opacified, particularly when compared to the upper pole branches.

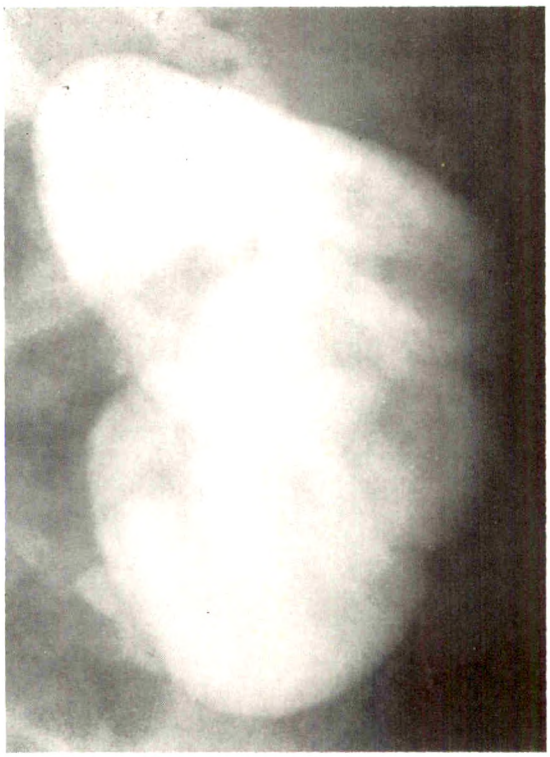


Fig. 7. Nephrographic phase. This is the patient seen in Figure 6*C*. There is a distinct loss of the cortico-medullary junction in the lower pole. The normal upper part of the kidney is within normal limits. A diagnosis of an acute renal carbuncle was made, but because of the lack of clinical symptomatology, the patient was not adequately treated. As predicted, she returned 10 days following the study with an exacerbation of the symptoms, and, at surgery, an acute carbuncle within the lower pole of the left kidney was drained.

the affected vessels branch sparsely and appear denuded (Fig. 6, *B* and *C*). Poor and slow perfusion within the infected area may also be apparent. Multiple factors probably account for these changes. The relatively poor arborization of the vessels is apparently caused by compression of these vessels by the surrounding inflammatory infiltration. It has also been demonstrated that in the presence of an inflammatory process, an extensive capillary network becomes functional.[2] This network is always existent, but under normal conditions nonfunctioning. When operational, normal exit *via* existing venule pathways is distinctly impaired and slowed. This phenomenon may be partially responsible for the slow perfusion noticed during the studies.

During the nephrographic phase, the normal cortico-medullary junction is frequently lost and replaced by a diffuse and relatively homogenous blush outlining the suggested mass noticed on the previous examinations (Fig. 7). This feature, when present, is both distinctive and important. In 1 patient who presented with a well localized acute carbuncle in which the vascular features were subtle, the loss of the cortico-medullary junction was diagnostic (Fig. 7). The over-all increased permeability and diffusion found at the capillary and venule level is responsible for this feature. In addition, most of the other findings described under the paragraph of nephrotomography may be seen in this stage.

DIFFERENTIAL DIAGNOSIS

The roentgenographic findings of acute carbuncle in the kidney must be distinguished from other interstitial processes that may involve this organ; *e.g.*, edema, interstitial nephritis, infiltrating tumors, etc. In all situations, the combined features noted on preliminary studies of interstitial disease, either diffuse or localized, associated with a perirenal inflammatory process should suggest the diagnosis of an acute carbuncle. Most other interstitial renal diseases do not demonstrate perirenal extension. In the differential diagnosis of renal mass lesions, the findings of the acute renal carbuncle noted at arteriography are characteristic, in which one encounters the absence of mass displacement despite previous features of a mass. The presence of either renal cell or renal pelvic carcinoma is also easily excluded in this phase, since these neoplasias are associated with a specific neovascularity.[7] In some pelvic tumors, particularly of the squamous variety, diffuse interstitial spread may be the only manifestation of the disease, and differentiation from an acute carbuncle may be difficult. However, the acute renal car-

buncle rarely invades the collecting systems directly. The distinction of neoplasm from chronic renal carbuncle, however, may be difficult since the latter has a highly developed abscess wall with neovascularity. Renal vein thrombosis is easily distinguished from the diffuse acute carbuncle when either the collateral venous circulation or the thrombus is demonstrated.

DISCUSSION

From the above findings and an analysis of the patients and their presentation (Table 1), a few interesting clinical and pathologic suggestions are noted. These, however, require further confirmation. In 2 of the 6 cases, the process was overwhelming and involved the entire kidney. Both these patients were males, above 50 years of age, and had prostatic disease, carcinoma in one patient, and benign hypertrophy in the other. The renal infection evolved as an ascending infection in which the causative agents were gram negative in origin.

The remaining 4 renal infections were of hematogenous origin. Interestingly enough, three were females, all in their twenties, and all suffering from drug addiction. The remaining patient was a young male with furunculosis. In contrast to the diffuse renal involvement associated with the ascending infections encountered in the elderly males, the renal carbuncles of hematogenous origin were all localized in nature. It is therefore conceivable that a definite pathologic pattern of the acute renal carbuncle may be present.

As far as the over-all roentgenographic features of renal infection are concerned, most have been confined to the diagnosis of acute pyelonephritis with changes limited to blunting of the calyces and dilatation of the ureter. Although the latter has received a great deal of stress in the literature, the features of acute carbuncle are far more impressive and just as vital. Failure to make a prompt diagnosis can result in loss of either the kidney or the patient. Moreover, the clinical picture may be obscured when the patient is partially treated with antibiotics. This stimulates the carbuncle to further coalesce, wall off, and proceed into the subacute or chronic phase. Inadequate therapy may also allow further perforation of the infection with involvement of surrounding neighboring structures. Consequently, early institution of therapy, either medical or surgical, is imperative and will often salvage the kidney.

The radiologist should now be alerted to this diagnosis, if and when the clinical status suggests an underlying infective process and the roentgen findings are as described above.

SUMMARY

The roentgenographic findings of acute renal carbuncle observed in 6 patients are described. Basically, these findings consisted of:

1. Interstitial disease associated with perirenal involvement demonstrated on preliminary studies;

2. No mass displacement of the vessels in the involved area on angiography despite the suggestion of a mass on the earlier examinations;

3. Loss of the cortico-medullary junction on the nephrogram.

The early diagnosis of acute carbuncle is essential since prompt therapy may salvage the kidney. With the ever increasing problem of drug addiction and its associated complications, acute renal carbuncle is slowly becoming a common disease of youth.

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REFERENCES

1. Bosniak, M. A. Personal communication.
2. Caplan, L. H., Siegelman, S. S., and Bosniak,

M. A. Angiography in inflammatory space-occupying lesions of kidney. *Radiology*, 1967, *88*, 14–23.

3. Coel, M. N., and Talner, L. B. Obstructive nephrogram due to renal vein thrombosis. *Radiology*, 1971, *101*, 573–574.
4. Emmet, J. L., and Witten, D. M. Clinical Urography. Volume 2. W. B. Saunders Company, Philadelphia, 1971.
5. Evans, J. A., Meyers, M. A., and Bosniak, M. A. Acute renal and perirenal infections. *Seminars in Roentgenol.*, 1971, *6*, 274–291.
6. Himmelfarb, E., Rabinowitz, J. G., Kinkhabwala, M. N., and Becker, J. A. Roentgen features of renal carbuncle. To be published.
7. Rabinowitz, J. G., Kinkhabwala, M., Himmelfarb, E., Robinson, T., Becker, J. A., Bosniak, M. A., and Madayag, M. M. Renal pelvic carcinoma: angiographic re-evaluation. *Radiology*, 1972, *102*, 551–554.
8. Robbins, S. L. Pathology. Third edition. W. B. Saunders Company, Philadelphia, 1967.

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ROENTGEN APPEARANCE OF BONE AND SOFT TISSUE CHANGES IN CHRONIC RENAL DISEASE*

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RENAL disease may be primarily tubular or glomerular. Tubular syndromes, such as vitamin D resistant rickets, renal tubular acidosis, and Fanconi syndrome result in loss of phosphate and other metabolites in the urine. The resultant hypophosphatemia leads to rickets and osteomalacia. Although osteitis fibrosa is said to sometimes complicate these conditions, as well as nutritional rickets, it is not predominantly a part of the roentgen spectrum.

Chronic glomerular renal disease with reduced renal mass and reduced glomerular filtration rate, however, produces a wide spectrum of roentgenologically detectable changes in bone and soft tissue.

The purpose of this paper is to describe the various types of roentgen changes in the latter condition, and to analyze the bases of these changes.

MATERIAL

The roentgenograms of 25 patients currently on the renal hemodialysis program at Mount Sinai Hospital were reviewed. Of these, 19 cases, or 76 per cent, showed roentgenographic bone changes, with 6 cases demonstrating additional soft tissue calcifications; and 6 cases, or 24 per cent, showing no detectable bone or soft tissue changes.

PATHOPHYSIOLOGY

Chronic glomerular renal disease with reduced renal mass and reduced glomerular filtration rate manifests itself in 2 basic forms: resistance to vitamin D; and reduced renal clearance of phosphate[8] (Fig. 1).

Resistance to vitamin D can begin in the early phase of chronic renal failure and become increasingly severe as the disease progresses.[8] The resultant effect of this phenomenon is rickets in the immature skeleton and osteomalacia in the adult. The mechanism in renal hyperphosphatemic disease is not similar to that in vitamin D deficient rickets or tubular disease with hypophosphatemia, where bone changes are due to a low calcium-phosphate ion product. This product may be elevated in patients with chronic renal failure. There are several theories of the manner in which bone matrix mineralization is blocked. Avioli and Slatopolsky, cited by Robinson,[12] proposed that there is an abnormal metabolism of vitamin D_3 in patients with chronic renal failure. This is responsible for decreased intestinal absorption of calcium. Vitamin D is also implicated as having a direct effect on osteoid mineralization. The production of vitamin D antagonists, as well as failure of end-organ response, has also been implicated.

Reduced glomerular filtration rate results in reduced phosphate renal clearance. This causes a rise in the phosphate level in body fluids. A significant inverse correlation is present between free calcium ion concentration and diffusible inorganic phosphate concentration in severe renal failure.[8] Plasma calcium ion concentration consequently decreases. By a direct negative feedback mechanism in response to hypocalcemia, the parathyroid glands may develop hyperplasia and hypertrophy with an increased secretion of parathormone. There is a suggestion that the plasma magnesium concentration may also be implicated with an increase causing inhibition, and a decrease causing stimulation of parathyroid secretion.[8] This leads to increased circulating parathyroid hormone which is the true state of secondary hyperparathyroidism.

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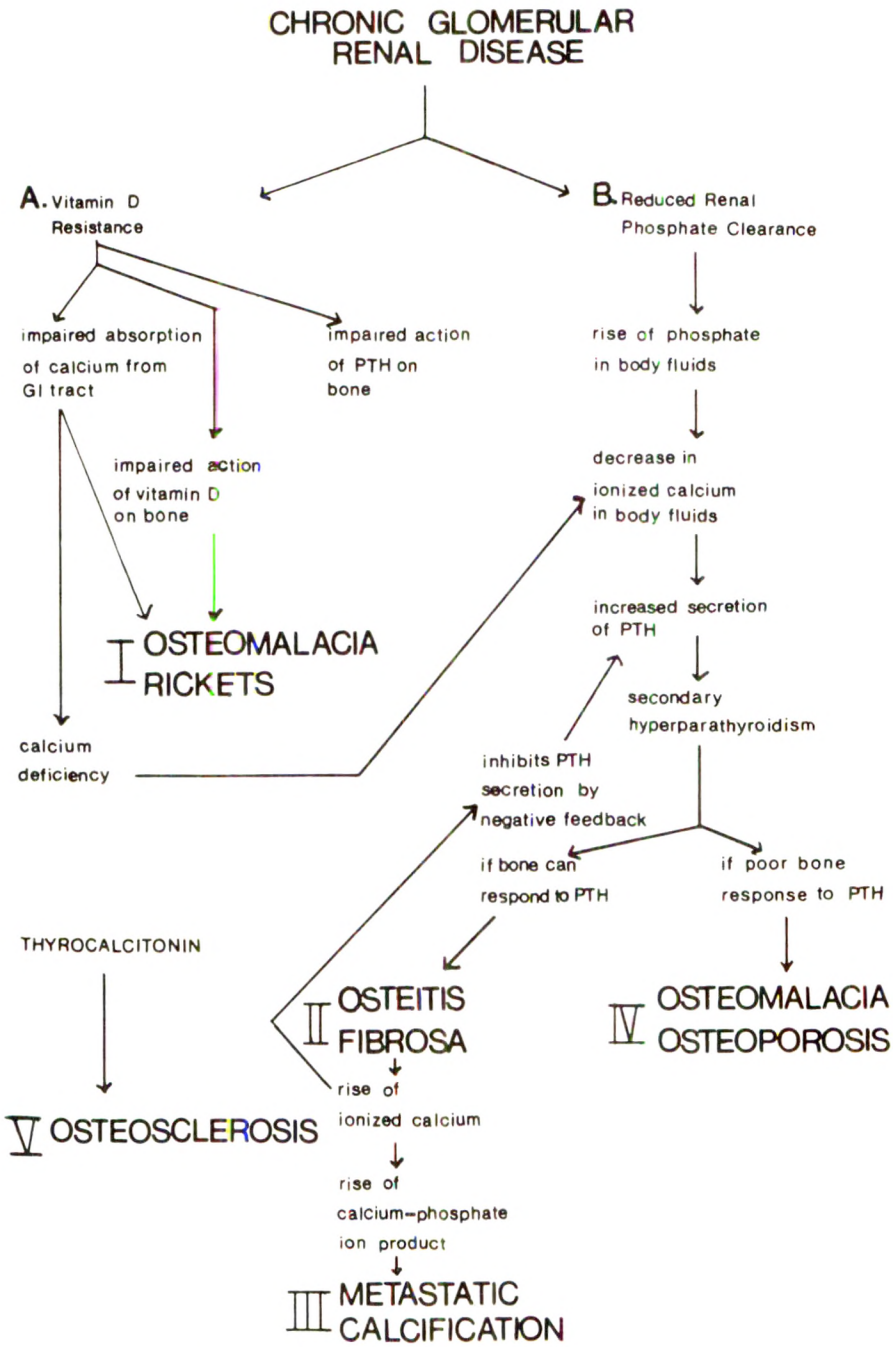


FIG. 1. Classification of bone and soft tissue changes in chronic glomerular renal disease. GI = gastrointestinal; PTH = parathyroid hormone; MET = metastatic.

The bone may respond to parathormone in various degrees. If the bone is able to respond with an increased activity of osteoclasts and probably osteocytes,[8] then the resultant effect is osteitis fibrosa. This leads to release of calcium. Parathormone also inhibits net tubular phosphate resorption, leading to an increase in urinary phosphate

which, however, is limited by renal failure. A rise in the calcium-phosphate ion product leads to metastatic calcification. The increase in serum calcium concentration exerts an inhibitory effect on parathormone secretion.

The bone may respond poorly to parathormone. In 1948, Albright and Reifenstein suggested that patients with osteomalacia fail to show a response to parathormone because this bone is covered with osteoid tissue which prevents resorption. The coating of unmineralized osteoid tissue over all bony surfaces makes mineralized tissue unavailable to maintain normocalcemia. Failure of calcification over an extended period may produce uncalcifiable osteoid.[7] Another factor is the possibility that the parathormone in patients with chronic renal failure is altered, and that the observed skeletal resistance to parathormone is in part due to this alteration.[8]

If there is lack of development of osteitis fibrosa, the predominant roentgen picture may be osteomalacia or osteoporosis.

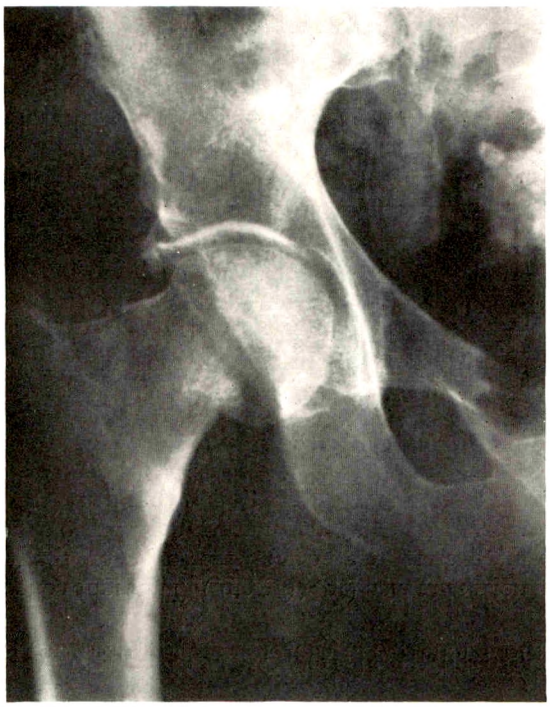


Fig. 2. Osteomalacia with fracture of the right femoral neck.

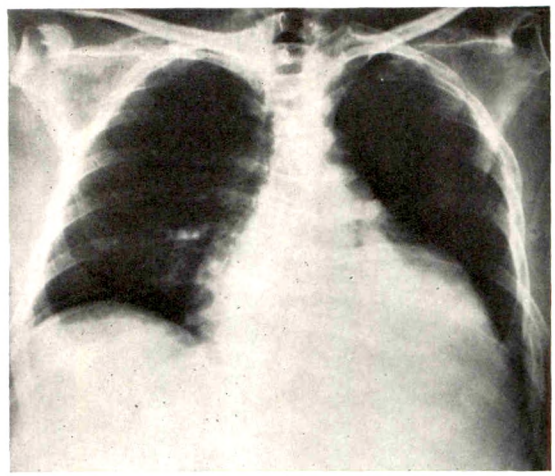


Fig. 3. Osteomalacia of the chest demonstrating multiple rib fractures.

Osteoporosis may be on the basis of the general condition of the patient or local immobilization due to bone pain or fracture.

Some patients with renal disease have an absolute overproduction of bone matrix, which may be mineralized sufficiently, so that the bones appear excessively dense on roentgenograms.[12] In 1932, Selye gave mice small doses of parathormone for an extended period of time and observed osteosclerosis. This has also been more recently observed with parathyroid administration in thyroidectomized rats.[15] Thyrocalcitonin has been the subject of much recent investigation, and has been shown to cause osteosclerosis.

In severe renal failure of long duration, elevation of the serum calcium concentration may not exert an inhibitory effect on parathyroid secretion. In this event, we speak of autonomy of the parathyroid gland, or tertiary hyperparathyroidism.

The incidence of bone changes in chronic renal disease can approach 25 per cent in untreated cases. In dialysis programs, bone changes become more severe as the dialysis progresses and the incidence can approach 80 to 90 per cent.

In successful renal transplantation, the visible changes tend to regress. A complication of renal transplantation, ischemic ne-

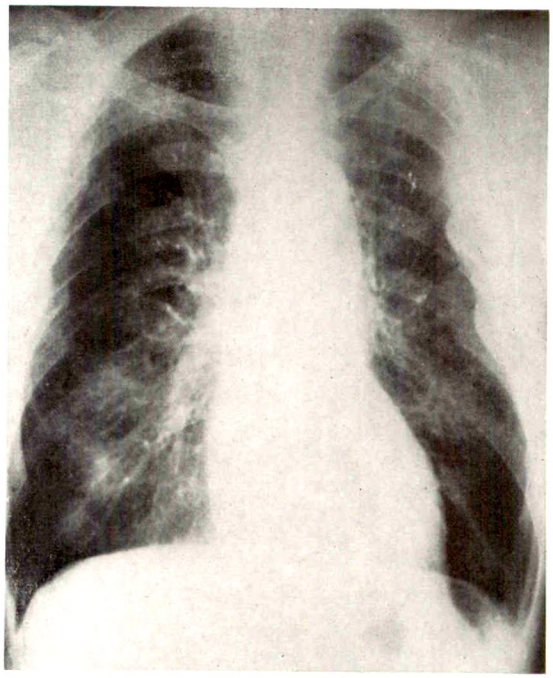


FIG. 4. Osteomalacia of the chest demonstrating multiple healed rib fractures with deformity of the thorax.

crosis of bone involving several joints, has been reported in a high percentage of cases.[2]

ROENTGENOGRAPHIC FINDINGS

It can thus be seen that the spectrum of changes includes osteomalacia, osteitis fibrosa, osteoporosis, osteosclerosis, metastatic calcification, rickets, if in the immature skeleton, and possibly ischemic necrosis if renal transplantation has been performed.

OSTEOMALACIA

Osteomalacia is characterized by the presence of nonmineralized osteoid tissue, which leads to loss of bone density, deformity, and structural weakness of bone. There may be coarsening of the trabecular pattern with thinning and lack of definition of the cortex. Deformities, particularly a "bell-shaped thorax" and a "tri-radiate pelvis" are seen as well as deformities of the long bones.

Pseudofractures, which most likely represent incomplete fractures, are present. Many of these progress to true fractures (Fig. 2). A common occurrence is deformity of the chest, with multiple fractures of the ribs (Fig. 3; and 4). Pseudofractures may also be seen in the scapulae, femora, pubes, and ischia (Fig. 5, *A* and *B*). They sometimes have a symmetric distribution. Pseudofractures are not limited to osteomalacia. In the immature skeleton, a rachitic pattern, at times with pseudofractures, may be seen (Fig. 6).

OSTEITIS FIBROSA

Osteitis fibrosa is based on osteoclastic and possibly osteocytic bone resorption and subsequent replacement with fibrous tissue. It is due to hyperparathyroidism, either primary or secondary. The roentgen changes are essentially similar in both. The roentgenographic hallmark of hyperparathyroidism involving bone with osteitis fibrosa is subperiosteal bone resorption. This has been documented in no other condition than hyperparathyroidism. The earliest changes are seen at the radial margins of the middle phalanges at the metaphyseal-

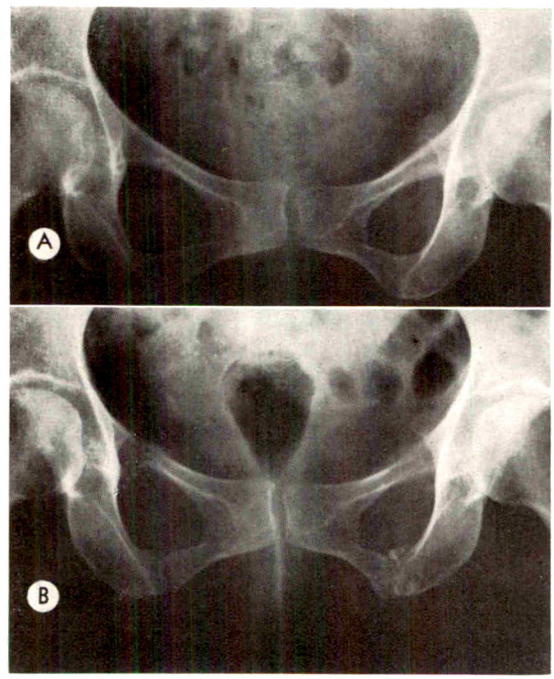


FIG. 5. Osteomalacia. (*A*) The pelvis is normal. (*B*) Same patient 13 months later. There are symmetric "pseudofractures" at the ischia and pubic bones.

diaphyseal junction (Fig. 7). Another common area of bone resorption is at the distal and inferior aspects of the clavicles (Fig. 8). These changes were first reported by Camp and Ochsner and described by Pugh as "A peculiar lace-like appearance of the bone beneath the periosteum." Recent studies show that the magnification technique with industrial film in roentgenographing the hand reveals earlier changes than conventional techniques.[10] Other common sites of bone resorption are: the proximal medial aspects of the humeri, tibiae, and femora; the upper margins of the ribs, the symphysis pubis, the ischia and the calcaneus; and the terminal tufts of the distal phalanges.

The skull may show a granular effect with loss of density. Resorption of the lamina dura, or the cortex of the tooth socket, may also be seen without widening of the periodontal membrane. This finding is not specific and can be seen in other conditions. There may be generalized loss of bone density, accentuation of the trabecular pattern, or, conversely a "ground-glass" appearance. Rarely "brown tumors" or cysts may be associated with secondary hyperparathyroidism, although these are much more common in the primary form (Fig. 9). They present as expansile lesions, may occur anywhere in the skeleton, show no periosteal reaction, and may present with a pathologic fracture.

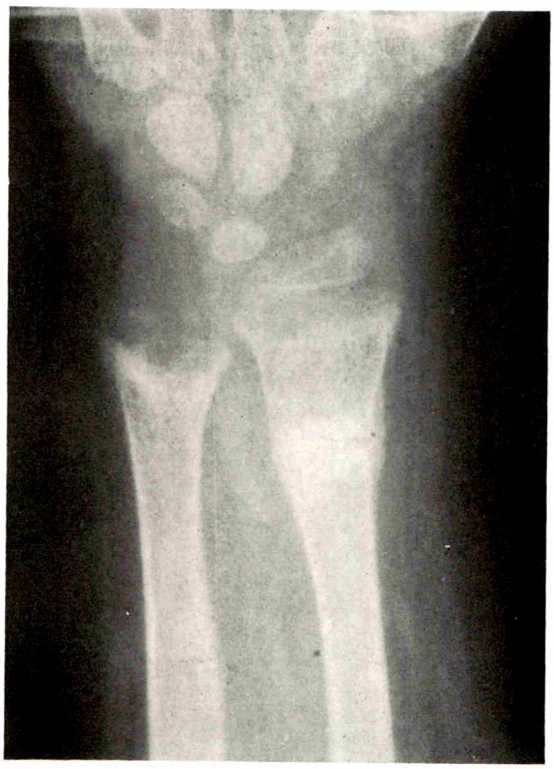


Fig. 6. Renal rickets. There is widening of the epiphyseal cartilage plate with cupping and fraying of the metaphysis. A "pseudofracture" of the distal radius is present.

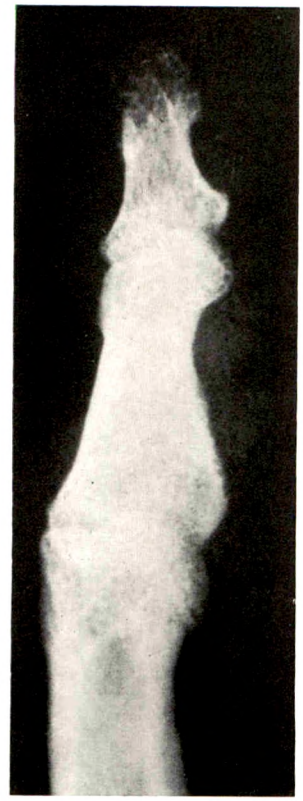


Fig. 7. Osteitis fibrosa; early change. There is minimal subperiosteal bone resorption of the radial side of the middle phalanx at the metaphyseal-diaphyseal junction.

OSTEOPOROSIS

Osteoporosis is based on loss of bone mass with normally structured residual bone present. Roentgenographically, several osteoporotic patterns can be discerned. Developing severe osteoporosis is characterized by a permeative radiolucent pattern (Fig. 10). A periarticular distribution of radiolucency may also be seen (Fig. 11), as well as a uni-

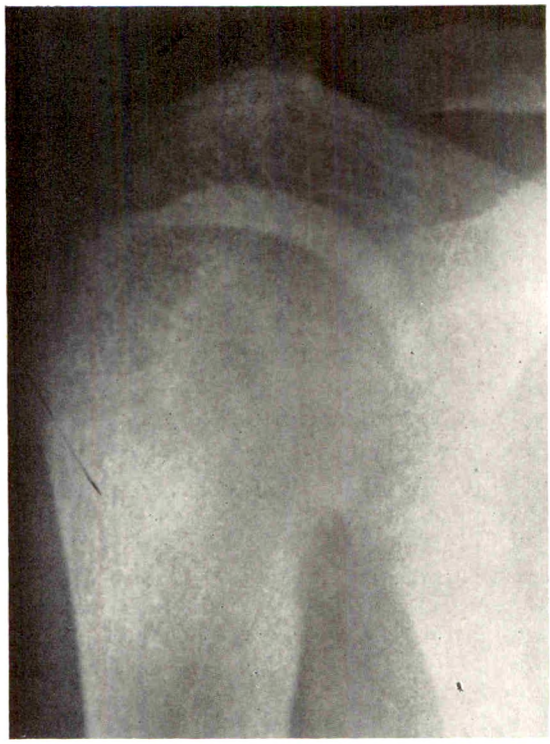


FIG. 8. Osteitis fibrosa. There is resorption of the distal clavicle as well as the upper medial cortex of the humerus.



FIG. 9. Osteitis fibrosa. Expansile lesion at the base of the fourth metacarpal which is probably a cyst or brown tumor. The lesion was not biopsied.

form loss of bone density in established osteoporosis. The cortex is sharp and thin. The bone trabeculae may be accentuated, particularly along lines of stress. There is loss of structural strength, and fractures, particularly in the spine, may be present. The osteoporosis seen in chronic renal disease may be due to general causes, or, locally, secondary to bone pain or fracture resulting in immobilization.

OSTEOSCLEROSIS

Increase in bone density may be seen in chronic renal disease. There may be a uniform increase, particularly in the axial skeleton (Fig. 12; and 13). A common pattern is osteosclerosis in the spine with the ends of the vertebral bodies showing an increase in density relative to the centers, but with a uniform gradient. This appearance has been called "rugger jersey spine." Osteosclerosis can also be seen as increased



FIG. 10. Permeative osteoporotic pattern in osteomalacia patient secondary to immobilization from hip fracture.

density with a generalized coarsening of the trabecular pattern.

SOFT TISSUE CALCIFICATION

Local and systemic factors interplay in the picture of soft tissue calcification in chronic renal disease.[11] The most frequent sites of soft tissue calcification are: arterial, ocular, periarticular, cutaneous, subcutaneous, and visceral.

There are 2 types of arterial calcification that can be roentgenologically recognized, atheromatous and medial sclerosis. Arterial calcification seen in this condition is almost always medial sclerosis, resulting in a usual absence of ischemia. Digital necrosis may develop in rare instances. Corneal and conjunctival calcifications are the most common sites of calcification in patients with long-term dialysis.

Articular chondrocalcinosis occurs in primary hyperparathyroidism, but is rare in chronic renal disease with secondary hyperparathyroidism. Patients on long-term hemodialysis may rarely contract acute inflammatory joint involvement. Some of the lesions may represent gout. In other instances there is no evidence of gout or pseudogout.



FIG. 11. Hand. Periarticular osteoporotic pattern.



FIG. 12. Uniform osteosclerosis of the lumbar spine. A "rugger-jersey" pattern is suggested in the lower vertebra.

Periarticular calcification may appear following the inflammatory stage (Fig. 14). Subcutaneous and cutaneous calcifications may be associated with severe pruritus. The lesions may be nodular and be deposited in the periarticular regions, or may take the form of subcutaneous plaques with discoloration of the overlying skin.

Visceral calcification may occur in the lungs, kidneys, stomach, heart, and, rarely, the liver. Calcifications in the lungs occur in the pulmonary alveolar septa and are

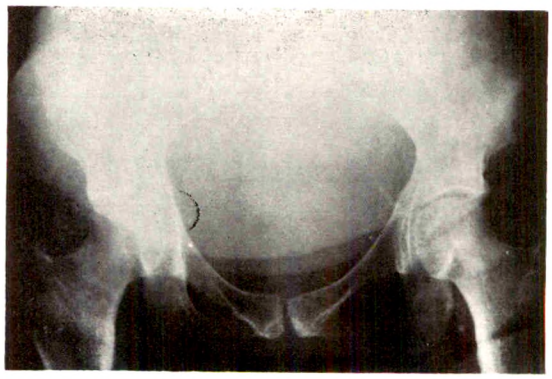


Fig. 13. Osteosclerosis of the pelvis associated with calcification of the left sacro-spinous ligament in a patient with chronic renal disease. These findings were formerly thought to be characteristic of fluorosis.

usually not visible roentgenographically, but may produce a fine reticular, punctate, nodular or segmental opacity. Severe myocardial calcification can be a major cause of death. Nephrocalcinosis is frequent.

ISCHEMIC NECROSIS

Ischemic necrosis has been reported following renal transplantation. This may be the result of immunosuppressive therapy. The roentgen appearance follows the usual course of the subarticular fracture and bone collapse. The sites frequently involved are: the hip, knee, and humeral head.[2]

DISCUSSION

The roentgenographic changes in bone and soft tissue in chronic glomerular renal disease can be seen as a highly complex picture with a multiplicity of factors involved. Early in the course of renal failure, a picture of osteomalacia may predominate. If bone can respond to parathormone, then the effects of secondary hyperparathyroidism will be pre-eminent. There may be an underlying osteoporosis due to general lack of mobility or to local factors of immobilization. In the immature skeleton, osteomalacia manifests itself as rickets with the typical widening of the epiphyseal cartilage plate, and cupping and fraying of the metaphysis. This may be seen in combination with subperiosteal bone resorption and pseudofractures.

Osteosclerosis can complicate the picture as well as soft tissue calcification. Those patients on renal dialysis have progressive changes of osteitis fibrosa. Following successful renal transplantation, the majority of patients become euparathyroid in 6 to 18 months with regression of changes of osteitis fibrosa.[6] A complication of renal transplantation is ischemic necrosis of bone.

The term renal osteodystrophy is some-

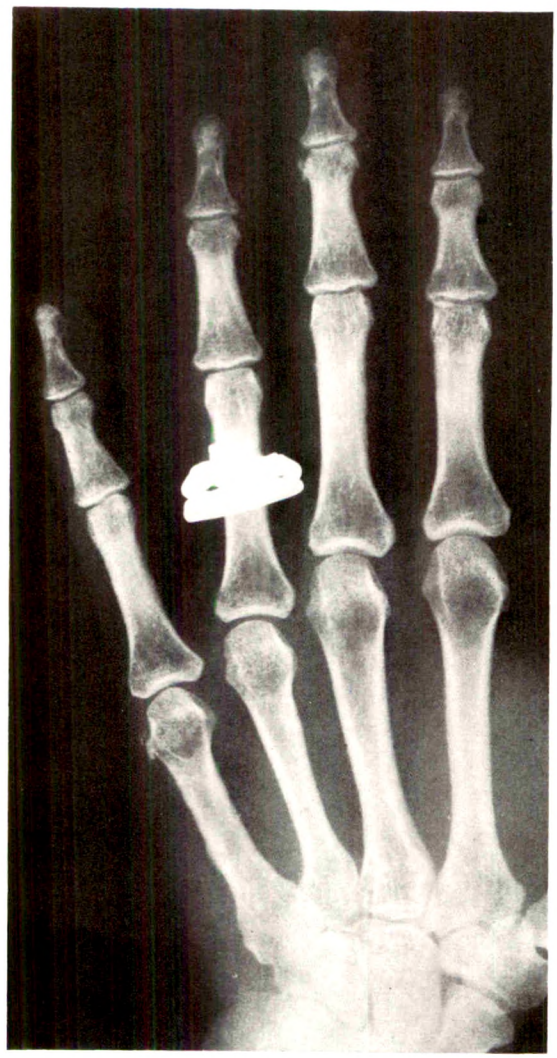


Fig. 14. Hand. Periarticular soft tissue calcifications of the distal interphalangeal joint of the middle finger and at the fifth metacarpo-phalangeal joint are noted.

times used to describe the bone changes in this condition. This term has several critics,[1] as osteodystrophy is not an appropriate term for the mature skeleton. Perhaps a more suitable term would be renal osteopathy.

SUMMARY

The basic pathophysiology and roentgen appearances of bone and soft tissue changes in chronic glomerular renal disease are described.

These form a complex picture composed of: osteomalacia; osteitis fibrosa; osteoporosis; osteosclerosis; and soft tissue calcifications.

The manifestations of each condition are discussed and illustrated.

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We thank Mrs. Sharon Cooper for preparing Figure 1.

REFERENCES

1. Aegerter, E., and Kirkpatrick, J. A., Jr. Orthopedic Diseases. Third edition. W. B. Saunders Company, Philadelphia, 1968, pp. 400.
2. Cruess, R. L., Blennerhassett, J., Macdonald, F. R., Maclean, L. D., and Dossetor, J. Aseptic necrosis following renal transplantation. *J. Bone & Joint Surg.*, 1968, *50-A*, 1577–1590.
3. Fine, R. N., Isaacson, A. S., Payne, V., and Grushkin, C. M. Renal osteodystrophy in children: effect of hemodialysis and renal homotransplantation. *J. Pediat.*, 1972, *80*, 243–249.
4. Fordham, C. C., and Williams, T. F. Brown tumor and secondary hyperparathyroidism. *New England J. Med.*, 1963, *269*, 129–131.
5. Greenfield, G. B. Radiology of Bone Diseases. J. B. Lippincott Company, Philadelphia, 1969, pp. 465.
6. Hampers, C. L., Katz, A. J., Wilson, R. E., and Merrill, J. P. Calcium metabolism and osteodystrophy after renal transplantation. *A.M.A. Arch. Int. Med.*, 1969, *124*, 282–291.
7. Jowsey, J., Massry, S. G., Coburn, J. W., and Kleeman, C. R. Microradiographic studies of bone in renal osteodystrophy. *A.M.A. Arch. Int. Med.*, 1969, *124*, 539–542.
8. Kleeman, C. R., Massry, S. G., Coburn, J. W., and Popovtzer, M. M. Divalent ion metabolism and osteodystrophy in chronic renal failure. Session 1: Uremic osteodystrophy–basic concepts and clinical considerations, problem and unanswered questions. *A.M.A. Arch. Int. Med.*, 1969, *124*, 262–268.
9. Massry, S. G., Coburn, J. W., Popovtzer, M. M., Shinaberger, J. H., Maxwell, M. H., and Kleeman, C. R. Secondary hyperparathyroidism in chronic renal failure: clinical spectrum in uremia, during hemodialyses, and after renal transplantation. *A.M.A. Arch. Int. Med.*, 1969, *124*, 431–441.
10. Meema, H. E., Rabinovich, S., Meema, S., Lloyd, G. J., and Oreopoulos, D. G. Improved radiological diagnosis of azotemic osteodystrophy. *Radiology*, 1972, *102*, 1–10.
11. Parfitt, A. M. Soft tissue calcification in uremia. *A.M.A. Arch. Int. Med.*, 1969, *124*, 544–556.
12. Robinson, R. A. Ultrastructural appearance of bone cells and matrix in renal osteodystrophy. *A.M.A. Arch. Int. Med.*, 1969, *124*, 519–529.
13. Stanbury, S. W. Bone disease in uremia. *Am. J. Med.*, 1968, *44*, 714–724.
14. Stanbury, S. W., Lumb, G. A., and Mawer, E. R. Osteodystrophy developing spontaneously in course of chronic renal failure. *A.M.A. Arch. Int. Med.*, 1969, *124*, 274–281.
15. Steiner, R. E. Physiological approach to radiology. *Radiology*, 1971, *100*, 497–508.
16. Weller, M., Edeiken, J., and Hodes, P. J. Renal osteodystrophy. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1968, *104*, 354–363.

RENAL ANGIOGRAPHY IN PATIENTS WITH HEMATURIA*

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IN SPITE of improved chemical, cytologic and roentgenologic methods, the group of patients with unexplained micro- and macroscopic hematuria remains large. In many of these cases persistent hematuria leads to repeated roentgenologic examinations, including angiography.

The purpose of the author was to determine the indications and value of renal angiography in patients with hematuria.

MATERIAL AND METHOD

A retrospective study was made of 120 patients examined by renal angiography during a 6 year period (1964–1969) in our department. In all cases clinical and laboratory examinations prior to angiography gave no reasonable explanation for the hematuria. All but 2 cases had active hematuria at the time of the angiographic examination. In 100 cases (Group I) urographic studies were quite normal; in 43 of these cases renal phlebography was also performed. In 20 cases (Group II) there were slight, nonspecific changes at urography, which were not considered to be the cause of the hematuria. These changes included: obstruction; filling defects considered to be clots; and slight irregularities in the shape of the kidney pelvis due to imperfect technique, arousing slight tumor suspicion. In 5 of these cases renal phlebography was performed. All patients, except 5 in Group I, were examined with selective renal angiography, the 5 being examined with total renal angiography.

This investigation does not attempt to give the over-all percentage distribution of the causes of hematuria. Those patients in whom the cause of their hematuria was obvious at urography or other examinations are not included.

The age of the patients was between 9 and 78 years; 70 men and 50 women were studied.

RESULTS

GROUP I. 100 PATIENTS WITH NORMAL UROGRAPHY

Since no significant difference in the angiographic findings could be found with regard to macroscopic and microscopic hematuria, no distinction is made between these groups in the results presented in Table I. Final clinical diagnosis was:

1. *Glomerulonephritis*. Fifteen cases were verified by biopsy and 9 cases were diagnosed by typical clinical course. In only 4 cases were angiographic changes seen, with slight general thinning of the cortex and an indistinct boundary between cortex and medulla. In 1 case there were changes suggesting fibrolipomatosis and in 2 cases arteriovenous fistulae were seen as a result of previous renal biopsy.

2. *Interstitial nephritis*. All 6 cases were confirmed by biopsy. In only 1 case were angiographic changes seen and these consisted of slightly reduced thickness of the cortex in both kidneys and slightly irregular renal surface. In addition, this patient had a cyst in one kidney, initially missed at urography.

3. *Pyelonephritis*. We studied 3 cases: in 2 the diagnosis was verified by biopsy. In 1 there was slight general thinning of the cortex and a poorly defined border between the cortex and the medulla. One year later a further angiographic study was performed and a slight, local induration of the left kidney had occurred. Biopsy revealed chronic glomerulonephritis and pyelonephritis. The other biopsy-verified case had a focal cortical scar measuring 2 cm. This was also found on renal phlebography

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TABLE I

GROUP I. 100 PATIENTS WITH NORMAL UROGRAPHY

Diagnosis	Total No. of Cases	Macroscopic Hematuria	Microscopic Hematuria	Mean Age (yr.)	Histologic Verification	Angiography		Phlebography		Comments
						Normal	Abnormal	Normal	Abnormal	
Glomerulonephritis	24	11	13	28	15	17	7	7		Pathologic angiography: 1 case, fibrolipomatosis; 2 cases, A-V fistula after biopsy; 4 cases, cortex reduction
Interstitial nephritis	6	3	3	27	6	5	1	3		Pathologic angiography: reduced cortex+cyst in right kidney
Pyelonephritis	3	3		36	2	1	2	1	2	Pathologic angiography: 2 cases, reduced cortex; same finding at renal phlebography (in 1 of these cases there were signs of recanalized vein thrombosis at renal phlebography)
Vascular changes in kidney	15	8	7	48	6	8	7	4	4	Pathologic angiography: 2 cases, angioma; 1 case, infarction; 1 case, aneurysm+nephrosclerosis; 2 cases, renal arterial stenosis; 1 case, fibromuscular hyperplasia Pathologic phlebography: 3 cases, kidney pelvis varices; 1 case, phlebitis
Coagulation disturbances	5	4	1	27		5		3		
Urinary tract disease (including calculus)	22	12	10	36	1	22		8		3 cases, prostatitis; 11 cases, cystitis-urethritis; 4 cases, bladder varices; 4 cases, calculus
Hematuria (unexplained)	25	16	9	33	3	25		11		
Total	100	57	43		33	83	17	37	6	

A—V = arteriovenous.

together with signs of recanalized renal vein thrombosis. In the third case angiography was normal, but at urography 8 months later the cortex in the left upper pole was reduced and changes of papillary necrosis were demonstrated.

4. *Vascular changes in the kidneys.* In 15 cases changes were found in the vasculature of the kidneys—changes which should reasonably account for the hematuria. In 4 of these 15 cases angiographic studies were normal, but hemangiomas or arteriovenous malformations were found at pathologic examination after nephrectomy. Nephrectomy was performed because of persistent hematuria, which resulted in anemia. In 11, angiographic studies (including renal phlebography) gave a definite diagnosis.

In 2 cases, a diagnosis of hemangioma or arteriovenous malformation was established (Fig. 1).

In 3, renal phlebography demonstrated varices of the renal pelvis (Fig. 2, *A* and *B*).

In 1 case, renal phlebography showed changes consistent with renal vein phlebitis (Fig. 3).

Other angiographic findings consisted of 2 cases with renal artery stenosis, 1 of aneurysm of the dorsal renal artery, 1 of minor renal infarction, and 1 of marked fibro-



Fig. 1. I.S., 60 year old female with persistent microscopic hematuria found on routine examinations. Angiography revealed a hemangioma in the lower pole of the left kidney, which later was resected. Microscopic diagnosis: hemangioma with arteriovenous anastomosis.

muscular hyperplasia of both renal arteries.

5. *Coagulation disturbances.* Many cases had been studied for coagulation disturbances. In 5, disturbances in the coagulation mechanism explained the hematuria. Two cases had von Willebrand's disease, 1 depression of hemo-phili-B-factor, and 1 depression of prothrombin and proconvertin and pathologic prothrombin consumption. The remaining case had a confusing disturbance in the coagulation mechanism probably due to collagenosis. In all 5 cases angiographic studies were normal.

6. *Other abnormalities of the urinary tract.* In this group are included patients with cystitis-urethritis (11 cases), stone formation (4 cases), varices or other vascular changes within the bladder or prostate (4 cases) and prostatitis (3 cases). In those cases with a diseased lower urinary tract cystoscopy gave a definitive diagnosis. As a rule cystoscopy was performed before angiography, but the bleeding was not considered to be fully explained by the cystoscopic findings. Angiographic studies were normal in all these cases. Of the 4 cases with urinary tract calculi, initial urographic studies were normal, but on repeat examination between 6 weeks and 2 years later, small calculi measuring less than 6 mm. were demonstrated. The repeat urographic examinations were made because of persistent hematuria.

7. *Essential hematuria.* The largest group consists of patients with hematuria for which no cause could be found. Sixteen patients had macroscopic and 9 microscopic hematuria. In all these cases angiography

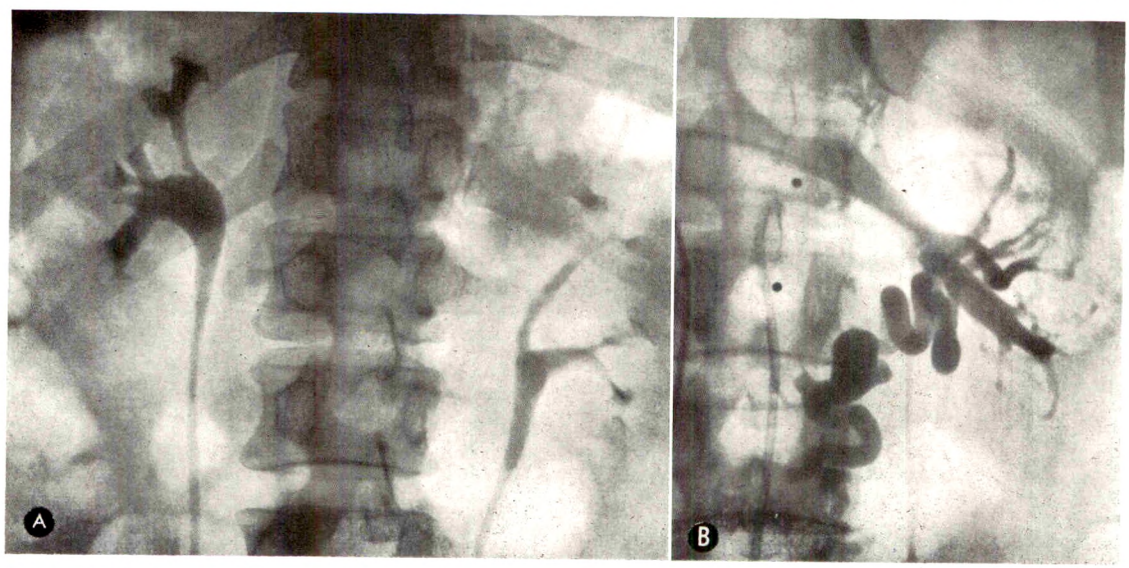


Fig. 2. G.H., 27 year old male with repeated attacks of macroscopic hematuria. (*A*) Normal urogram. There are no indentations in the ureter. (*B*) Renal phlebogram. A tortuous varicose vein is seen surrounding the kidney pelvis and the proximal part of the ureter.

was normal. Renal phlebography was performed in 11, all with normal findings. In 3 cases renal biopsy was performed with normal findings.

In the follow-up of 32 patients with initially unexplained hematuria, calculus was seen in 2 patients 8 months and 2 years after the first urographic study. In 5 cases renal biopsy was positive ½–3 years after the first angiographic study. Of these, the diagnosis was chronic glomerulonephritis in 4 cases and interstitial nephritis in one.

GROUP II. 20 PATIENTS WITH NONSPECIFIC CHANGES

Of the 20 cases, in which urographic studies showed nonspecific changes, the final clinical diagnosis was (Table II):

1. *Glomerulonephritis*. One case. At angiography bilateral changes characteristic of nephrosclerosis were found.

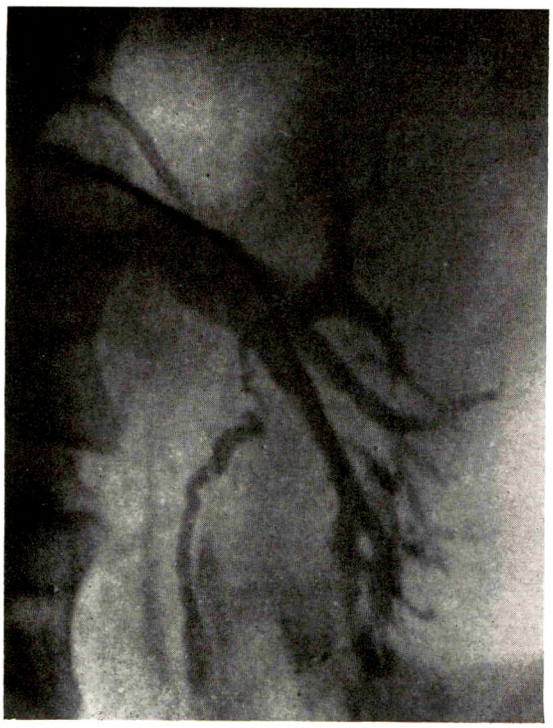


FIG. 3. J.N., 19 year old male with progressive ankylosing spondylitis from the age of 15 years. At repeated routine examinations, microscopic hematuria was found.

Renal phlebogram. In the medial part of the kidney there is filling of an irregular vein, considered to represent a renal vein phlebitis.

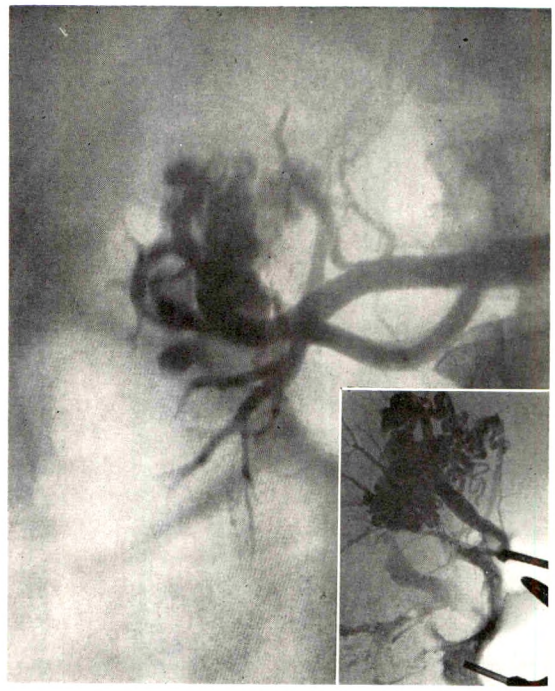


FIG. 4. I.L., 52 year old female with sudden painless macroscopic hematuria. At urography obstruction of the right kidney was found. Pyelography 1 day later revealed dilated right ureter, filled with clots.

Angiogram demonstrates a large arteriovenous malformation in the right kidney. Inset. Angiogram of surgical specimen after nephrectomy. Microscopic diagnosis: arteriovenous angioma.

2. *Interstitial nephritis*. One case. Angiography was normal. Diagnosis was made by biopsy.

3. *Pyelonephritis*. One case. Angiography was normal.

4. *Vascular changes in the kidneys*. Four cases. In 2 of these, hemangiomas were found at angiography (Fig. 4), and in 1 several small arterial aneurysms in peripheral intrarenal vessels were seen (Fig. 5). These changes had the appearance of periarteritis nodosa.[6] The fourth patient had an arteriosclerotic aneurysm and an arteriovenous anastomosis was found at angiography.

5. *Other abnormalities of the urinary tract*. The urographic findings and the final clinical diagnosis are presented in Table III. Various abnormalities were observed. In 1 patient the filling of the calyces of the left

TABLE II

GROUP II. 20 PATIENTS WITH NONSPECIFIC UROGRAPHIC CHANGES

Diagnosis	Total No. of Cases	Macroscopic Hematuria	Microscopic Hematuria	Mean Age (yr.)	Histologic Verification	Angiography		Phlebography		Urography Findings	Comments
						Normal	Abnormal	Normal	Abnormal		
Glomerulonephritis	1		1	57	—		1	1		Incomplete filling of renal pelvis	Nephrosclerosis
Interstitial nephritis	1	1		39	1	1				Obstruction	Normal
Pyelonephritis	1	1		9		1				Irregularities in kidney pelvis	Normal
Vascular changes in kidney	4	4		44	2	1	3			1 case, dilatation of kidney pelvis; 2 cases, obstruction; 1 case, slight obstruction + clots in kidney pelvis	Pathologic angiography: 1 case, several arterial aneurysms; 2 cases, hemangioma; 1 case aneurysm + A-V shunt
Urinary tract disease	8	5	3	50	3	7	1	4		Table III	Table III
Renal tumor	3	3		51	3		3			1 case, obstruction; 1 case, slight deformation of calyces; 1 case, lowered contrast concentration	Pathologic angiography: 1 case, carcinoma; 2 cases, cyst
Hematuria (unexplained)	2	2		48		2				1 case, horse-shoe kidney; 1 case, slight deformation of renal pelvis	Angiography: 1 case, horse-shoe kidney; 1 case, normal
Total	20	16	4			12	8	5			

kidney was imperfect at urography, and a renal tumor could not be excluded. Angiography was normal. Cystoscopy was performed 10 days later due to persistent microscopic hematuria; a bladder tumor, which proved to be a highly differentiated papillary carcinoma, was seen.

6. *Tumor.* One patient had renal carcinoma. At urography left-sided obstruction was seen. A control examination 2

TABLE III

URINARY TRACT DISEASE

Patient	Clinical Diagnosis	Finding at Urography	Angiography
G.M.	Carcinoma of urinary bladder	Incomplete filling of kidney pelvis	Normal
Y.W.	Prostatitis	Dislocation of calyces	Normal
E.M.	Prostate bleedings (+hydronephrosis)	Left-sided hydronephrosis	Hydronephrosis
A.N.	Cystitis	Filling defect of kidney pelvis	Normal
K.F.	Urethritis	Broadening of kidney	Normal
E.A.	Nephrolithiasis	Filling defect of kidney pelvis	Normal
A.S.	Nephrolithiasis (+carcinoma of prostate)	Slight dislocation of calyces	Normal (2×2 mm. calculus)
T.W.	Uretherolithiasis	Obstruction without concrement	Normal

weeks later was considered to be normal (Fig. 6*A*). As the bleeding from the urinary tract persisted, angiography was performed to reveal a carcinoma in the left lower kidney pole (Fig. 6*B*). The group includes 2 patients with a renal cyst.

7. *Essential hematuria.* Two patients. At urography a horse-shoe kidney was seen in one and a slight dislocation of one calyx in the other. In both cases angiography was normal.

DISCUSSION

Patients with either macroscopic or microscopic hematuria present considerable difficulties in their diagnoses.

If, at cystoscopy, a convincing source of bleeding is found in the bladder, unnecessary roentgenologic examinations can be avoided.

Even though a pathologic lesion, for instance cystitis, is seen at cystoscopy, it is not necessarily responsible for the bleeding. Each case must be carefully reviewed to determine whether the investigation must include angiography. On completing all examinations an over-all evaluation can be made.

In the present selected material of 120 patients, the diagnosis had been arrived at by renal angiography in 18 cases (vessel abnormalities and renal tumors or cysts), while in 11 cases other pathologic changes have been found; *i.e.*, reduced thickness of the cortex. In 4 of the 48 cases examined with renal phlebography the cause of the bleeding could be explained. It must be noted that about $\frac{1}{4}$ of the cases in this material suffered from disorders undetectable by means of angiography, such as diseases of the lower urinary tract, calculus and disturbances of the coagulation mechanism. This emphasizes the fact that the patients must undergo a thorough examination before angiography is resorted to.

Out of the 25 patients with glomerulonephritis, 4 had angiographic changes with reduced thickness of the cortex and in the nephrographic phase the cortex was indistinctly demarcated. Of 4 patients with pyelonephritis, 2 had cortical reduction, localized in one, generalized in the other. In this investigation, as in that of Gill and Pudvan,[16] no specific angiographic changes were found capable of differentiating between glomerulonephritis, pyelonephritis and interstitial nephritis. The diagnostic value of angiography in early stages of these lesions is slight. If the clinical and laboratory findings point to one of these diseases, the indication for angiography is small. A biopsy provides more information and should therefore be performed before angiography is planned, despite reported complications.[4,15]

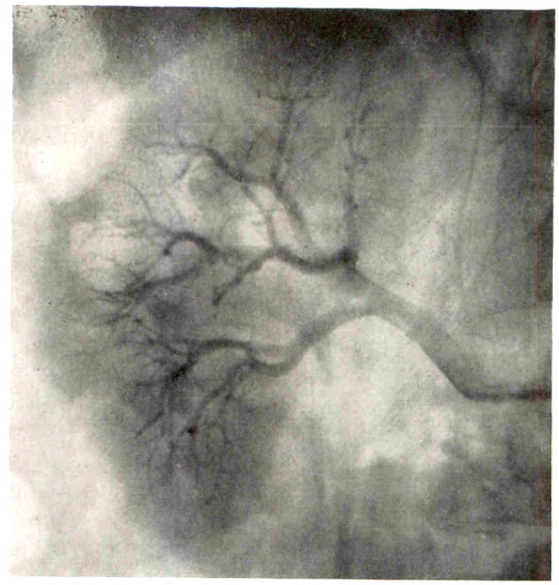


Fig. 5. A.P., 59 year old male with macroscopic hematuria. Three years previously a left-sided nephrectomy was performed because of pyelonephritis. At urography a dilated right kidney pelvis with several clots was seen.

Selective angiogram of the right kidney reveals several small arterial aneurysms with the appearance of periarteritis nodosa; the circulation time was normal.

Nineteen patients had renal vessel changes as probable cause of the bleeding. It is claimed[11,14] that an arteriovenous shunt can only be found by means of angiography. Similar to other reports[1] our material indicates that the absence of findings at angiography does not exclude such changes. Ekelund *et al.*[5] have shown

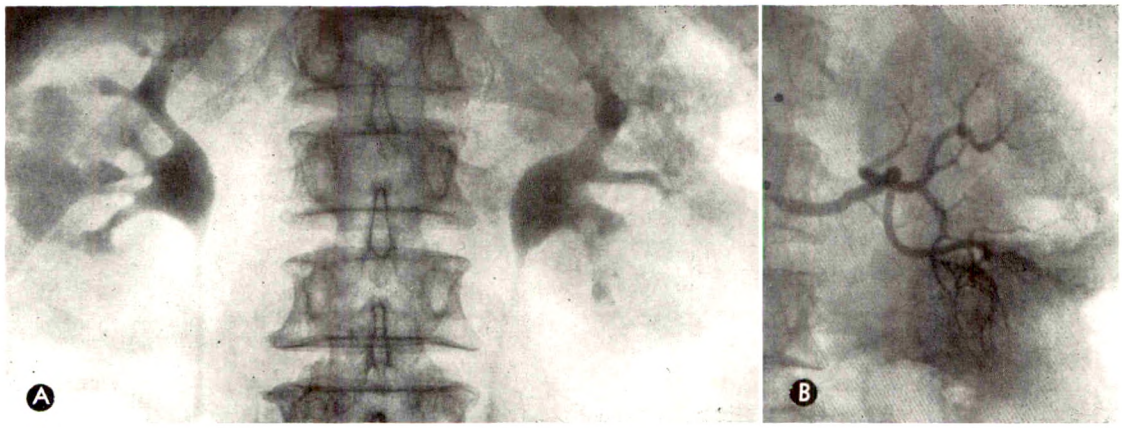


FIG. 6. T.J., 61 year old male, had macroscopic hematuria and left-sided pains for 10 days. At an emergency urography, left-sided obstruction was seen.

(*A*) Two weeks later a control urography was performed, considered to be normal. (*B*) As the hematuria persisted, renal angiography was carried out a few days later. Dislocation of the vessels in the lower pole and the presence of some small tumor vessels indicate a carcinoma. Microscopic diagnosis: carcinoma of hypernephroid type.

that it is possible to detect minute arteriovenous shunts by dye dilution technique, and thus determination of renal blood flow during catheterization should be performed.

From Tables I and II it is apparent that the percentage of renal vascular lesions is unexpectedly high in this investigation. It is probable that hemangioma and arteriovenous shunts are more common than has been expected previously.

In 8 cases, slight dislocation of one calyx or poor filling of the renal pelvis was found at urography so that tumor growth could not be excluded. In all such cases renal angiography must be done.[13] Often the finding is a normal variation,[7] but a renal carcinoma or cyst can also be the cause (Fig. 6*B*).

In cases with small papilloma or carcinoma of the kidney pelvis, angiography is often normal, but urography or pyelography are of decisive importance.

It is obvious from this investigation that the largest group is formed by cases of hematuria, the cause of which cannot be determined. Lee and Davis[12] described 1,000 cases with macroscopic hematuria. Among them only 8.5 per cent had essential hematuria. Angiography was not performed in their material. In half of the patients only cystoscopy was performed and the other half was examined with cystoscopy and pyelography. Urography was performed on only 59 patients. No vascular changes capable of causing hematuria were accounted for in their investigation. From present experience it can be established that their statistics were incomplete. If angiography had been performed the results would have been different.

Flatmark[9] reported 335 patients admitted to hospital because of macroscopic hematuria. Angiographic studies were not performed. In 30 per cent of his cases he found no cause for the hematuria. There was 1 case with a renal hemangioma found at microscopic examination after nephrectomy. In another report Flatmark[8] described a follow-up examination of 95 patients with essential hematuria. The time of observation varied between 2 and 12 years. In that material he found 6 patients with renal carcinoma, 2 with urinary bladder carcinoma, and 1 with carcinoma of the prostate. There were no cases with hemangioma or arteriovenous shunting.

La Cour Andersen and Zachariae[2] carried out a similar follow-up examination of 105 patients. The time of observation differs between 4 and 9 years. Among these patients there were 2 with renal carcinoma and 1 with carcinoma of the urinary

bladder. There were no cases of hemangioma or other vascular changes.

The follow-up of 32 patients in our material with initially essential hematuria gave quite a different result. There was no case with urinary tract carcinoma. In 5 cases biopsy gave the diagnosis of glomerulonephritis or interstitial nephritis 0.5–3 years after angiography. There may be several similar cases among patients with unexplained hematuria, where no biopsy has been performed, as the study of Burkholder *et al.*[3] has shown.

CONCLUSIONS AND SUMMARY

A retrospective study was carried out on 120 patients with hematuria, in 100 of whom angiography was performed after normal urographic findings, and in the remaining 20 after slight, unspecific urographic changes, which did not explain the cause of the bleeding.

The investigation shows that the diagnostic value of angiography in early stages of glomerulonephritis, pyelonephritis and interstitial nephritis is limited. If case-history, other roentgenologic examinations and laboratory findings suggest one of these diagnoses, there is no indication for angiography, and renal biopsy should be performed.

In all cases with a suspected tumor at urography, angiography should be performed.

In this investigation there were 19 patients with renal vascular changes which could be the cause of hematuria. Angiography was the main diagnostic method. The frequency of such changes appears to be greater than has been hitherto observed. Some of the hemangiomas are so minute that they cannot be observed at angiography, but might be detected by flow studies. A follow-up of 32 patients with initially essential hematuria has revealed 2 cases with calculus, 4 with glomerulonephritis and 1 with interstitial nephritis, but no malignancy, as opposed to observations in other reports, where angiography was not performed.

Vascular changes and tumors are frequent enough to make angiography a necessary procedure, but in instances where the urographic studies appear normal, it should be postponed until other clinical methods have failed to yield any diagnostic clue.

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REFERENCES

1. Andersson, L., Boijsen, E., and Henriksson, H. Hämangiom und andere Gefässabnormitäten der Niere als Ursachen der Hämaturie. *Der Urologe*, 1967, *6*, 119–126.
2. la Cour Andersen, J., and Zachariae, F. Essentiell hematuri. *Ugeskrift for Laege*, 1965, *127*, 252–255.
3. Burkholder, G. V., Dotin, L. N., Thomason, B., and Prince, B. D. Unexplained hematuria. *J.A.M.A.*, 1969, *210*, 1729–1733.
4. Ekelund, L. Arteriovenous fistulae secondary to renal biopsy: experimental study in rabbit. *Acta radiol.* (Diag.), 1970, *10*, 218–233.
5. Ekelund, L., Göthlin, J., and Olin, T. Arteriovenous fistulae in rabbit kidney studied by dye-dilution technique and by angiography. *Scandinav. J. Urol. & Nephrol.*, 1972, *6*, 84–90.
6. Efsen, F., and Lorenzen, U. Nephroangiography in periarteritis nodosa. *Acta radiol.* (Diag.), 1968, *7*, 225–230.
7. Felson, B., and Moskowitz, M. Renal pseudotumors: regenerated nodule and other lumps, bumps and dromedary humps. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *107*, 720–729.
8. Flatmark, A. "Essentiell" hematuri. *Nord. Med.*, 1959, *61*, 215–218.
9. Flatmark, A. Hematuri. *Nord. med.*, 1960, *63*, 181–184.
10. Gill, W., and Pudvan, W. Arteriographic diagnosis of renal parenchymal diseases. *Radiology*, 1970, *96*, 81–84.
11. Gunterberg, B. Renal arteriovenous malformation. *Acta radiol.* (Diag.), 1968, *7*, 425–430.
12. Lee, L., and Davis, E. Gross urinary hemorrhage: symptom, not a disease. *J.A.M.A.*, 1953, *153*, 782–784.
13. Olsson, O. In: Angiography. Second edition. Edited by H. Abrams. Little, Brown & Company, Boston, 1971.
14. Rodriguez, S., and Befeler, D. Renal angiomas. *Am. J. Surg.*, 1967, *113*, 574–578.
15. Slotkin, E., and Madsen, P. Complications of renal biopsy: incidence in 5000 reported cases. *J. Urol.*, 1962, *87*, 13–15.

CONTRALATERAL DISPLACEMENT OF THE KIDNEY BY SOLITARY RENAL CYST*

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AN UNCOMMON, yet important roentgenographic finding is *extreme renal displacement*, "a descriptive term . . . reserved for those instances in which the kidney from one side has been displaced to such an extent that it comes to lie on the opposite side of the body."[3] It has been reported in instances of benign retroperitoneal tumors, usually not attached to the kidney,[5] but also with huge enlargement of intraperitoneal organs such as the spleen.[2,4,6] Very seldom has it been reported in the presence of malignant retroperitoneal neoplasms, renal or nonrenal in origin.[3,6] To the best of our knowledge, only 2 cases of a solitary renal cyst displacing the kidney contralaterally have been mentioned in the literature.[6,8]

Recently 2 cases of extreme renal displacement secondary to a benign solitary renal cyst came under our observation and are here reported because of the unusual and interesting roentgenographic findings.

REPORT OF CASES

CASE 1. In July 1971, a 76 year old Caucasian male was admitted to White Memorial Medical Center because of symptoms of prostatism. He complained of urinary frequency, nocturia, slow stream with hesitancy and occasional dysuria. He had no history of gross hematuria or urinary tract infections and denied any weight loss, tiredness, weakness or history of an enlarging abdomen. He had regular bowel movements with no change in the pattern of bowel habit or in the consistency of the feces. The general physical examination was unremarkable except for the abdomen. This was rather globus and protuberant and palpation was very difficult because of obesity of the patient and rigidity of the abdominal wall. A small hernia defect could be felt in the epigastric area, but no distinct masses were palpable. The liver, spleen, and kidneys were also not palpable. The left abdomen was full and firm but resilient with dullness to percussion. The right abdomen was tympanitic to percussion. There was a reduction of Traube's area. The bowel sounds were normal and there was no bruit. On rectal examination there was good sphincter tone with no masses, tenderness or bleeding. The prostate was enlarged (90–100 gm.), regular, quite firm but with no hard nodules.

Laboratory tests were unremarkable except for a slightly elevated blood urea nitrogen of 27 mg. per cent and a sedimentation rate of 54 mm.

The diagnostic roentgenographic investigation commenced with an intravenous infusion pyelography. The scout roentgenogram of the abdomen revealed a large mass occupying the entire left side of the abdomen, but not obliterating the left psoas muscle margin which, instead, was clearly visible. No renal outline could be identified on the left. Following the intravenous infusion of contrast material, a normal right pelvicalyceal system was opacified with no blunting, dilatation or displacement, and with a slender ureter, undisplaced throughout its course. Partly superimposed on the lower portion of the right kidney and its ureteropelvic junction, was a second pelvicalyceal system arranged in a nearly transverse axis. This second pelvicalyceal system showed mild dilatation and a bizarre configuration without dilatation of its ureter. However, much of its ureter was not well outlined (Fig. 1). Tomography (Fig. 2) was employed which seemed to confirm the impression of a normal right kidney and a severely displaced and rotated left kidney across the midline over to the right side by a huge left abdominal mass. Furthermore, the displaced kidney did not ap-

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pear to be fused to the right kidney and the lack of fusion, coupled with the high overlapping location of the second kidney, made congenital crossed ectopia highly unlikely. The second kidney lay anterior to the right kidney. The examination revealed no evidence that the left abdominal mass was a part of the displaced kidney. The roentgenographic density of the mass did not appear to be as great as one would suspect most solid masses of this size to produce. Nevertheless, the marked degree of displacement of the left kidney was consistent with a left retroperitoneal mass lesion, and because of the intact psoas muscle margin and the already described density of the mass, a diagnosis of possible retroperitoneal lipoma or slowly growing liposarcoma was entertained.

Subsequently, the patient was examined by barium enema and by upper gastrointestinal series and small bowel series. These demonstrated a marked anterior displacement of the descending colon, consistent with a left retroperitoneal mass, but also demonstrated displacement of intraperitoneal structures; *i.e.*, the stomach superiorly and the small bowel almost entirely into the right abdomen (Fig. 3). This displacement of intraperitoneal structures was attributed to the huge size of the mass.

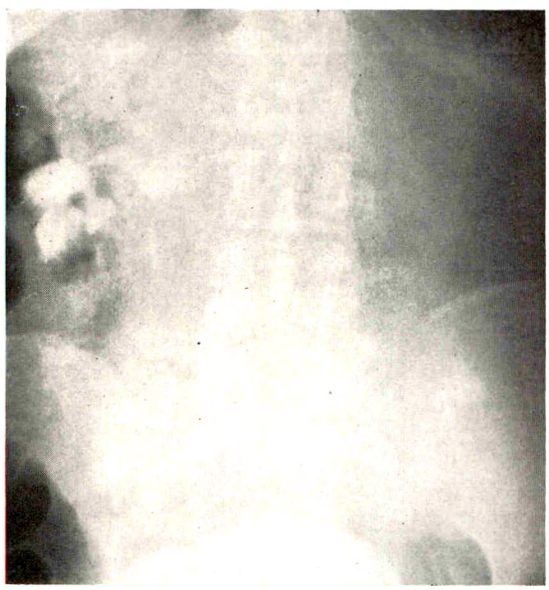


Fig. 1. Case 1. A large retroperitoneal mass on the left does not obliterate the psoas muscle margin, but produces severe displacement and rotation of the left kidney resulting in superimposition of the mildly dilated bizarre collecting system of the left kidney over the lower portion of the right kidney and its ureteropelvic junction.

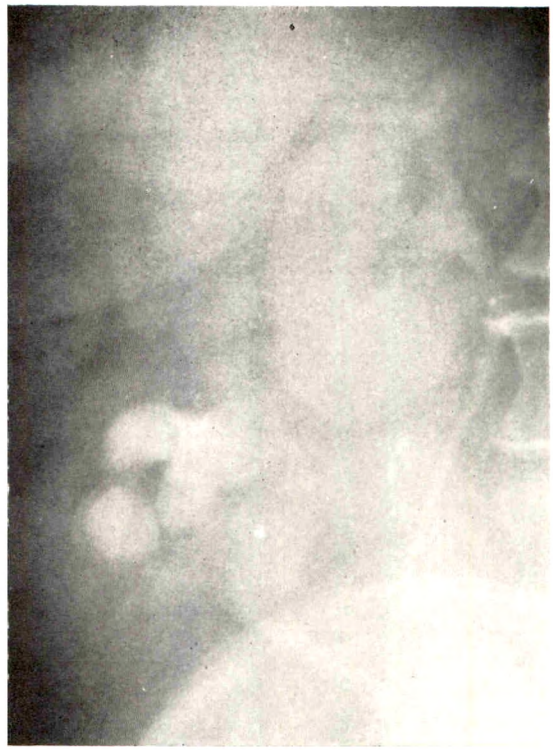


Fig. 2. Case 1. Tomogram (zonogram) in a steep oblique projection demonstrates the displaced left kidney anterior to the right kidney. The kidneys are distinctly separate from one another ruling out crossed fused ectopia.

Aortography was performed by the injection of contrast material through a catheter with the tip in the aorta at the level opposite the upper margin of the first lumbar vertebral segment. A number of significant abnormal findings were observed (Fig. 4, *A* and *B*). The splenic artery was displaced superiorly and, although its usual tortuosity was maintained, its proximal portion extended well to the right of the midline. The spleen was not enlarged, but its lateral margin appeared more medial than usual. A marked displacement of the superior mesenteric artery to the right was noted. The origin of the right renal artery was in a normal location. The left renal artery originated from the left side of the aorta approximately 2.5 cm. cephalad to the right renal artery and demonstrated a marked displacement to the right side, so that a portion of its course was superimposed on that of the superior mesenteric artery. Both kidneys were observed

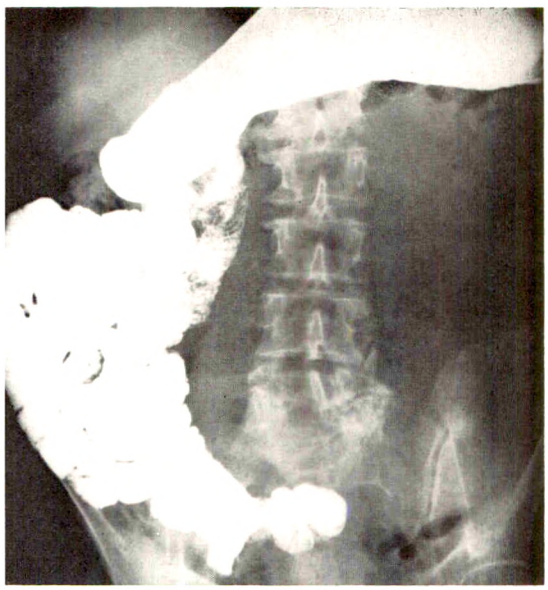


FIG. 3. Case 1. The huge left abdominal mass also displaces intraperitoneal structures. The stomach is displaced upward and the entire small bowel is in the right abdomen.

to be to the right of the midline and appeared separate from one another with the left partially superimposed on the right. They appeared about equal and normal in size. The inferior mesenteric artery was also displaced to the right and the lower abdominal aorta deviated considerably to the right of the midline. The huge left retroperitoneal mass did not opacify or stain and it showed no tumor vascularity. There was, however, some stretching of lumbar arteries on the left. The mass was, therefore, believed to be avascular. Again, the aortography gave no definite clue that the left retroperitoneal mass had its origin from, or was attached to, the displaced left kidney. No selective arteriography was performed.

With a diagnosis of retroperitoneal tumor, the patient underwent an exploratory laparotomy through a midline incision. At surgery a huge renal cyst was found. It filled the entire left abdomen and displaced all of the intraperitoneal and retroperitoneal structures to the right side except for the descending colon which was displaced more anteriorly than medially. The mass was attached to the left kidney which was just below and anterior to the right kidney. The cyst involved the kidney parenchyma very minimally, taking origin from a shallow base of 6 by 8 cm. in diameter (Fig. 5). It contained more than 3,000 ml. of clear, light yellow fluid and had a thin avascular wall.

The cyst wall was resected and the kidney

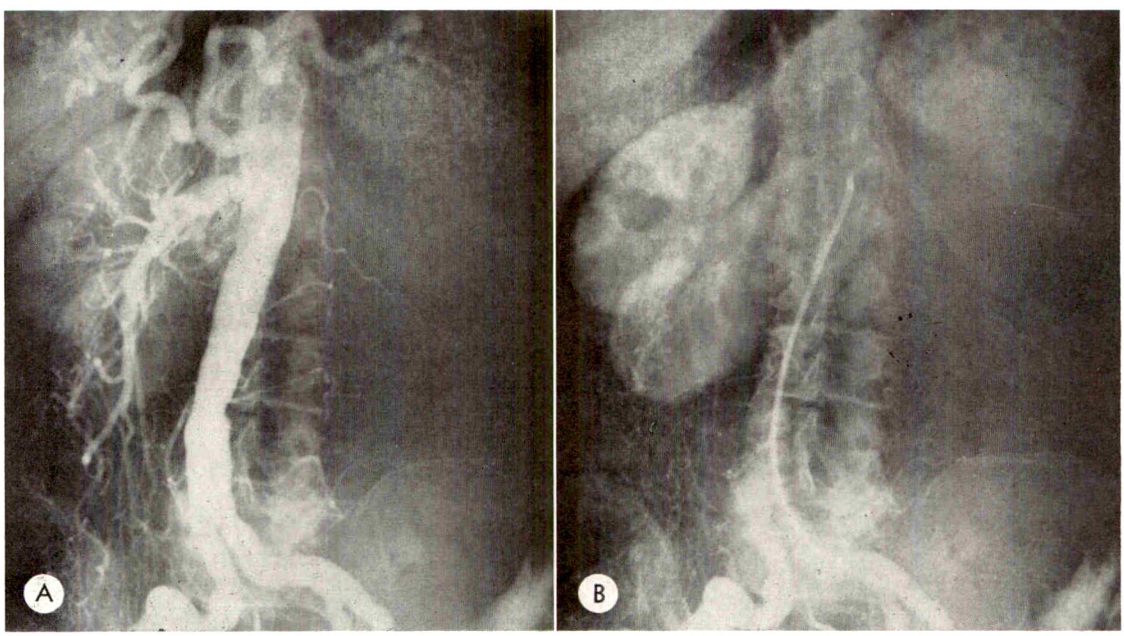


FIG. 4. Case 1. (*A* and *B*) Aortograms demonstrate the contralateral displacement of the left kidney and left renal artery by the large avascular mass on the left. Note the stretching of lumbar arteries on the left and the deviation of the lower abdominal aorta to the right. Also note the marked displacement to the right of the superior mesenteric artery as well as the displacement to the right of the proximal portion of the splenic artery. The contrast material in the left lower quadrant is extravasation and is unrelated to the mass.

was returned to its normal anatomic position, but no nephropexy was performed.

The gross specimen consisted of a yellowish-white, thin and transparent sheet measuring 22 by 18 cm. On a microscopic section, it was made up of fibrous connective tissue not covered by any epithelial lining. This was consistent with the diagnosis of solitary renal cyst. Cytology of the fluid filling the cyst showed the presence of very few polymorphonuclear leukocytes and macrophages. No signs of malignancy were seen.

The recovery of the patient was uneventful. Excretory urography 4 months later was normal.

Case II. This case was brought to our attention shortly after the work-up and surgical exploration of Case I. It was presented by a guest lecturer during a course on renal angiography to the Departments of Urology and Radiology. The roentgenographic findings were remarkably similar. This patient likewise demonstrated a large ill-defined mass in the left abdomen, but in this instance the upper portion of the left psoas muscle margin was obliterated. No renal shadow could be recognized on the left and excretory urography revealed a bizarre pelvicalyceal opacification on the right with the suggestion of 2 ureters on the right, opposite the upper and mid portions of the lumbar spine. One of these ureters appeared to deviate toward the midline over the fourth lumbar vertebral segment and below that point was not visualized (Fig. 6). Aortography (Fig. 7) and selective celiac angiography (Fig. 8) revealed the proximal portion of the splenic artery to the right of the midline and showed cephalad displacement although no tortuosity of the splenic artery. The spleen was not enlarged and occupied a high left subdiaphragmatic position. Aortography revealed a displacement of the superior mesenteric artery to the right as in Case I, but not as great a degree of right deviation of the lower abdominal aorta. The right renal artery originated from the aorta at a level opposite the lumbodorsal junction of the spine and demonstrated an obliquely downward course to the normally positioned right kidney. The left renal artery is best visualized by selective left renal angiography (Fig. 9, *A* and *B*). It arises from the left side of the aorta slightly cephalad to the origin of the right renal artery and ex-

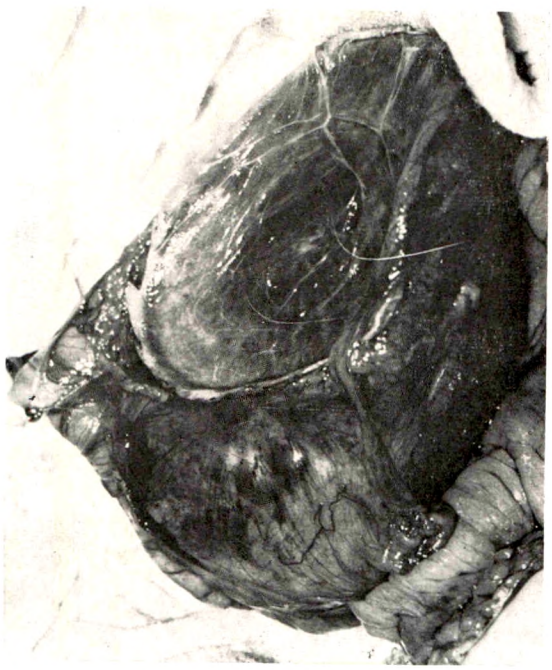


Fig. 5. Case I. Surgical specimen of the left kidney demonstrating the base of the cyst.

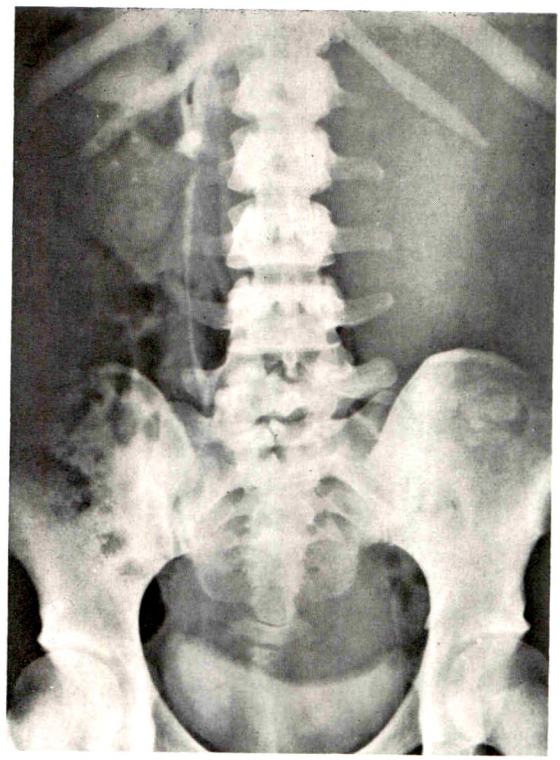


Fig. 6. Case II. Again, a large left abdominal mass is present. In this patient, the mass obliterates the upper portion of the psoas muscle margin. There is no kidney visible on the left and the mass has produced contralateral displacement of the left kidney and upper left ureter.

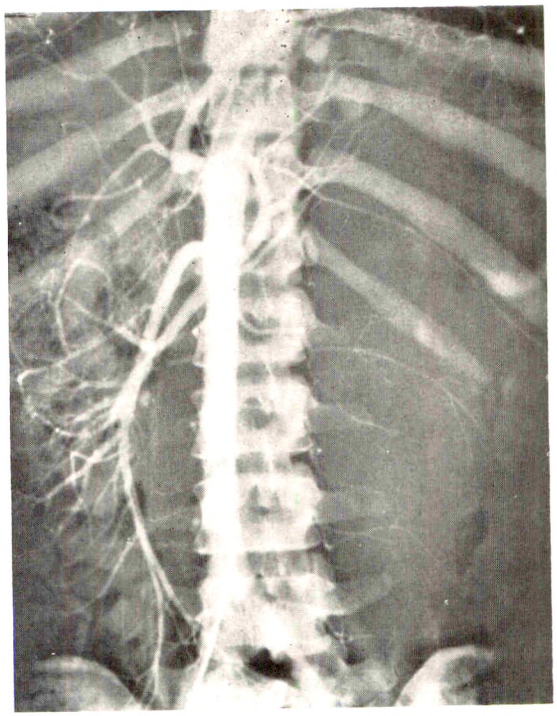


FIG. 7. Case II. The aortogram is remarkably similar to that in Case I (Fig. 4*A*). The retroperitoneal mass is avascular. Stretching and elongation of the lumbar arteries by the mass has occurred. Note the marked displacement of the superior mesenteric artery to the right.

tends in a reverse C curve upward or cephalad overlying the mid portions of the eleventh and twelfth thoracic vertebral segments. The left kidney lies obliquely across the midline in the upper abdomen, the upper pole extending to the left of the midline with the middle and lower pole portions in the right upper quadrant of the abdomen. The kidney, in addition to being displaced, is rotated and the displaced left kidney reveals a hydronephrotic collecting system with the upper portion of its ureter on the right but medial to the right ureter. Again, in this patient, the large retroperitoneal mass was avascular demonstrating no neovasculature or stain. The lumbar arteries on the left showed stretching and extension in a manner similar to that observed in Case I.

At surgical exploration, as in Case I, a huge renal cyst of the left kidney was found to produce the contralateral displacement.

DISCUSSION

Displacement of a kidney from its normal anatomic position is an important roentgenographic sign and may be, in some instances, a significant clue in arriving at a correct diagnosis. An unequivocal degree of such displacement was formerly considered as highly favoring the presence of retroperitoneal tumor (72.5–73 per cent of the cases).[1,7] More recently it has also been described in cases of huge enlargement of intraperitoneal organs such as spleen and liver.[4] Extreme renal displacement can also be seen with intraperitoneal mass lesions,[4] but is more commonly observed with retroperitoneal masses.[2,3,5,6]

It is, however important to differentiate between a minor displacement of the axis or pelvis of a kidney and an extreme renal displacement to the opposite side of the body. Except for a few cases of renal adenocarcinoma[6] and cystic adrenal carcinoma,[2] an extreme renal displacement has been seen mostly in patients with benign, or very low grade malignant, retroperitoneal tumors.[3,5] This is easily understandable. Only a slowly growing tumor, more expansive than infiltrating in nature, can develop over a long period of time and to such an extent as to displace the kidney contralaterally and not impair its shape and function. Very seldom, in fact, has a displacement of such

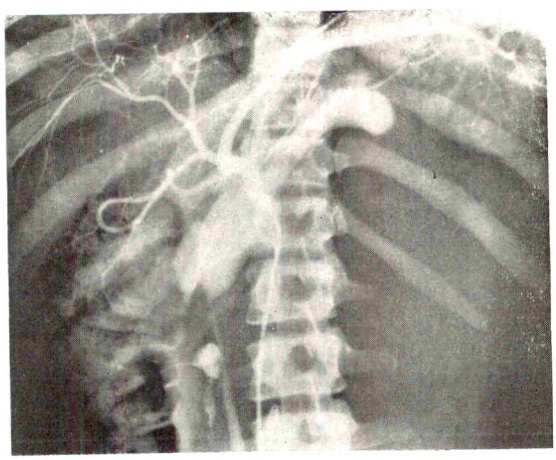


FIG. 8. Case II. Selective celiac angiogram outlines the stretched splenic artery and shows its proximal portion to the right of the midline. The spleen occupies a high subdiaphragmatic position. The dilated collecting system of the displaced and rotated left kidney is well opacified from prior injection and the upper left ureter is noted medial to the right ureter.

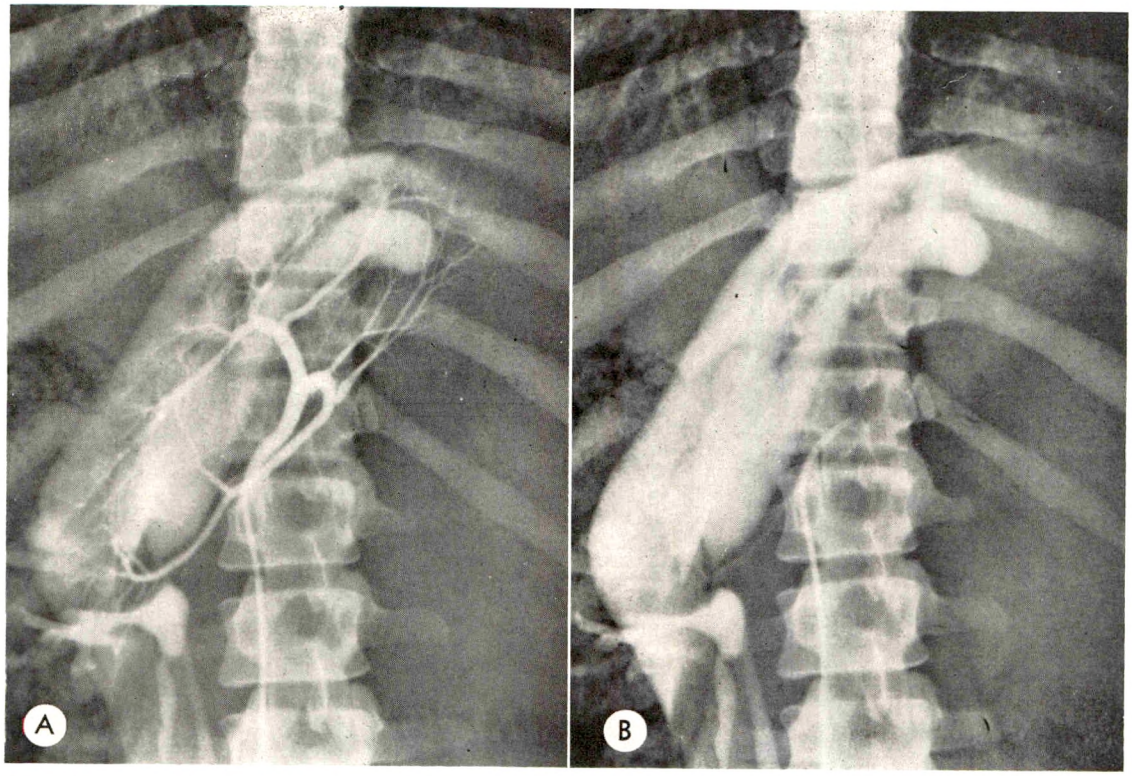


Fig. 9. Case 11. (*A* and *B*) Selective left renal angiograms demonstrate the contralateral displacement of the left kidney. The left renal artery differs in its course from that in Case 1, and reveals an upward reverse C course overlying the eleventh and twelfth thoracic vertebrae.

magnitude been described with a high degree of hydronephrosis and disturbance of the renal function. The slight elevation of the blood urea nitrogen in our Case 1 was due to the obstructive prostate.

The differentiation between minimal or moderate renal displacement and contralateral displacement can therefore be quite helpful in making the diagnosis and in estimating prognosis. In fact, in spite of the size of the mass and the extreme displacement of the retroperitoneal and perhaps intraperitoneal structures, the patient, with such a contralateral displacement of the kidney, may still be amenable to operation because of the benign nature of the tumor.

To the best of our knowledge, no case of a solitary renal cyst displacing the kidney contralaterally has been described in detail. Wesson[8] reported a case of an infected solitary cyst displacing the left kidney to the right. Ney and Friedenberg,[6] in their atlas, presented a roentgenographic illustration of a huge cyst of the left kidney displacing the organ to the right side of the spine. We believe that it is very uncommon for a renal cyst to contralaterally displace the kidney. Only a cyst of huge proportions arising at the periphery of a kidney and not interfering significantly with kidney parenchyma is likely to displace the kidney itself to such an extent that it will be displaced to the opposite side of the body.

SUMMARY

Two cases of extreme (contralateral) renal displacement produced by benign solitary renal cyst have recently been observed. Our search of the radiologic and urologic literature has failed to uncover previous detailed description of this occurrence. Although an apparently uncommon occurrence, such a possibility must now be considered in the differential diagnosis of

masses producing extreme renal displacement.

Furthermore, it is re-emphasized that the occurrence of severe renal displacement by a retroperitoneal mass is likely to be produced by a lesion of benign or low grade malignant character, rather than by a highly malignant lesion. It is, therefore, likely that such a lesion is amenable to surgical excision.

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REFERENCES

1. DEWEERD, J. H. Lipomatous retroperitoneal tumors: urographic findings. *J. Urol.*, 1954, *71*, 421–426.
2. EMMETT, J. L., and WILFEN, D. M. Clinical Urography. W. B. Saunders Company, Philadelphia, 1971, pp. 1199, 1210, 1246.
3. ENGEL, W. J. Significance of renal displacement. *J. Urol.*, 1956, *76*, 478–487.
4. FORDE, W. J., OSTROLENK, D. G., and FINBY, N. Renal displacement associated with enlargement of spleen. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1960, *84*, 889–897
5. GREENE, L. F. Extreme renal displacement due to retroperitoneal tumors. *J. Urol.*, 1948, *59*, 174–178.
6. NEY, C., and FRIEDENBERG, R. M. Radiographic Atlas of the Genitourinary System. J. B. Lippincott Company, Philadelphia, 1966, pp. 76, 80, 250.
7. SCHULTE, T. L., and EMMETT, J. L. Urography in differential diagnosis of retroperitoneal tumors. *J. Urol.*, 1939, *42*, 215–219.
8. WESSON, M. B. Urologic Roentgenology. Lea & Febiger, Philadelphia, 1946, pp. 186.

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MEDULLARY SPONGE KIDNEY ASSOCIATED WITH CONGENITAL HEMIHYPERTROPHY (ASYMMETRY)*

A CASE REPORT AND SURVEY OF THE LITERATURE

By RONALD L. EISENBERG, M.D., *and* RICHARD C. PFISTER, M.D.
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CONGENITAL hemihypertrophy,[15] the infrequent overgrowth of one side of the body beyond the normal range of asymmetry, and medullary sponge kidney,[5] the relatively uncommon ectasia of the distal collecting tubules, have recently been noted to coexist in a frequency that appears to exceed the statistical occurrence of chance events.

In this communication, a sixth case is presented and the 5 cases reported since 1965 are summarized.[8,9,11,13]

REPORT OF A CASE

F.B. was born on January 9, 1940, after a normal spontaneous delivery. At the age of 5 weeks his mother noted that his right leg was both wider and longer than the left. This difference was progressive (Table 1). He was followed in the Growth Clinic (Fig. 1) of the Children's Hospital Medical Center of Boston where teleroentgenograms were obtained every 6 months (Fig. 2). At the age of 13 years, a right tibial-fibular proximal epiphyseal arrest was performed (Table 1). He had no evidence of genitourinary disease. No congenital anomalies or renal disease exist in the family.

In October, 1970, he was admitted to the Massachusetts General Hospital after a sudden onset of left flank pain. An intravenous urogram was obtained, and on the preliminary roent-

TABLE I

ROENTGENOGRAPHIC MEASUREMENTS OF THE LOWER EXTREMITIES (cm.)

Date of Examination		Right	Left	Length Difference	Total Length Difference
January 10, 1946	Femur	28.8	27.6	1.2	3.1
	Tibia	24.6	22.7	1.9	
October 21, 1953		43.0	41.2	1.8	4.4
		36.8	34.2	2.6	
February 9, 1963*		48.7	46.8	1.9	2.1
		38.6	38.4	0.2	

* Ten years postoperative.

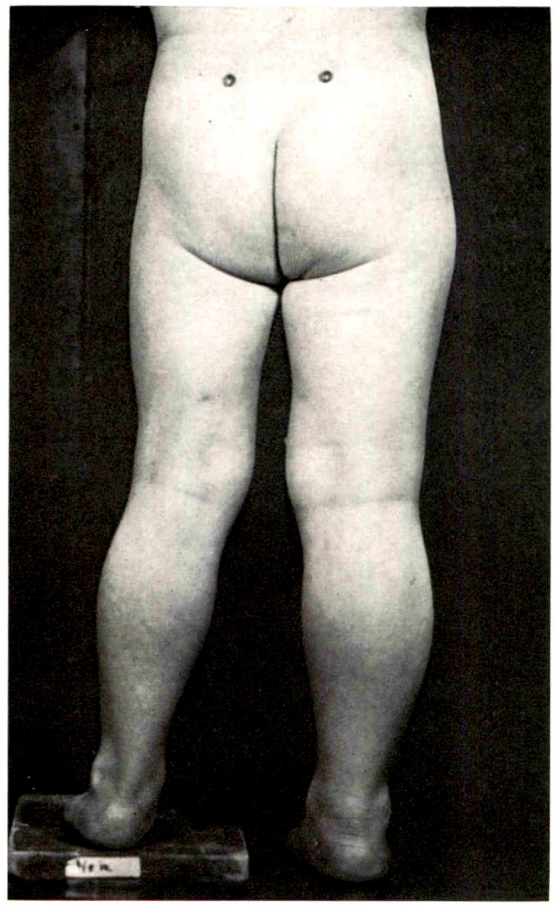


FIG. 1. Photograph of the patient at $3\frac{1}{2}$ years of age. The hypertrophied right leg is enlarged in girth and length. The normal left leg rests on a wooden block of appropriate height to produce a level pelvis, indicated by marking spots on skin above the buttocks.

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Fig. 2. Teleroentgenogram (anteroposterior) at age 10 years. The right femur and tibia-fibula are elongated. The thickness of the soft tissues is also greater on this hypertrophied side.

genogram of the abdomen, diffuse left nephrocalcinosis with a probable left ureteral calculus adjacent to the third lumbar vertebral body was seen. No calculi were observed in the right kidney. The left kidney, which was enlarged, measured 15×10 cm., whereas the right was normal in size (12×6.5 cm.). The right collecting system was normal. However, on the left there was delayed function and a prolonged nephrogram; 8 hours after injection, a 6 mm. stone, several centimeters distal to the ureteropelvic junction, was demonstrated to be obstructing the left ureter. Incidentally noted was gallbladder opacification by the urographic contrast medium[17] (Fig. 3). At a subsequent postoperative date, infusion nephrotomography[14] revealed that punctate calcifications on the left were present in approximately two-thirds of the papillae (Fig. 4*A*), but linear striations and rounded clusters of contrast material were seen in all of the renal pyramids (Fig. 4*B*). A 3 cm. cyst involving the suprahilar lip of the left kidney was noted (Fig. 4*C*). In addition, there was faint papillary blush in the pyramids of the right kidney.

The serum calcium, phosphorus, sodium, potassium, chloride, and bicarbonate values were normal. The blood urea nitrogen was 20, and the serum creatinine 1.7 mg. per 100 ml. Urinalysis revealed a specific gravity of 1.020, pH 5.0, negative albumin, with 20 white blood cells and 200 red blood cells per high power

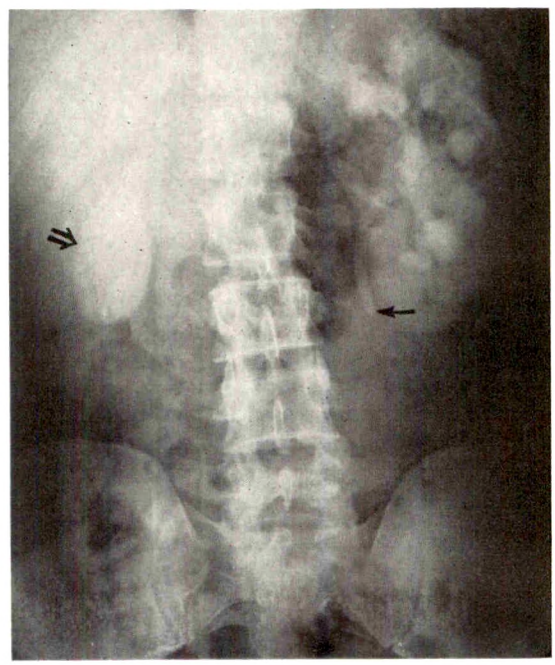


Fig. 3. Delayed (8 hour) abdominal roentgenogram following intravenous urography. Obstructing calculus (solid arrow) in the mid left ureter. Hepatic extraction of the urographic contrast medium has resulted in gallbladder opacification (double-tailed arrow) despite a normally functioning right kidney.

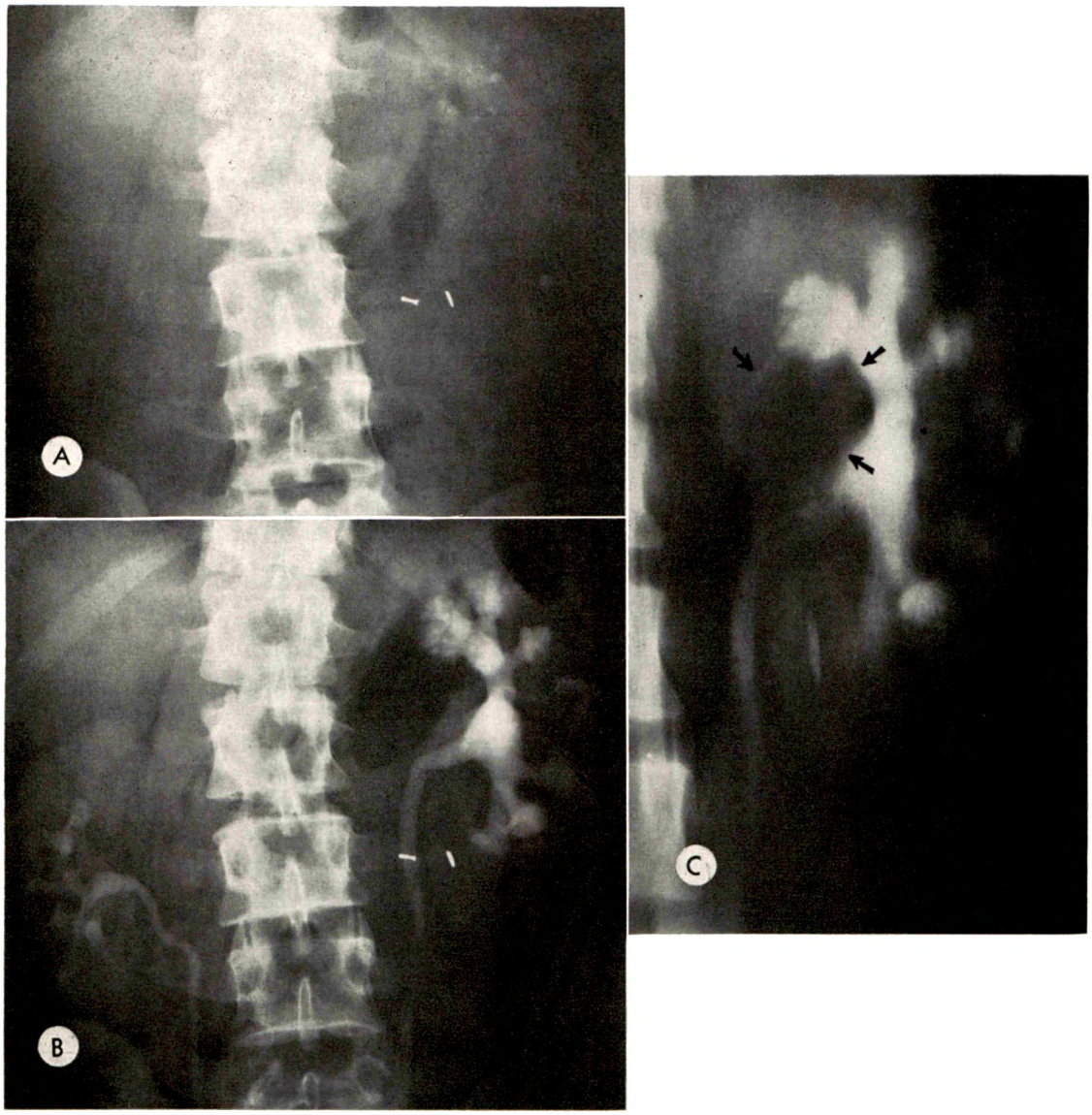


Fig. 4. (*A*) Seven days following ureterolithotomy (surgical clips) and renal biopsy. Preliminary roentgenogram of the kidneys shows unilateral nephrocalcinosis in an enlarged left kidney. (*B*) Excretory urogram. Normal right kidney with typical features of medullary sponge disease in the left kidney. (*C*) Nephrotomogram demonstrates a 3 cm. simple cyst (arrows), ectatic tubules within the renal pyramids, and blood clot in the middle calyx (the latter subsequently resolved).

field. A urine culture failed to grow any pathogenic organisms.

When the stone failed to pass after conservative treatment, a left ureterolithotomy was performed, and an open biopsy of the left kidney was obtained which contained cortex and outer medulla, both of which were histologically normal. His hemihypertrophy remained apparent on close inspection, and several measurements were obtained (Table II) prior to discharge from the hospital.

DISCUSSION

Congenital hemihypertrophy, of which less than 200 cases have been reported,[1] has been associated with other malformations in approximately one-half of the cases.[18] Several of the malformations were urogenital tract anomalies such as cryptorchidism, hypospadias, renal hypertrophy (unilateral or bilateral)[16] and clitoral or penile enlargement. Several neoplasms

TABLE II

EXTERNAL MEASUREMENTS (cm.) IN 1970

	Right	Left	Difference
Upper Extremity			
Length	76.0	73.4	2.6
Circumference			
(a) Biceps	27.3	25.4	1.9
(b) Distal forearm	16.2	15.6	0.8
Chest			
Circumference			
(at nipple line)	45.8	40.6	5.2
Lower Extremity			
Length*	91.5	89.0	2.5
Circumference			
(a) Lower thigh	35.6	33.0	2.6
(b) Calf	30.5	28.0	2.5

* Seventeen years postoperative.

(Wilms' tumor,[2] adrenal cortical tumor,[3,13] and hepatoblastoma[3]) were discovered. Our case is the first reported incidence of a contralateral medullary sponge kidney associated with congenital hemihypertrophy (Table III).

The etiology of hemihypertrophy is unknown, and numerous theories have been put forward to explain the phenomenon. These include:[4,11,13] (1) abnormalities of the ovum (fusion of 2 separately fertilized meiotic products to form a chimera); (2) abnormality of twinning–incomplete form of uniovular twinning; (3) chromosomal abnormalities (sporadic mutant); (4) endocrine abnormalities; (5) lesion of the nervous system resulting in an altered neurotrophic action to the hypertrophic area; and (6) vascular or lymphatic abnormalities (anatomic and/or functional) of the hypertrophic area.

Medullary sponge kidney has also been postulated as a developmental anomaly. Norris and Herman[10] described the occurrence of cystic dilatation of the tubules during normal renal embryogenesis and suggested that the persistence of these cystic dilatations into later life is responsible for the development of polycystic kidneys. (This was published before the first pathologic descriptions of medullary sponge kidney by Cocchi and Ricci[1] in 1949, and 2 years after the first roentgenologic description by Lenarduzzi[6] in 1939.) Osathanondh and Potter,[12] on the basis of microdissection studies, concluded that medullary sponge kidney is most likely due to a hyperplasia of the collecting tubules that lie in the medullary pyramids. These tubules arise from the division of the ureteral duct which has grown out from the lower mesonephric duct as early as the fifth week of embryonic development. Thus, both hypotheses state that medul-

TABLE III

MEDULLARY SPONGE KIDNEY AND CONGENITAL HEMIHYPERTROPHY

Author	Sex	Age	Hemihypertrophy	Medullary Sponge Kidney Bilateral	Unilateral
Morris *et al.*[9]	M	31	Right		Ipsilateral
Levy *et al.*[8]	M	38	Left	+	
	F	34	Right	+	
Oehninger *et al.*[11]	F	22	Right	+	
Parker and Skalko[13]	M	29	Left	+	
Eisenberg and Pfister	M	30	Right		Contralateral

lary sponge kidney is an abnormality of the developing embryo.

Four reported cases of medullary sponge kidney have been associated with Ehler's-Danlos syndrome[7,9] (an inherited excessive extensibility of collagenous structures manifested clinically by fragility and hyperelasticity of the skin and loose-jointedness), 1 case with congenital hypertrophic pyloric stenosis,[9] and now 6 cases with hemihypertrophy.

SUMMARY

The concomitant occurrence of medullary sponge kidney and hemihypertrophy (asymmetry) appears to be a rare event. A case is reported and added to 5 previous cases found in the literature.

The precise etiologies of these disorders are unknown; however, both are suspected of being congenital in origin, arising during embryonic development, and representing mesodermal dysgenesis.

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REFERENCES

1. Cocchi, R., and Ricci, V. Sur une rare maladie cystique multiple des pyramides rénales, le "rein en éponge." *J. d'urol., Paris*, 1949, *55*, 497–519.
2. Fraumeni, J. F., Feiser, C. F., and Manning, M. D. Wilms' tumor and congenital hemihypertrophy: report of five new cases and review of literature. *Pediatrics*, 1967, *40*, 886–899.
3. Fraumeni, J. F., and Miller, R. W. Adrenocortical neoplasm with hemihypertrophy, brain tumors, and other disorders. *J. Pediat.*, 1967, *70*, 129–138.
4. Gerloczy, E., Schuler, D., Letenyei, K., Kiss, S., and Hervei, S. Hemihypertrophy: ten year followup and chromosomal study. *Acta paediat., Acad. Sc. Hung.*, 1965, 6 (Fasc. 4), 423–427.
5. Lalli, A. Medullary sponge kidney disease. *Radiology*, 1969, *92*, 92–96.
6. Lenarduzzi, G. Reporto pielografico poco commune dilatazione delle vie urinarie intrarenali. *Radiol. med.*, 1939, *26*, 346.
7. Levine, A. S., and Michael, A. F. Ehler's-Danlos syndrome with renal tubular acidosis and medullary sponge kidneys. *J. Pediat.*, 1967, *107*, 107–113.
8. Levy, M., MacKinnon, K. J., and Dossetor, J. B. Hemihypertrophy and medullary sponge kidney. *Canad. M. A. J.*, 1967, *96*, 1322–1326.
9. Morris, R. C., Yamauchi, H., Palubinskas, A. J., and Howenstein, J. Medullary sponge kidney. *Am. J. Med.*, 1965, *38*, 883–892.
10. Norris, R. F., and Herman, L. Pathogenesis of polycystic kidneys: reconstruction of cystic elements in four cases. *J. Urol.*, 1941, *46*, 147–176.
11. Oehninger, C., Moron, R., Russi, D., Acosta-Ferreira, W., and Artucio, H. Riñón en esponja asociado a hemihipertrofia. *Arch. Func. Roux-Ocefa.*, 1967, *1*, 305–313.
12. Osathanondh, V., and Potter, E. L. Pathogenesis of polycystic kidneys. *A.M.A. Arch. Path.*, 1964, *77*, 510–512.
13. Parker, D. A., and Skalko, R. D. Congenital asymmetry: report of 10 cases with associated developmental abnormalities. *Pediatrics*, 1969, *44*, 584–589.
14. Pfister, R. C., and Shea, R. E. Nephrotomography: performance and interpretation. *Radiol. Clin. North America*, 1971, *9*, 41–62.
15. Ringrose, R. E., Jabbour, J. T., and Keele, D. K. Hemihypertrophy. *Pediatrics*, 1965, *36*, 434–448.
16. Roggensack, G., and McAlister, W. H. Bilateral nephromegaly in child with hemihypertrophy. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *109*, 546–549.
17. Shea, R. E., and Pfister, R. C. Opacification of gallbladder by urographic contrast media. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1969, *107*, 763–768.
18. Ward, J., and Lerner, H. H. Review of subject of congenital hemihypertrophy and complete case report. *J. Pediat.*, 1947, *31*, 403–414.

UNILATERAL TOTAL RENAL DYSPLASIA IN CHILDREN*

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RENAL dysplasia has increasingly become recognized as an important clinical entity. Unilateral renal dysplasia is the most common abdominal mass in the newborn infant[19] and is the most common cystic disorder of the kidney in children.[16]

Dysplasia of the kidney is a form of renal cystic disease characterized by disturbed differentiation of nephrogenic tissue with persistence of structures inappropriate to the gestational age of the patient.[17] The process may involve one or both kidneys; it may involve the entire kidney (total), only a part of it (segmental), or there may be scattered foci of dysplasia throughout the kidney (focal). The lesion has been referred to in the literature as unilateral polycystic disease,[5,18,31] aplasia with cystic degeneration,[1,28] cystic hypoplasia,[14] metanephric cystic remnants,[29] congenital cystic hydrocalycosis,[10] unilateral multilocular cysts,[8] multiple simple cysts,[6] congenital cystic disease with hydronephrosis,[21] metanephric cystic tumor,[19] cystic hamartoma,[15] and unilateral multicystic kidney.[7,26] Renal dysplasia has been differentiated from familial forms of polycystic disease of the kidney[25] and has been designated as a Type II polycystic kidney.[22]

The prognosis differs in the various types of cystic disease of the kidney, and the individual clinical course varies among the types of renal dysplasia.[25] In studies on renal dysplasia based on autopsy material, the emphasis has been placed on pathologic aspects.[3,4,23,24] The prognosis in rare cases of bilateral total renal dysplasia is understandably poor. However, because the approach to unilateral renal dysplasia has been mostly from autopsy material and few long-term, clinical studies on this entity have been reported, the natural history has not been sufficiently documented. While unilateral renal dysplasia is thought to have a good prognosis, some studies demonstrate a less favorable outlook because of abnormalities in the contralateral kidney or coexistent lethal malformations in other organs.[13,23,24] Our study was undertaken to determine the outcome of a group of patients with unilateral total renal dysplasia, and the status of the opposite kidney.

MATERIAL AND METHOD

Microscopic and, where available, gross material of renal dysplasia were reviewed in the Division of Surgical Pathology, Barnes Hospital, along with the clinical records of 27 children from St. Louis Children's Hospital and Barnes Hospital. We excluded cases of bilateral renal dysplasia and cases of renal dysplasia found incidentally at autopsy in patients dying with significant congenital malformations of other organ systems. Three adults in whom unilateral renal dysplasia was found at abdominal surgery for other reasons were excluded. Follow-up information was sought by reviewing the clinic charts, by sending a questionnaire to the patient's private physicians, and by direct patient contact. Eight patients with suboptimal early roentgenograms were returned for intravenous urography.

RESULTS

PATHOLOGIC FEATURES

A totally dysplastic kidney is usually described as a large, nonuniform mass of

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variable sized cysts resembling a "bunch of grapes"[16,24] (Fig. 1). However, the gross appearance is variable in both the size and number of cysts.

The status of the ureter is important in understanding the pathogenesis of dysplasia. In 6 of 27 surgical cases, the ureter was not mentioned in either the surgical note or the pathologic gross description. In the remaining patients the ureter was described as abnormal, usually atretic. No patient had a normal ureter noted at surgery or on the pathology report. The conclusion is justified, that in most cases the ureter from the dysplastic kidney was either absent or malformed. The weight of the dysplastic kidney varied from 25 to 1,100 grams. The size of the cysts varied from 1 mm. to 7 cm. There was a predominance of left-sided involvement (17 of 27).

Microscopic features of dysplasia are:

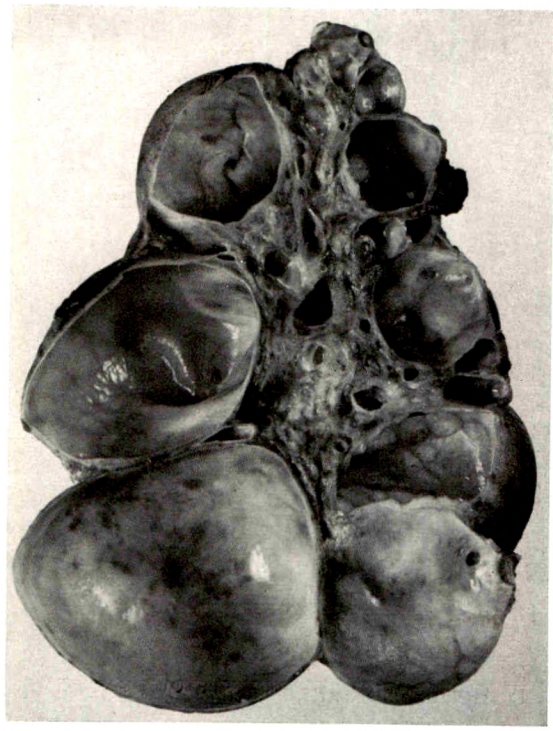


Fig. 1. Gross photograph of a totally dysplastic kidney. The mass consists of cysts of varying sizes held together by flimsy gray-white connective tissue.

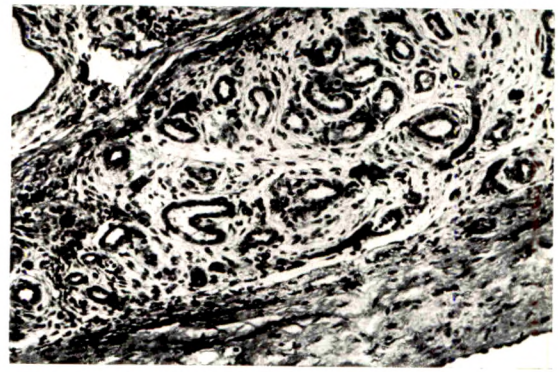


Fig. 2. Disorderly aggregates of small tubules lined with low hyperchromatic epithelium in a dysplastic kidney (hematoxylin and eosin ×320).

(1) focally dilated ducts lined by tall cuboidal or columnar epithelium surrounded by a concentric mantle of undifferentiated mesenchyme in which smooth muscle can often be demonstrated; (2) smaller ductules that ramify in metanephrogenic tissue; and (3) abundant, mesenchymal derivatives[16] often including cartilage (Fig. 2; and 3, *A* and *B*). All cases exhibited these characteristics. Cartilage was found in 10 of 27 cases. In 3 cases normal appearing glomeruli were noted, whereas in others an abortive formation of glomeruli was found.

Clinical Features

Twenty-four of 27 patients presented with an abdominal mass palpated by the patient or physician. The remaining presented with respiratory distress, nausea and vomiting, fever, polyuria, and trauma to the abdomen. There was a preponderance of males (17 of 27) which is probably not significant, as previous studies showed no sex difference in total dysplasia.[4,24] Seventeen of the infants presented at less than 1 month of age. Fifteen of these patients were totally asymptomatic despite their abdominal mass.

Two patients had coincident cardiac defects; 1 pulmonary stenosis and 1 a ventricular septal defect with right middle lobe emphysema. The latter subsided spontaneously over 5 years. Three patients had

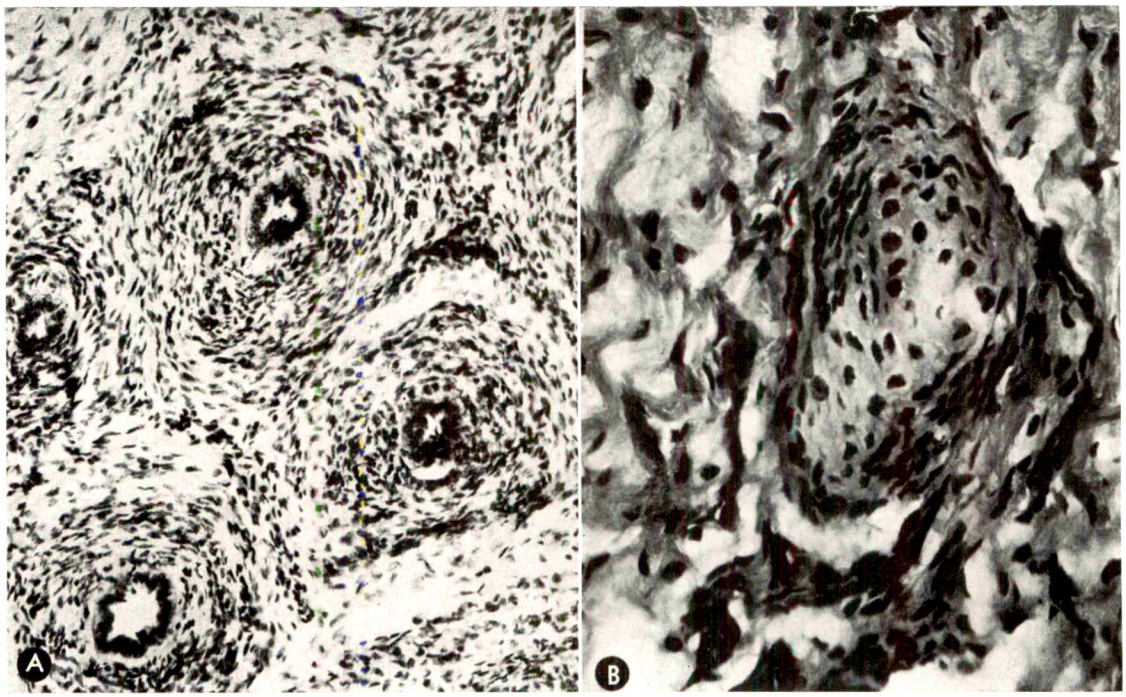


FIG. 3. (*A*) Dilated ductular structures surrounded by immature mesenchymatous stroma in a dysplastic kidney (hematoxylin and eosin ×72). (*B*) Spicule of cartilage in the stroma of a dysplastic kidney (hematoxylin and eosin × 600).

inguinal hernias which were subsequently repaired without difficulty. One child had a ureteral stricture in the contralateral nondysplastic kidney. One premature infant had respiratory distress syndrome but did well after the first few days of life. Another was premature but had no difficulty. They both weighed more than 4 pounds.

Differential diagnosis of the abdominal mass included hydronephrosis, Wilms' tumor, neuroblastoma and renal vein occlusion. One child received a preoperative course of radiation therapy because of an erroneous diagnosis of Wilms' tumor. All patients on discharge from the hospital were asymptomatic.

LABORATORY RESULTS

Routine laboratory values including hemoglobins, white blood cell counts, serum electrolytes and blood urea nitrogens were normal. Urinalysis on admission in the vast majority of patients was normal. Only occasionally were protein, white blood cells or red blood cells present in the urinary sediment. Urinary cultures in 3 patients were negative. Creatinine and inulin clearances, where done, were normal.

ROENTGEN FINDINGS

A presumptive diagnosis of renal dysplasia can be made by a correlation of roentgen findings in the proper clinical setting. Roentgenograms of 15 patients were available for review. The records of the remaining patients were studied. The plain roentgenograms of the abdomen usually showed a mass which in two instances was lobulated. The mass can occupy much of the entire abdomen, including crossing the midline. The sparsity of perirenal fat in infancy and the small size of some masses can result in normal plain roentgenograms. Although calcification has been reported,[12] none was present in our patients. Inferior venacavography showed a patent, nondisplaced vessel in 3, a dis-

placed vena cava in 2, and complete occlusion in another 2 patients. In the latter 2 patients the vena cava became normal when the large dysplastic kidney compressing it was removed. In all patients the affected kidney did not excrete contrast media by intravenous urography. In patients later in the series, high dose urography with total body opacification showed cyst walls to opacify with the cysts remaining radiolucent (Fig. 4; and 5). This was an excellent reliable sign of renal dysplasia. Cystography was performed 7 times in an attempt to reflux the nonexcreting kidney. All cystograms were normal. When performed, cystoscopy with retrograde pyelography showed a blind ureter. Three instances of perforation of the ureter by a retrograde catheter occurred (Fig. 6). In 1 instance percutaneous injection of contrast medium into a cyst was performed and outlined only 1 cyst, although cyst communication has been seen. Angiography showed small renal arteries with disorganized small vessels and poor perfusion in 2 patients.

The opposite kidney was normal in all instances except in 1 patient, where there

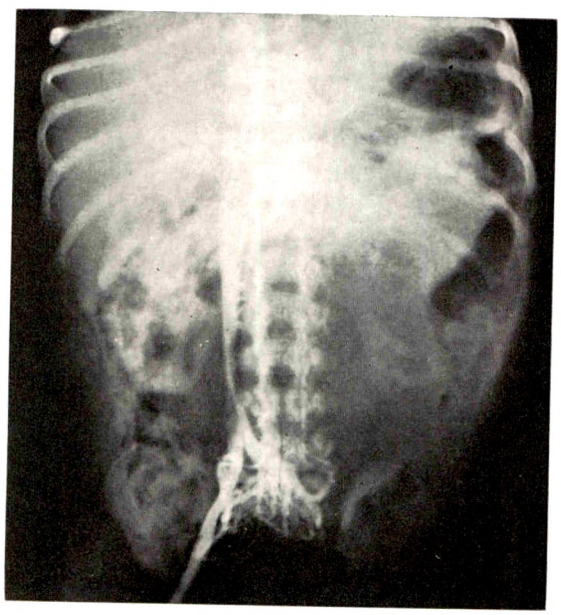


Fig. 4. Intravenous urogram showing radiolucent left multicystic dysplastic kidney on total body opacification crossing the midline with compression of the inferior vena cava.

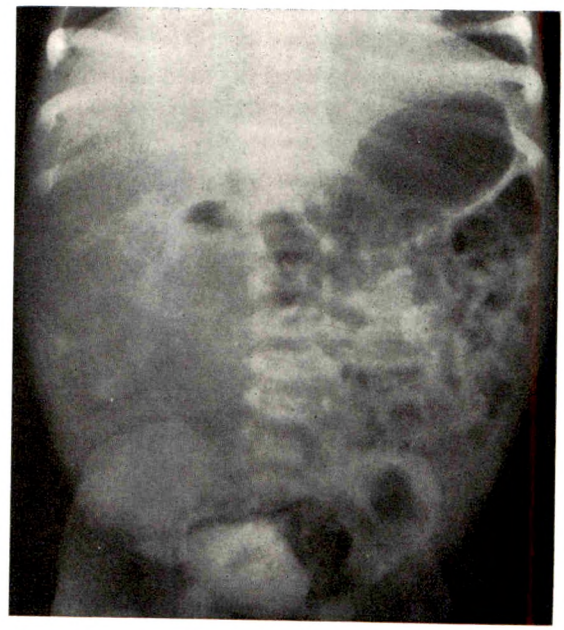


Fig. 5. Right multicystic dysplastic kidney on total body opacification demonstrating the radiopaque cyst walls. The left kidney at this study and 6 years later was normal.

was a moderately severe stricture in the middle third of the ureter (Fig. 7).

On review of the available roentgenograms and using the standards as established by one of the authors,[11] all patients had compensatory hypertrophy of the opposite kidney, where that kidney could be satisfactorily measured. One newborn patient had a kidney increased in size 6 standard deviations above the mean. That patient has continued to do well. Compensatory hypertrophy can occur in the newborn period.[17] Follow-up urography was obtained on 8 patients where good anatomic detail was not available on the initial roentgenograms and showed only compensatory hypertrophy. The 2 patients with recurring infection continued to have an anatomically normal remaining kidney by intravenous urography and cystography.

FOLLOW-UP STUDIES

Information was obtained regarding 18 of 27 patients. The remaining 9 patients were alive and well at the time of their last clinic or private physician appointment.

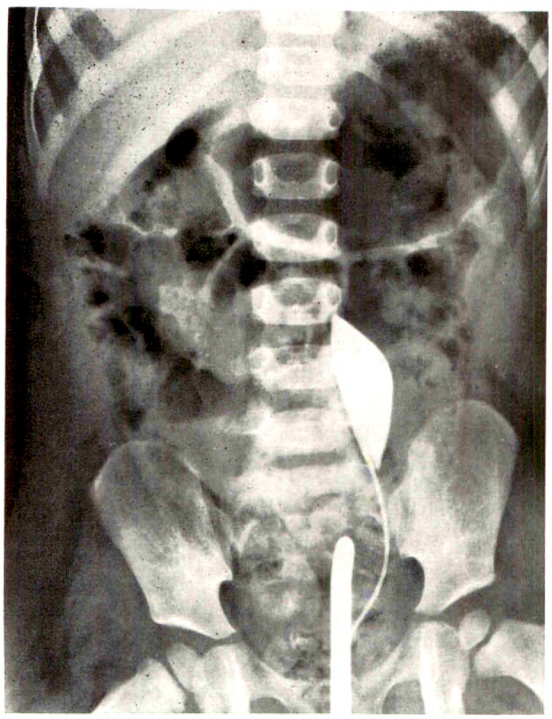


FIG. 6. Left retrograde pyelogram with perforation of the ureter and retroperitoneal extravasation of contrast medium.

Fourteen of the 18 patients are completely well with normal growth and development. The 2 patients with congenital heart disease are included in this group. One has undergone successful surgical correction of her pulmonary stenosis. Two children have had single episodes of urinary tract infection unassociated with roentgen anatomic abnormalities in the remaining kidney and controlled with antibiotics. One child has had a midureteral stricture operated on and has done well with minimal residual caliectasis. None of the children has hypertension or azotemia. None is known to have died. The patients have been followed from between 2–28 years. The mean duration of follow-up is 9.1 years. The conclusion is justified that renal function and structure are usually normal in the remaining kidney of surgically treated children with unilateral renal dysplasia.

DISCUSSION

Confusion on renal cystic diseases has led to disagreement on their clinicopathologic features and natural history. Renal dysplasia has a characteristic microscopic picture which allows its differentiation from other renal cystic diseases. The obvious prognosis when both kidneys are involved is early death.

Several series of unilateral total dysplasia have been reported. Those series which support a good prognosis,[3,9,25,30,32] usually have few patients and the period of follow-up is short. These primarily include patients who present with abdominal masses. The larger series,[4,23,24] are heavily weighed toward autopsy material and have a high incidence of abnormality of the opposite



FIG. 7. Intravenous urogram showing a stricture of the middle third of the right kidney.

kidney. One series[24] reported 20 cases of "unilateral multicystic disease" and found the contralateral kidney abnormal in 16 of these. In this series only 7 of 20 cases were referred for evaluation of an abdominal mass, while 8 of these 20 patients were admitted primarily for treatment of other anomalies. Greene *et al.*[13] found the contralateral kidney abnormal in 12 of 30 patients.

Our report of 27 children with unilateral total renal dysplasia is based on a series of surgical specimens. Our incidence of 6 patients with associated congenital defects parallels the findings in another series.[4]

The pathologic aspects of our cases were similar to those seen in other series. The ipsilateral ureter was abnormal in all cases in which it was examined. This is in keeping with current concepts on the etiology of renal dysplasia. Emphasis is placed on the role of the ureteric bud in inducing differentiation of the metanephrogenic ridge[22] and that this mechanism is somehow disturbed when the ureter is abnormal. The presence of total dysplasia associated with the absence of the ureter suggests that the lesion may result in part from an inherent tendency of nephrogenic tissue to differentiate atypically in the absence of normal stimuli. Most series have observed no familial tendency.[4,22,23,30] In our patients 1 identical twin had renal dysplasia and the other did not.

The clinical and laboratory findings in our patients resembled those in series which emphasize clinical features.[9,27,28,32] Normal urinalysis is the rule. In our patients the renal function and urographic structure of the contralateral kidney were normal except for 1 patient with ureteral stricture.

SUMMARY

Twenty-seven children with total unilateral renal dysplasia were followed from 2 to 28 years with a mean duration of follow-up of 9.1 years.

Only 1 instance of disease (a midureteral stricture) was found in the opposite kidney.

In patients whose presentation is that of an abdominal mass which proves to be a totally dysplastic kidney, the prognosis is excellent.

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REFERENCES

1. Abeshouse, B. S. Congenital renal aplasia with calcified cystic degeneration. *J. Internat. Coll. Surgeons*, 1956, *26*, 283–296.
2. Arey, J. B. Cystic lesions of kidney in infants and children. *J. Pediat.*, 1959, *54*, 429–446.
3. Ashley, D. J., and Mostofi, F. K. Renal agenesis and dysgenesis. *J. Urol.*, 1960, *83*, 211–230.
4. Bernstein, J. Developmental abnormalities of renal parenchyma—renal hypoplasia and dysplasia. In: Pathology Annual. Edited by S. C. Sommers. Appleton-Century-Crofts, Inc., New York, 1968.
5. Bunge, R. G., and Harness, W. N. Unilateral polycystic kidney in infant. *J. Urol.*, 1959, *65*, 972–975.
6. Christeson, W. W. Simple renal cysts in newborn. *J. Urol.*, 1954, *72*, 1137–1139.
7. Cimmino, C. V. Congenital unilateral multicystic disease of kidney: entity? Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1964, *92*, 281–285.
8. Dakin, W. A. Unilateral multilocular cystic kidney. *Canad. M. A. J.*, 1940, *42*, 531–533.
9. Fine, M. G., and Burns, E. Unilateral multicystic kidney: report of six cases and discussion of literature. *J. Urol.*, 1959, *81*, 42–48.
10. Fink, A. J., Garlick, W. B., and Stein, A. Congenital cystic hydrocalycosis. *J. Urol.*, 1957, *78*, 22–27.
11. Friedenberg, M. J., Walz, B. J., McAlister, W. H., Locksmith, J. P., and Gallagher, T. L. Roentgen size of normal kidneys: computer analysis of 1286 patients. *Radiology*, 1965, *84*, 1022–1030.
12. Gleason, D. C., McAlister, W. H., and Kissane, J. M. Cystic disease of kidneys in children. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1967, *100*, 135–146.
13. Greene, L. F., Feinziag, W., and Dahlin, D. C. Multicystic dysplasia of kidney: with special reference to contralateral kidney. *J. Urol.*, 1971, *105*, 482–487.
14. Hawes, G. A. Renal hypoplasia: case report. *J. Urol.*, 1939, *42*, 500–506.
15. Kempf, F. K. Persistenz der Vorniere und des

nephrogenes Zwischenblastems mit Hamartoma im linken Nierenlager. *Ztschr. Urol.*, 1952, *45*, 36–44.

16. KISSANE, J. M. Congenital malformations. In: Pathology of the Kidney. Edited by R. M. Heptinstall. Little, Brown & Company, Boston, 1966, pp. 63–117.
17. LAUFER, I., and GRISCOM, N. T. Compensatory renal hypertrophy: absence *in utero* and development in early life. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1971, *113*, 464–467.
18. LIH, B., and WAND, R. E. Unilateral polycystic kidney in newborn. *J. Urol.*, 1952, *68*, 60–62.
19. LONGINO, L. A., and MARTIN, L. W. Abdominal masses in newborn infant. *Pediatrics*, 1958, *21*, 596–604.
20. MOORE, T. Unilateral cystic kidneys. *Brit. J. Urol.*, 1957, *29*, 3–14.
21. O'DONNELL, L. D., and PRESTI, J. C. Unilateral congenital cystic kidney in infant complicated by hydronephrosis. *J. Urol.*, 1949, *62*, 651–654.
22. OSATHANONDH, V., and POTTER, E. L. Pathogenesis of polycystic kidneys. *A.M.A. Arch. Path.*, 1964, *77*, 474–512.
23. PARKTULAINEN, K. V., HJELT, L., and SIROLA, K. Congenital multicystic dysplasia of kidney. *Acta chir. scandinav.*, 1959, *244*, 8–46.
24. PATHAK, I. G., and WILLIAMS, D. I. Multicystic and cystic dysplastic kidneys. *Brit. J. Urol.*, 1964, *36*, 318–331.
25. RISDON, R. A. Renal dysplasia. Part I. Clinicopathological study of 76 cases. *J. Clin. Path.*, 1971, *24*, 57–71.
26. SCHWARTZ, J. Unusual unilateral multicystic kidney in infant. *J. Urol.*, 1936, *35*, 259–263.
27. SPENCE, H. M. Congenital unilateral multicystic kidney: entity to be distinguished from polycystic kidney disease and other cystic disorders. *J. Urol.*, 1955, *74*, 693–706.
28. TEDESCHI, C. G., and HOLTHAM, W. H. Renal ossification: report of occurrence in aplastic kidney. *J. Urol.*, 1952, *68*, 50–59.
29. UTHOFF, J. Diagnosis of case of solitary kidney with accompanying vestigial cystic remnant of other kidney. *Urol. & Cutan. Rev.*, 1936, *40*, 559–562.
30. VELLIOS, F., and GARRETT, R. A. Congenital unilateral multicystic disease of kidney. *Am. J. Clin. Path.*, 1961, *35*, 244–254.
31. WAKELEY, C. Case of unilateral polycystic kidney in child. *Brit. J. Surg.*, 1930, *18*, 162–165.
32. WEINBERG, S. R., O'CONNOR, W. J., and SENGER, F. L. Unilateral multicystic renal disease of newborn. *A.M.A. Am. J. Dis. Child.*, 1956, *92*, 576–580.

PNEUMOTHORAX AND PNEUMOMEDIASTINUM ASSOCIATED WITH RENAL MALFORMATIONS IN NEWBORN INFANTS*

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THE clinical and pathologic features of renal agenesis (Potter's syndrome) have been well described and documented.[1,3,7–10] When present, recognition of the characteristic facies of this syndrome should assist in the early diagnosis of this uniformly fatal disease entity. The face, as described by Potter[8,10] shows an increased space between the eyes and a prominent fold which arises at the inner canthus and sweeps downward and laterally below the eyes. This is associated with a flattened nose, receding chin, and enlargement and decreased chondrification of the low-set ears. The over-all appearance suggests premature senility (Fig. 1).

Bilateral pulmonary hypoplasia is an almost constant finding in renal agenesis and may also be present in infants with cystic dysplastic kidneys.[1,3,4,10] It is this marked hypoplasia of the lungs rather than the renal anomaly which contributes to the early demise of these infants. As yet, there is no satisfactory explanation for the association of pulmonary hypoplasia with renal agenesis or cystic dysplasia. Gruenwald[4] noted that there was no correlation between the size of the kidneys and the degree of pulmonary hypoplasia.

Blanc *et al.*[1] noted that pulmonary hypoplasia is a constant finding when oligohydramnios has been present antenatally and that although it was most commonly associated with renal malformations with absence of, or inadequate, urine excretion, pulmonary hypoplasia was present when there was a nonrenal etiology of oligohydramnios as well. Therefore, he postulated that pulmonary hypoplasia was likely due to increased external pressure secondary to the deficiency of amniotic fluid.

The purpose of this paper is to present roentgenologic findings which have been uniformly present on initial presentation in 9 patients with severe renal malformation seen at The Montreal Children's Hospital between 1963 and 1971. The presenting



FIG. 1. Case 1. Typical Potter's facies in infant with renal agenesis. Note prominent epicanthal folds, low position of ears and senile appearance.

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TABLE I

SURVIVAL OF INFANTS WITH POTTER'S SYNDROME AS RELATED TO GESTATIONAL PERIOD AND BIRTH WEIGHT

Case	Sex	Gestation (wk.)	Birth Weight (gm.)	Age at Admission	Age at Death
I B.	M	40	2,230	45 min.	6 hr.
II J.	M	32	1,535	2 hr. 50 min.	14 hr.
III D.	M	36	1,720	4 hr.	10 hr.
*IV R.	F	41	2,665	3 hr.	33 da.
V C.	M	35	1,820	1 hr.	3 hr.
VI Hu.	M	38	2,210	50 min.	9 hr.
VII Hi.	F	39	2,440	2½ hr.	8 hr.
VIII L.	M	37	2,600	2½ hr.	7 hr.
IX Bu.	M	40	2,900	2 hr.	3 hr.

* This baby had bilateral pneumothorax present shortly after birth; they were drained and she subsequently died of renal failure. The other 8 infants died a respiratory death.

signs are massive unilateral or bilateral tension pneumothoraces which may be associated with pneumomediastinum.

MATERIAL

Nine infants with a proven autopsy diagnosis of bilateral renal agenesis or cystic dysplasia (Potter Type II and IV),[7] with roentgenologic evidence of pneumothorax, usually bilateral, or unilateral pneumothorax and pneumomediastinum, are reported. The sex of the infants, gestational age, birth weight, and time of death are detailed in Table I. Seven of the infants were males. Only 3 infants, 2 males and 1 female, were products of term pregnancies. Eight of the 9 infants died within 14 hours of birth. The other infant (Case IV), a full term female, survived for 33 days and died of renal failure.

All 9 infants were born in outside hospitals and immediately transferred to The Montreal Children's Hospital because of severe respiratory distress beginning at birth and present prior to any attempt at resuscitation. In all cases, the roentgenographic features were already present at the time of admission from the referring institution (Table I).

CLINICAL FINDINGS

Upon admission to The Montreal Children's Hospital, all infants were in severe respiratory distress. At transfer, there was a history of active resuscitative measures taken because of the distress present at birth in 7 of the 9 infants. The record of the management of the other 2 infants was not available. However, their clinical appearance on admission is in keeping with severe respiratory failure from birth onward. Clinical examination on admission revealed respiratory distress in all cases, severe enough to require active supportive measures.

The facies of 7 of the 9 infants were sufficiently characteristic to warrant the clinical retrospective diagnosis of renal agenesis (Table II).

The cause of death in the 8 infants dying within a few hours of birth was respiratory failure. Case IV died of renal failure at 33 days, after successful drainage of bilateral pneumothoraces.

ROENTGENOLOGIC FINDINGS

(Table II)

Chest roentgenograms were made within an hour of admission in each case. Six of the infants were shown to have bilateral pneumothoraces, 3 were associated with pneumomediastinum (Fig. 2, *A* and *B*). Three had unilateral pneumothorax, 1 of which was also associated with pneumomediastinum. In all cases, there was a large

TABLE II

RENAL AND PULMONARY FINDINGS IN 9 CASES OF POTTER'S SYNDROME

Case	Renal Anomalies	Typical Facies (Potter)	Initial Roentgenologic Findings	Pathologic Findings	Lungs
I B.	Bilateral renal and ureteric agenesis, absent bladder trigone, epididymis, vas deferens	Yes	Bilateral pneumothorax	Bilateral pneumothorax	Hypoplasia
II J.	Bilateral renal dysplasia (Potter Type II)	Yes	Left pneumothorax	Bilateral pneumothorax	Hypoplasia
III D.	Bilateral renal and ureteric agenesis, absent bladder trigone, epididymis, vas deferens	Yes	Bilateral pneumothorax, pneumomediastinum	Left pneumothorax, pneumomediastinum, bilateral hydrothorax	Hypoplasia
IV R.	Bilateral renal dysplasia (Potter Type II)	Low set ears	Bilateral pneumothorax	Interstitial emphysema—right, bilateral fibrinous pleurisy (post thoracentesis)	Not grossly hypoplastic
V C.	Bilateral renal and ureteric agenesis, absent bladder trigone	Yes	Right pneumothorax, pneumomediastinum	Bilateral pneumothorax, pneumomediastinum	Hypoplasia
VI Hu.	Bilateral renal and ureteric agenesis, absent bladder trigone, epididymis, vas deferens	Yes	Bilateral pneumothorax, pneumomediastinum	Bilateral pneumothorax	Hypoplasia
VII Hi.	Bilateral renal and ureteric agenesis, absent bladder trigone, epididymis, vas deferens	Yes	Bilateral pneumothorax	Bilateral pneumothorax	Hypoplasia
VIII L.	Bilateral cystic dysplasia (Potter Type IV), stenosis of proximal ureters; patent urachus, posterior urethral valves	Sloping forehead, slanting eyes, low set ears, high arched palate	Bilateral pneumothorax, pneumomediastinum	Bilateral pneumothorax, pneumomediastinum	Hypoplasia
IX Bu.	Cystic dysplasia (Potter Type II), horseshoe kidneys, stenotic and double right ureter	Yes	Left pneumothorax	Resolved pneumothorax, subpleural bullae	Hypoplasia

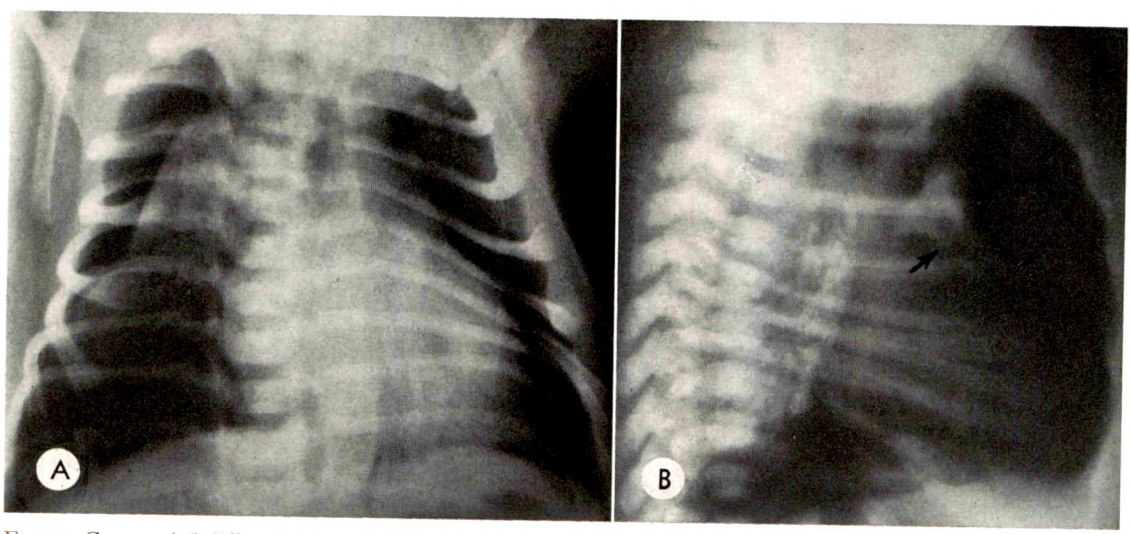


FIG. 2. Case I. (*A*) Bilateral pneumothorax and pneumomediastinum at 2 hours of age. (*B*) Lateral view shows thymus (arrows) outlined by pneumomediastinum.

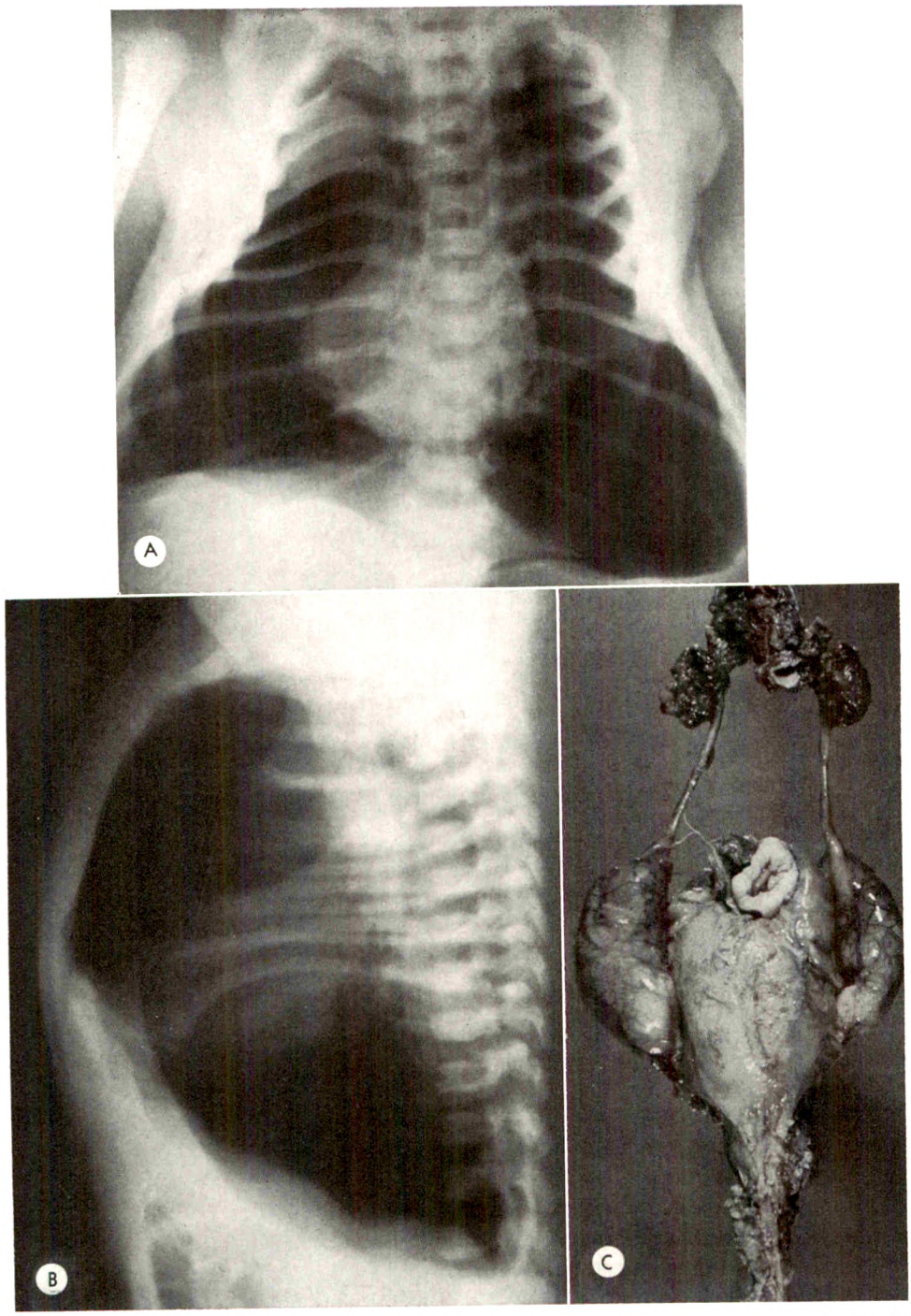


Fig. 3. Case viii. (*A*) Large bilateral pneumothoraces and pneumomediastinum at age $3\frac{1}{2}$ hours. Flaring of the lower chest wall. (*B*) Horizontal beam lateral view shows marked thoracic overdistention due to pneumothorax and pneumomediastinum. (*C*) Autopsy specimen showing small cystic dysplastic kidneys (top), stenotic proximal ureters and dilatation of lower ureters, bladder and posterior urethra due to posterior urethral obstruction.

amount of extrapulmonary air which was easily demonstrable roentgenographically. Six of the 9 infants had more than 1 chest roentgenogram during their period of survival. In no case was there any evidence of pneumothorax or pneumomediastinum other than those documented on the first roentgenologic examination. The initial examination was carried out within 5 hours of birth in all of the infants.

PATHOLOGIC FINDINGS

(Table II)

Five of the 9 infants were found to have bilateral renal and ureteric agenesis associated with absence of the trigone of the bladder, epididymis, and vas deferens (mesonephric failure). Three infants had bilateral renal dysplasia (Potter Type II).[7] The infant who survived for 33 days was one of those with Potter Type II renal dysplasia. One infant had absent abdominal musculature or "prune belly" syndrome,[12] and posterior urethral valves (Fig. 3, *A–C*). The kidneys of this infant were classified as being Potter Type IV dysplasia secondary to urethral obstruction.[7]

The intrathoracic findings are presented in Table II. With the exception of the infant who survived for 33 days, the lungs of all infants were moderately to severely hypoplastic, 7 of these weighing less than 50 per cent of normal. The lungs of Case IV were not hypoplastic, probably as a result of 1 month postnatal growth. All infants with the exception of Case IV showed evidence of either previous pneumothorax or pneumomediastinum. The presence of pneumomediastinum or pneumothorax seen at autopsy but not seen on roentgenograms could be attributed to development of these entities between the time of roentgenography and time of autopsy.

The associated abnormalities of the skin, extremities and other organs are shown in Table III.

DISCUSSION

Pulmonary hypoplasia with severe renal malformations is well known and several authors have reported the association of the condition with pneumothorax and pneumomediastinum.[1,4,6] However, what needs to be stressed is the virtual diagnostic value of such pneumothoraces, particularly when they are bilateral, appearing initially from birth onward without underlying cause. Although the facies is frequently characteristic enough in retrospect to the seasoned observer of the classical Potter's syndrome, it is not a constant finding in cystic dysplasia[6] and the uniform presentation of the condition as a problem of respiration rather than of renal insufficiency may dilute the practical value of the facial appearance alone, particularly when it is distorted by gasping respiratory efforts.

Pneumothorax is not an uncommon complication of both the respiratory distress syndrome (hyaline membrane disease) and pneumonia, particularly that due to aspiration of meconium in the lung. In both instances, the pathogenesis is largely dependent on respiratory efforts made in the face of a partial obstruction of the airways.[2] In hyaline membrane disease, however, pneumothorax classically occurs after 48 to 72 hours, when the infant who is recovering begins to make more effective and vigorous efforts. While pneumothorax does occur early, particularly in post-mature infants with meconium aspiration, both the clinical and roentgenologic evidence of underlying disease will usually distinguish this from the roentgenologic events reported here. The fact that Case IV survived for 33 days after management of the pulmonary problem in an infant on renal failure regime, not only underlines the importance of the pulmonary debility in the immediate neonatal period with this complex, but does suggest some possibility of future salvage of some cases *via* transplantation or dialysis procedures if vigorous and early attention to the pulmonary problem is successful. In this connection it is of interest that, although bilateral pneumothorax was present in this infant, the lungs were not anatomically hypoplastic at the age of 33 days. This suggests a postnatal maturational effect.[11]

TABLE III

ASSOCIATED FINDINGS IN 9 CASES OF POTTER'S SYNDROME

Case	Skin	Extremities	Other
I B.	Excessively lax	Hyperextensible knee joints	
II J.	Excessively lax	Hyperextensible knee and elbow joints, congenital dislocation hip—bilateral	
III D.	Excessively lax	Congenital dislocation hip—left	Polygyria
IV R.	Edematous	Bilateral simian creases, hammer heels	Focal cerebromalacia, cardiomegaly with patent ductus arteriosus, hydroperitoneum
V C.	Not mentioned	Congenital dislocation hip—bilateral, spade-like hands	
VI Hu.	Excessively lax	Spade-like hands	Erythroblastosis (anti-B)
VII Hi.	Excessively lax	Hyperextensible finger and ankle joints	Subdural, subarachnoid, intraventricular hemorrhage, hypoplastic uterus and vagina, hemivertebra, congenital heart disease
VIII L.	Thick, prominent folds	Incurved foot	Absent abdominal musculature, malrotation of bowel
IX Bu.	Wrinkled	Spade-like hands, bilateral talipes equinovarus	Bifid scrotum, imperforate anus

It is likely that the introduction of positive intrapulmonary pressure, whether due to voluntary respiratory efforts on the part of the infants or due to active resuscitative measures, resulted in the rupture of the alveoli of the hypoplastic lungs with pneumothorax and pneumomediastinum. In contrast, a lung with a normal amount of pulmonary alveoli should be less apt to rupture under the usual resuscitative techniques, since transpulmonary pressures required for initial lung expansion are often far in excess of those normally generated with even the most active resuscitative measures.[5]

The cases presented above illustrate the wide spectrum of renal abnormalities with which pulmonary hypoplasia may be associated. The most common malformation is total renal agenesis. Cystic dysplasia of the kidneys (Potter Type II and IV) was also present frequently. Any of these abnormalities might be expected to result in oligohydramnios strengthening the observation of Blanc *et al.*[1] that oligohydramnios is commonly associated with pulmonary hypoplasia.

While some patients had obvious Potter's facies or other deformities pointing to underlying renal abnormalities, the presence of unexplained pneumothoraces or pneumomediastinum initially at birth is a more constant feature which should alert the clinician to the presence of severe renal disease.

SUMMARY

Nine newborn infants, with severe otherwise unexplained respiratory distress beginning immediately at birth, were found to

have pneumothoraces or pneumomediastinum on chest roentgenograms.

Typical facies associated with Potter's syndrome was identified in 7.

At autopsy, renal anomalies, in the form of agenesis or cystic dysplasia, were present.

The 8 infants who died in the immediate neonatal period showed pulmonary hypoplasia at autopsy—a finding which was absent in the infant who survived to 33 days of life.

The presence of unexplained pneumothorax and pneumomediastinum in the immediate newborn period should alert the clinician to the presence of underlying renal abnormalities, even in the absence of suggestive external features.

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REFERENCES

1. Blanc, W. A., Apperson, J. W., and McNally, J. Pathology of newborn and of placenta in oligohydramnios. *Bull. Sloane Hosp. Wom.*, 1962, *8*, 51–64.
2. Chernick, V., and Avery, M. E. Spontaneous alveolar rupture at birth. *Pediatrics*, 1963, *32*, 816–824.
3. Davidson, W. M., and Ross, G. I. M. Bilateral absence of kidneys and related congenital anomalies. *J. Path. & Bact.*, 1954, *68*, 459–472.
4. Gruenwald, P. Hypoplasia of lungs. *J. Mt. Sinai Hosp., New York*, 1957, *24*, 913–919.
5. Karlberg, P. J. E. Breathing and its control in premature infants. In: Physiology and Prematurity Transactions of the second conference 1957, Princeton, N. J. Edited by J. T. Lanman. Josiah Macy Jr., Foundation, New York, 1958.
6. Liberman, M. M., Abraham, J. M., and France, N. E. Association between pneumomediastinum and renal anomalies. *Arch. Dis. Childhood*, 1969, *44*, 471–475.
7. Osathanondh, V., and Potter, E. L. Pathogenesis of polycystic kidneys. *A.M.A. Arch. Path.*, 1964, *77*, 459–512.
8. Potter, E. L. Bilateral renal agenesis. *J. Pediat.*, 1946, *29*, 68–76.
9. Potter, E. L. Facial characteristics of infants with bilateral renal agenesis. *Am. J. Obst. & Gynec.*, 1946, *51*, 885–888.
10. Potter, E. L. Pathology of The Fetus and Infant. Second edition. Year Book Publishers, Inc., Chicago, 1961.
11. Reid, L. Embryology of lung. In: Development of the Lung. Ciba Foundation Symposium. Edited by A. V. S. deReuck and R. Porter. J. & A. Churchill Ltd., London, 1967.
12. Silverman, F. N., and Huang, N. Congenital absence of abdominal muscles associated with malformation of genitourinary and alimentary tracts: report of cases and review of literature. *Am. J. Dis. Child.*, 1950, *80*, 91–124.

THE ELUSIVE ECTOPIC URETEROCELES*

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ECTOPIC ureteroceles are dangerous congenital anomalies. They frequently lead to recurrent urinary tract infections and ureteral obstruction which can cause irreversible damage to the kidneys.[1,7,17,18,21] Furthermore, they are the most common cause of acute bladder outlet obstruction in infant girls.[21] Ectopic ureteroceles occur more frequently in girls than in boys,[12,18,21] but in either sex they may be associated with serious urologic abnormalities. It is important that radiologists and urologists alike be aware of the roentgenographic features of ectopic ureteroceles for they are not always easily recognized.

In our experience, both radiologists and urologists often fail to diagnose ectopic ureteroceles. This occasionally happens because the observer does not recognize the classic roentgenologic features. But diagnostic problems can also arise because many types of ectopic ureterocele constantly undergo changes in size, shape and position.[17,18,20] They may easily become undetectable on conventional roentgenograms. Indeed, they have truly earned the title "The Elusive Ectopic Ureteroceles." Even though ectopic ureteroceles can be concealed, the radiologist can detect them by using certain simple precautions which will be discussed.

Apart from the dynamic factors which make the roentgenologic diagnosis difficult, several types of ectopic ureterocele occur which will elude detection unless the radiologist is aware of their existence. Previous classifications in the radiologic literature have not been sufficiently elaborate to be an adequate guide to the radiologist.[1,4,6,11–13,20] In this article, we present a modified version of a newer, more comprehensive classification[19] and review current theories of the embryology of ectopic ureteroceles. We have selected illustrative cases which will demonstrate certain pertinent points regarding the dynamic nature, classification, unusual presentations and complications of ectopic ureteroceles.

I. DEFINITIONS AND CLASSIFICATION

The ureter which drains the lower pole of a duplex kidney normally opens in the lateral angle of the trigone of the bladder and is called the *orthotopic ureter*.[18] By contrast, the ureter which drains the upper pole of a duplex kidney does not open in the lateral angle of the trigone and is known as the *ectopic ureter*.[6,11] Indeed, any ureter which does not open in the lateral angle of the trigone is called an ectopic ureter.[6,11]

An ectopic ureterocele is a cystic ballooning of the lower end of an ectopic ureter lying between the mucosa and muscle of the bladder.[22] Complete ureteral duplication is present on the same side, but may be found on the opposite side in about 50 per cent of cases.[13] Rarely–usually in boys–ectopic ureteroceles occur without ureteral duplication.[21] The right and left sides are equally involved.

We have modified the following comprehensive classification of ectopic ureteroceles from Stephens[19] (Fig. 1–4). It will serve to re-acquaint the radiologist with the possible variations of the abnormality.

A. STENOTIC ECTOPIC URETEROCELES

(about 40 per cent of cases)

This type of ectopic ureterocele is characterized by a congenitally small orifice which communicates with the bladder lu-

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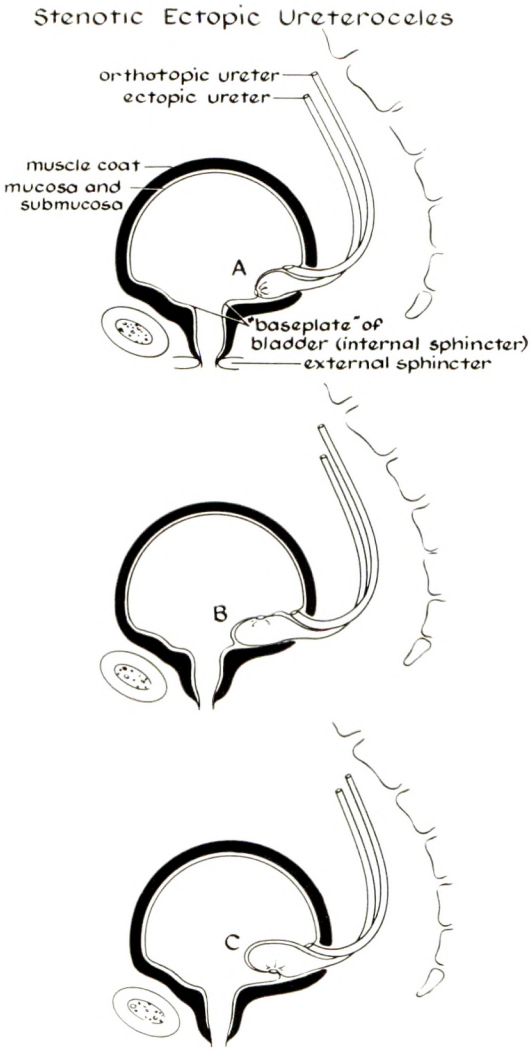


Fig. 1. Diagrammatic illustrations of stenotic ectopic ureteroceles. (Redrawn from Stephens.[19])

men (Fig. 1). The orifice of the ectopic ureterocele can be located in 3 different places in the ureterocele: (1) at the terminal tip of the bulbous dilatation of the ureter; (2) on the superior aspect of the ectopic ureterocele; (3) on the inferior surface of the ectopic ureterocele.

B. SPHINCTERIC ECTOPIC URETEROCELES

(about 40 per cent of cases)

The ectopic ureterocele terminates within the internal sphincter (Fig. 2). The ureter distal to the ectopic ureterocele may continue distally to open into the genitourinary tract in 1 of 2 locations: (1) in the posterior urethra; (2) distal to the external sphincter; this occurs only in females. A sphincteric ectopic ureterocele can have a normal or an enlarged ureteral orifice.

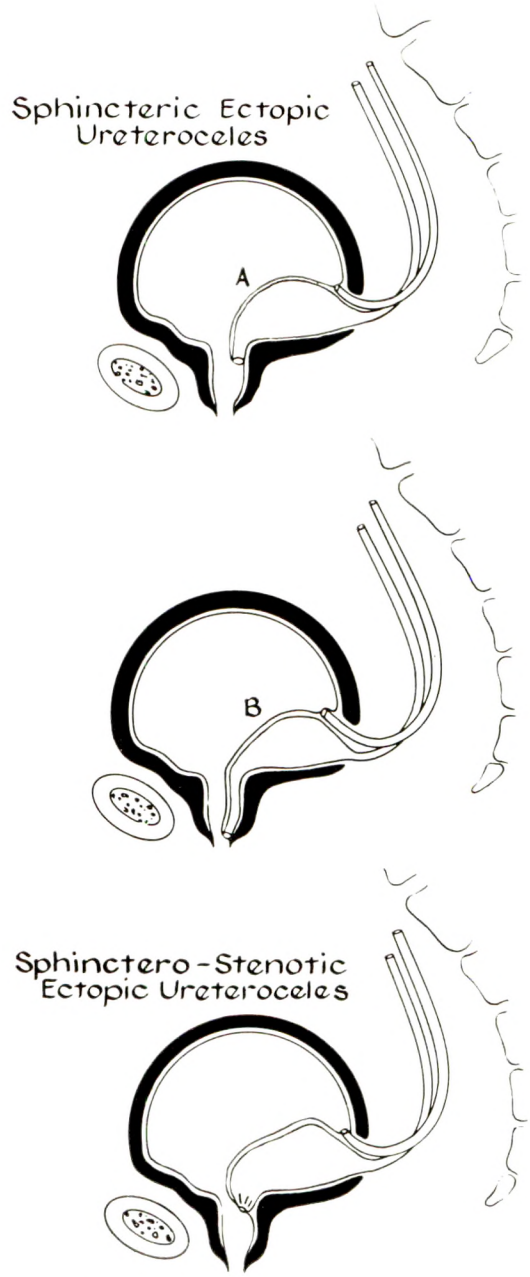


Fig. 2. Diagrammatic illustrations of sphincteric ectopic ureteroceles. (Redrawn from Stephens.[19])

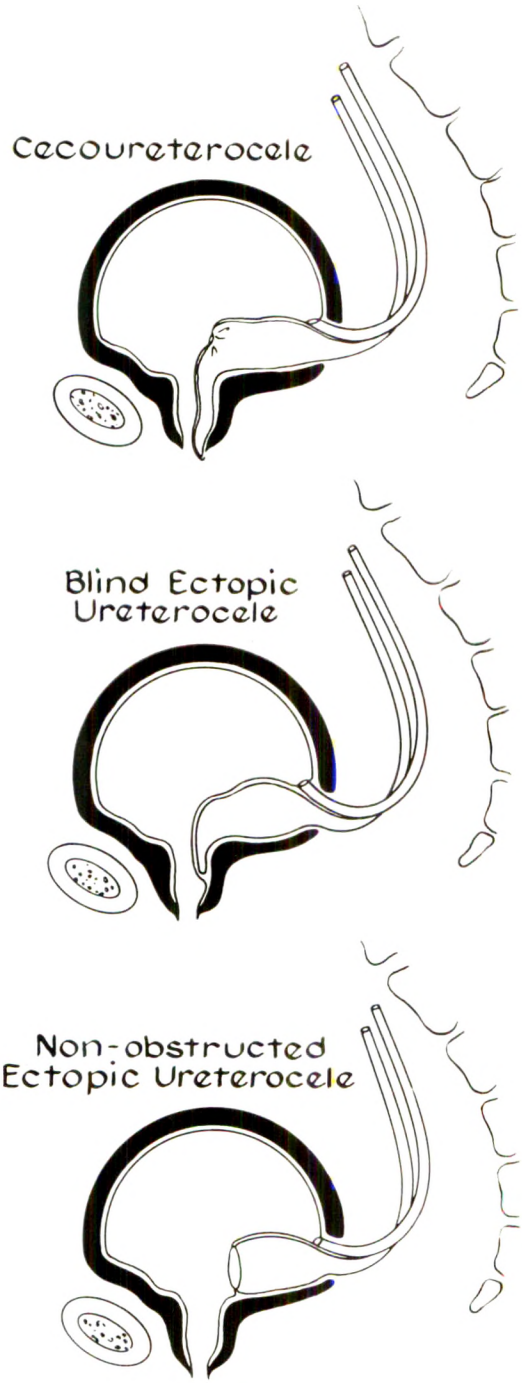


FIG. 3. Diagrammatic illustrations of 3 rare types of ureteroceles. (Redrawn from Stephens.[19])

C. SPHINCTEROSTENOTIC ECTOPIC URETEROCELE

(about 5 per cent of cases)

The stenosed ureteral orifice is situated in the floor of the urethra or beyond (Fig. 2).

D. CECOURETEROCELE

(about 5 per cent of cases)

The lumen of the ectopic ureterocele extends beyond its orifice as a long tongue or "cecum" in the urethral submucosa. The orifice of the ectopic ureterocele communicates with the bladder lumen and is large and incompetent (Fig. 3).

E. BLIND ECTOPIC URETEROCELE

(about 5 per cent of cases)

Atrophy of the ureter distal to the ureterocele which must then end blindly (Fig. 3).

F. NONOBSTRUCTED ECTOPIC URETEROCELE

(about 5 per cent of cases)

Terminal expansion of the ureter which has a large orifice existing in the bladder (Fig. 3).

G. PSEUDOECTOPIC URETEROCELE

(rare)

We call pseudoectopic ureterocele any coiled, dilated, distal ureter which forms a mass in the submucosa of the bladder[18,22] (Fig. 4).



FIG. 4. Diagrammatic illustration of a pseudoectopic ureterocele.

II. EMBRYOLOGY

It is important that the radiologist understand the normal and abnormal embryology of the ureter in order to evaluate the theories which try to explain the development of ectopic ureteroceles. The theories themselves have been widely misunderstood.

A. NORMAL EMBRYOLOGY[8,11,19]

(Fig. 5)

Between the 30th and 35th day, the urorectal septum descends and partitions the cloaca into a posterior anorectal and an anterior vesicourethral canal. At this stage, the Wolffian duct connects the mesonephros to the vesicourethral canal. A bud —the future ureter—arises from the dorsal surface of the lower end of the Wolffian duct. The Wolffian duct expands and becomes incorporated into the vesicourethral canal. Then the Wolffian duct migrates into the upper part of the urethra. Simultaneously, the ureter moves up and out to open in the lateral angle of the trigone. It may also be possible that the Wolffian duct forms a caudal loop, which is taken into the vesicourethral canal, but is separated from the lumen of the canal by an epithelial membrane.[8,9] When the membrane disappears, the resulting orifice opens much lower in the vesicourethral canal.

In the female, the Müllerian ducts open in the urethra medial to the Wolffian ducts. The Müllerian ducts then grow, incorporating into themselves the Wolffian ducts. The Müllerian ducts migrate from the bladder neck down the urethra to the vestibule. The vesicourethral canal expands, and becomes more like the usual bladder shape.

The Wolffian duct in the male becomes the seminal vesicles and vas deferens. In the female, the Wolffian duct atrophies to form Gartner's duct.

B. URETERAL DUPLICATION[11,19]

(Fig. 5)

Two completely separate ureters will form if 2 ureteral buds arise from 1 Wolffian duct. First, the distal end of the Wolffian duct becomes absorbed into the vesicourethral canal until the lower ureter's orifice (which is connected to the lower part of the Wolffian duct) emerges in the canal. While the Wolffian duct, with the upper ureter still attached to it, migrates medially and downwards, the orifice of the lower ureter progresses upwards and outwards. As a result of further absorption of the distal end of the Wolffian duct, the orifice of the ureter which drains the upper pole eventually opens directly into the vesicourethral canal.

Virtually all duplicated ureters pass through the bladder wall in a common tunnel.[18] On rare occasions, a ureteral bud which retains a high attachment to the Wolffian duct will form a ureter which pursues a completely extravesical course.[19]

In the female, during migration of the Müllerian and Wolffian ducts from the bladder neck to the vestibule, the latter may carry an ectopic ureter with it.[19] This ureter lies submucosally. It can open anywhere along the posterior wall of the urethra or the urethrovaginal septum or the hymen.

In about 85 per cent of cases of complete ureteral duplication, the orifice of the ureter which drains the upper pole opens below and medial to the ureter draining the lower pole (the "Weigert–R. Meyer Rule").[14,18] In the remaining 15 per cent of cases, the ureteral orifices can open anywhere along Stephen's "ectopic pathway." This pathway hooks like a shepherd's crook around the superior and medial aspects of the orthotopic orifice[18] (Fig. 6).

C. DEVELOPMENTAL THEORIES OF ECTOPIC URETEROCELES

Several theories have been proposed to explain why an ectopic ureterocele forms:

(1) *Chwalle's Theory*[3] (Fig. 7). This theory postulates that the persistence of Chwalle's membrane,[3,9] which covers the ureteral orifices until about 60 days, leads to the formation of a ureterocele.

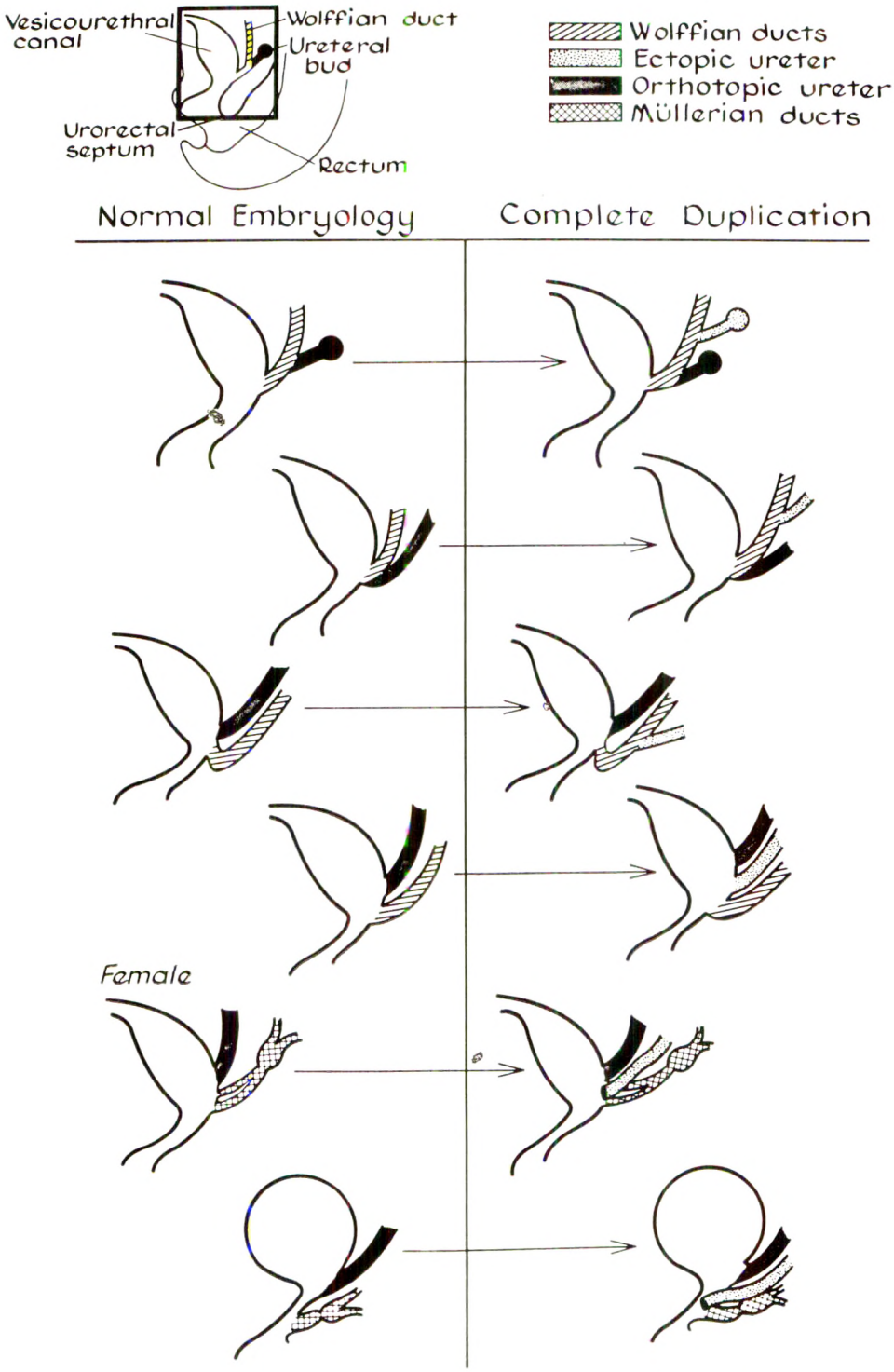


FIG. 5. Normal embryology and embryology of ureteral duplications.

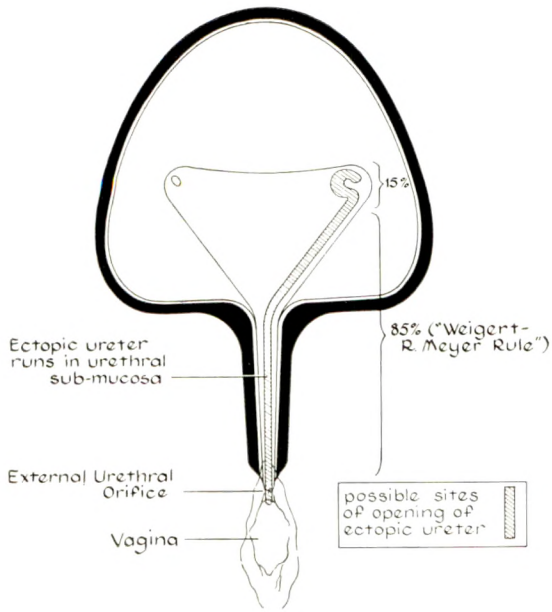


Fig. 6. Stephens' ectopic pathway. (Redrawn from Stephens.[18])

(2) *Ericsson's Theory*[7] (Fig. 8). Ericsson's theory proposes that the membrane between the looped Wolffian duct and the vesicourethral canal is not reabsorbed: a caudal opening forms in the membrane for the Wolffian duct. The orifice of the ectopic ureter is buried beneath the membrane.

Both Chwalle's and Ericsson's theories might explain how stenotic ectopic ureteroceles are formed, but they do not explain the sphincteric types. Stephens[19] has suggested a more logical and comprehensive theory which can account for all types, including those which have large and incompetent orifices.

(3) *Stephens' Theory*[19] (Fig. 9). Stephens theorizes that failure of expansion of the ureteral orifice and not occlusion by a membrane causes the narrow orifice of stenotic ectopic ureteroceles.

When the ureter has migrated normally, so that its orifice lies in the lateral angle of the trigone, the stimulus which causes the bladder to expand does not affect the ureter. By contrast, the bladder stimulus will affect the distal end of an ectopic ureter which expands, like the bladder, to form a ureterocele. If the ureteral bud is of normal size, the orifice of the ectopic ureterocele will also be normal. On the other hand, if the ureteral bud is abnormally large, the ectopic ureterocele will have a large orifice. This theory explains why an ectopic ureterocele can form, even though the ureteral orifice is normal or larger than normal.

Stephens suggests that blind ectopic ureteroceles result if, in the female, an ectopic ureter is attached to the Wolffian duct when it atrophies. The distal end of the ectopic ureter will atrophy along with the Wolffian duct. Cecoureteroceles, however, will form if a small part of an ectopic ureterocele overlaps the Müllerian duct. The Müllerian duct, when it migrates down the urethra, carries the overlap with it as a blind pouch or cecum.

III. PATHOPHYSIOLOGY

An ectopic ureterocele is not a static structure.[17,18,20] Radiologists should appreciate that ectopic ureteroceles, es-

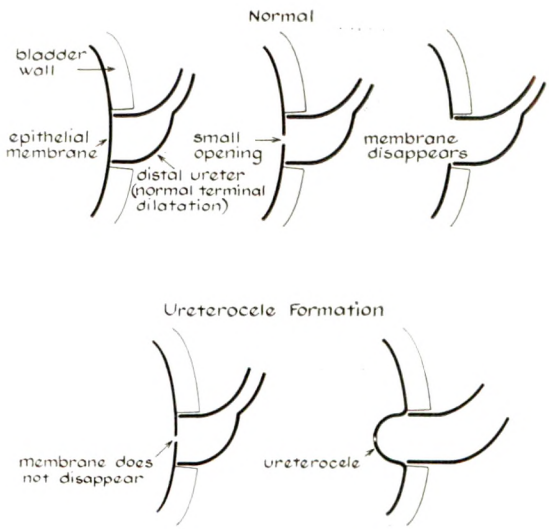


Fig. 7. Chwalle's theory of embryologic development of ectopic ureteroceles.

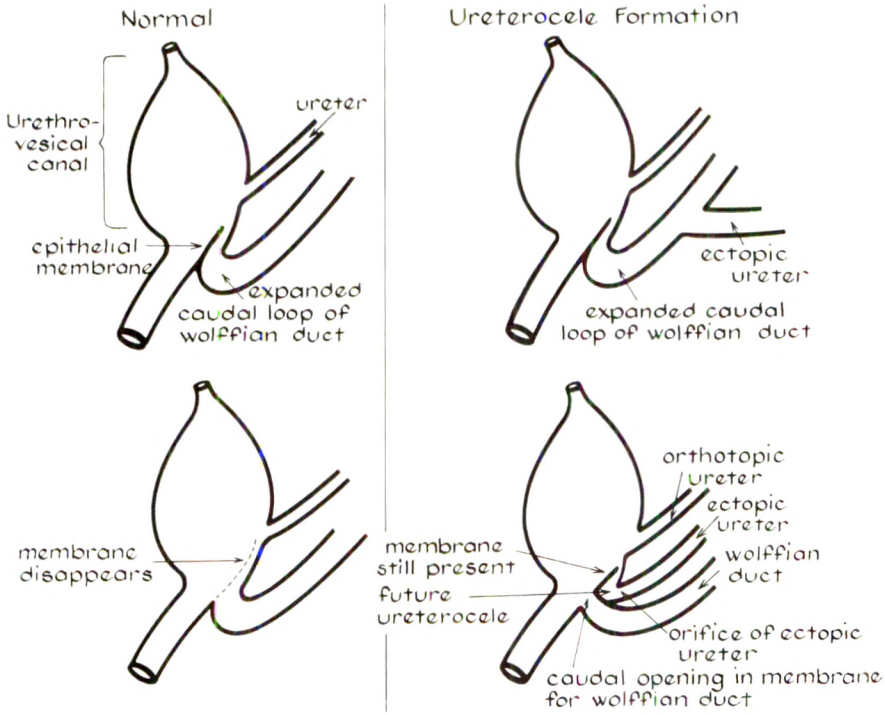


FIG. 8. Ericsson's theory of embryologic development of ectopic ureteroceles.

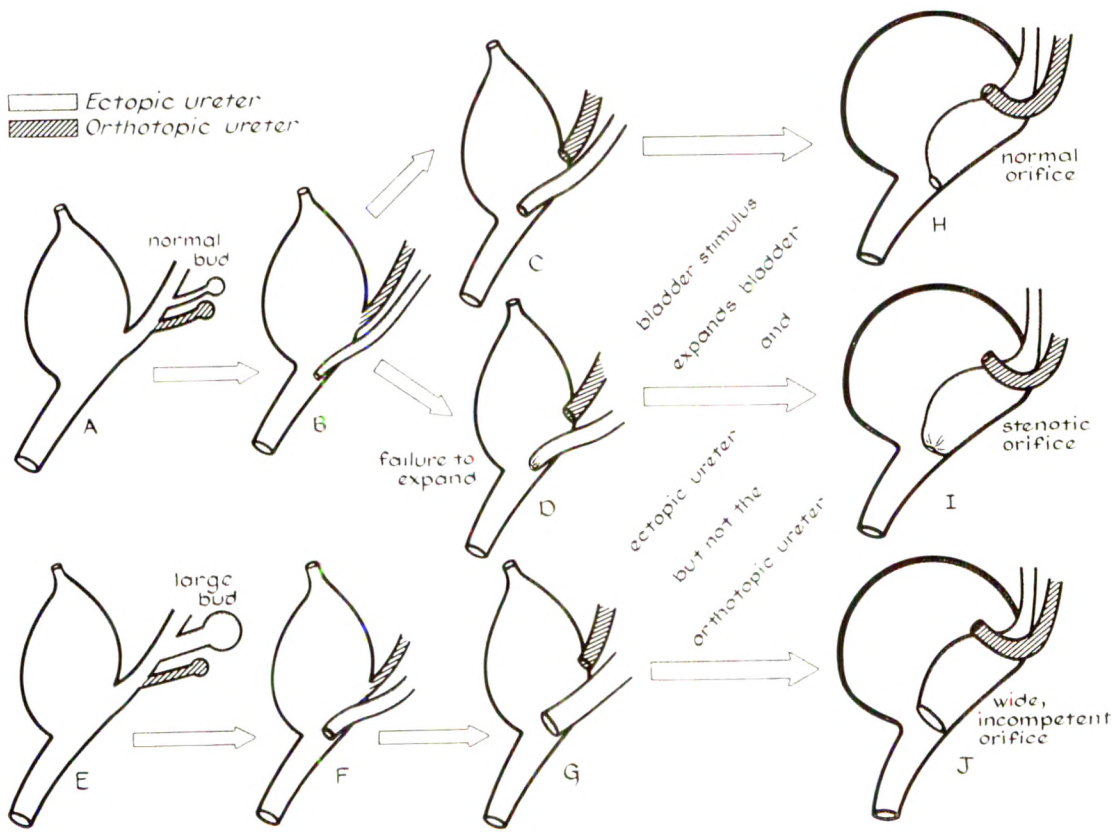


FIG. 9. Stephens' theory of embryologic development of ectopic ureteroceles.

pecially the sphincteric type, undergo many dynamic changes which will affect the roentgenographic appearances.

A. Effect on Ureter Bearing the Ectopic Ureterocele

(1) *Obstruction.* Obstruction of the ureter can occur in 2 ways. If severe stenosis of the orifice of an ectopic ureterocele exists, not only will the ectopic ureterocele remain permanently distended,[17,18] but hydronephrosis and hydroureter will occur. Because the orifice of a sphincteric ectopic ureterocele opens into the bladder neck or beyond, the internal sphincter obstructs the ureter when the bladder neck is closed. Obstruction of a sphincteric ureterocele's orifice when the bladder neck closes, plus urethroureteral reflux during voiding in some cases, is the actual cause of the hydroureter and hydronephrosis.[17,18]

(2) *Reflux.* Reflux of urine from the bladder neck or urethra will fill a sphincteric ectopic ureterocele in about 45 per cent of cases[13] (Fig. 10, *A* and *B*). If the bladder neck is open and the pressure in the urethra exceeds the pressure in the emptied sphincteric ectopic ureterocele, urine will flow in a retrograde fashion into the ureterocele. Sometimes an ectopic ureterocele ruptures spontaneously.[13] Because a ruptured ectopic ureterocele communicates freely with the bladder lumen, it allows vesicoureteral reflux.[13]

(3) *Emptying.* During voiding, when the sphincteric obstruction is released, a sphincteric ectopic ureterocele can peristalse; both the bladder and the ectopic ureterocele empty together[17] (Fig. 10*A*). The ability of a sphincteric ectopic ureterocele to contract depends on the amount of muscle in its wall. Muscle can be plentiful, or partially or totally absent. About 80 per cent of sphincteric ectopic ureteroceles possess sufficient muscle for peristalsis to occur.[17] The stenotic type of ectopic ureterocele can also contract and empty if mild stenosis exists, because it usually has a strong muscle wall.[18]

(4) *Prolapse.* A distended ectopic ureterocele can prolapse down the urethra during voiding.[7,18,21] Prolapse can persist even after cessation of voiding. Three factors will enhance prolapse into the urethra:[18] (a) filling from the urethra during voiding; (b) retention because of stenosis of the orifice; (c) peristalsis of an enlarged urine-filled ureter which refills the ectopic ureterocele from above.

About 40 per cent of prolapsed ectopic ureteroceles obstruct the urethra.[12] Sphincterostenotic ectopic ureteroceles cause the most severe obstruction from prolapse during voiding.[18] Occasionally, the prolapsed portion will necrose.[21]

B. Dysplastic Changes in the Upper Pole

The defect exists from the time the fetal kidney begins to excrete.[7,22] Frequently, the resultant obstruction in fetal life will cause Potter Type 4 cystic changes in the affected upper pole.[16,22]

C. The Rest of the Urinary Tract

(1) *Bladder Neck Obstruction.* Ectopic ureteroceles are the most common cause of neonatal bladder outlet obstruction in girls.[21]

(2) *Ureteral Orifices not Bearing the Ectopic Ureterocele.* The ectopic ureter traverses the muscular wall of the bladder in the same tunnel as the orthotopic ureter. Initially, it is situated in front of the orthotopic ureter, but it then swings behind the orthotopic ureter's orifice, where it lies in the submucosa between this orifice and the muscle wall of the bladder.[18] A distended ectopic ureterocele will elevate, flatten and stretch the orthotopic ureter's orifice and either obstruct it or make it incompetent.[18] An ectopic ureterocele can extend across the trigone and obstruct or interfere with the antireflux mechanisms of the ureter (or ureters) on the opposite side.

(3) *Infection.* Commonly, patients who

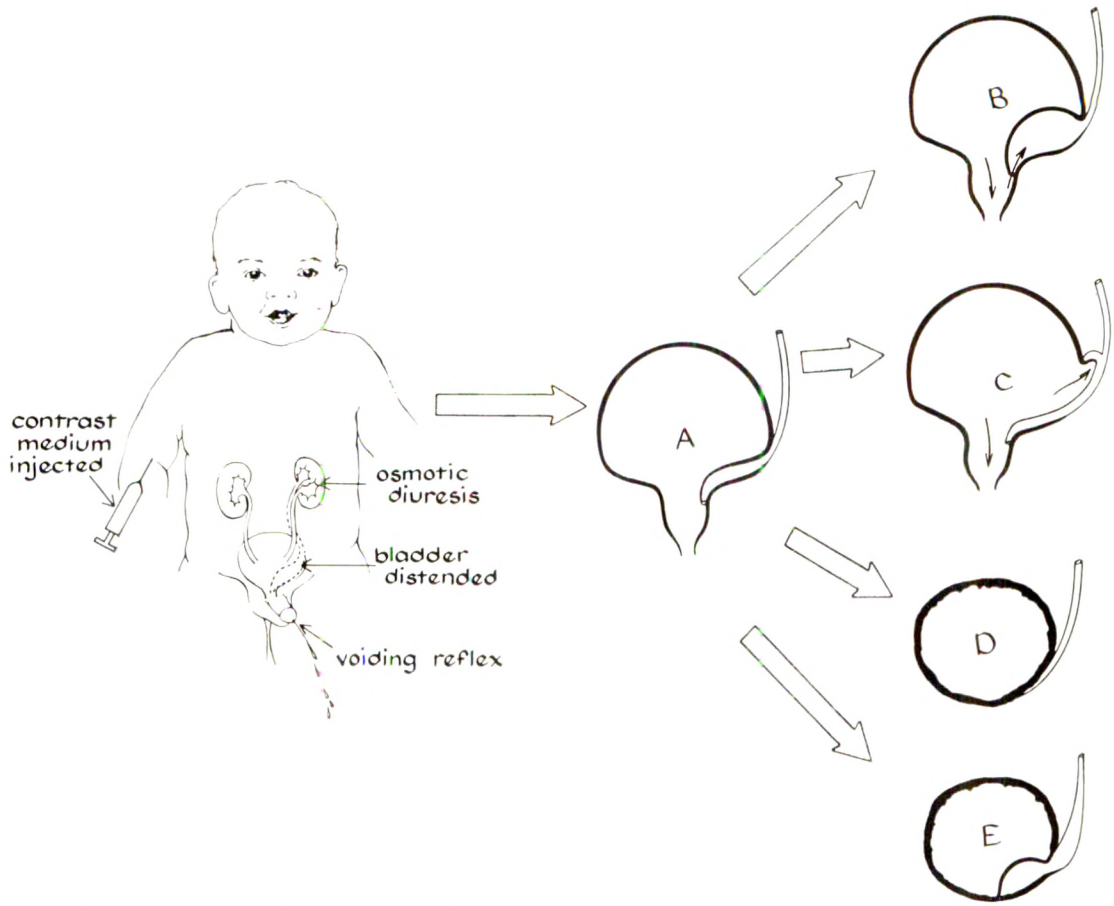


FIG. 10. Dynamic changes in the sphincteric ectopic ureterocele following contrast medium injection. After the injection, osmotic diuresis occurs and the child may void. Diagram *A* shows a bladder and ectopic ureterocele contracting and emptying together. Diagram *B* shows reflux from the urethra into the ectopic ureterocele. Diagram *C* shows how the ectopic ureterocele may collapse and protrude from the bladder lumen, like a diverticulum. Diagram *D* shows how an empty ureterocele may no longer be visible. Diagram *E* shows an ectopic ureterocele refilled from above by peristalsis of a dilated ureter. All these phenomena may also occur during voiding cystourethrography.

have ectopic ureteroceles present with recurrent urinary tract infections.[1,7,17,18,21] Furthermore, the collecting system associated with an ectopic ureterocele frequently fills with pus.

IV. ROENTGENOGRAPHIC FEATURES

While we feel that plain abdominal roentgenograms can be useful, the most valuable studies for revealing ectopic ureteroceles are an excretory urogram followed by a voiding cystourethrogram. On the preliminary study, the affected renal outline usually appears large.[11] Rarely, calculi will form in the ectopic ureterocele.[12]

A. EXCRETORY UROGRAPHY

A typical case will reveal two striking features:[1,7] (1) a mass in the upper pole of the affected kidney; and (2) a large radiolucent defect in the bladder which exhibits the features of a mural mass lesion.

(1) *The Mass in the Upper Pole of the Kidney*. The mass is composed of hydronephrotic calyces plus cystic renal parenchyma of the upper pole. During the nephrogram phase of the urogram, the thinned renal parenchyma may appear as a thin, opaque rim surrounding relatively lucent hydronephrotic calyces.

The thickness of the renal parenchyma in the upper pole does not usually reflect the amount of functioning renal tissue present, because the parenchyma is usually cystic. If gross hydronephrosis exists, the renal parenchyma will be scarcely visible.

Normally, the renal length divided by the distance from the upper pole to the uppermost contrast medium filled calyx is more than 3.3 to 1. A ratio of less than 2.6 to 1 suggests a duplication with a nonexcreting upper pole.[13]

The mass in the upper pole forces the lower pole downwards and laterally (the "wilting flower" sign). Normally, the distance between the upper minor calyx and a line joining the medial border of the nearest spinal pedicles measures about the same on both sides. The ratio between the 2 sides is normally less than 1.5 to 1.[13] A ratio over 1.5 to 1 suggests a duplication with a nonexcreting upper pole.[13]

Usually, the upper calyceal infundibulum of the lower pole in the duplex kidney is shorter and narrower than the infundibula of the lower calyces. If the infundibulum of the apparent upper pole calyx seems disproportionately short,[13,21] the radiologist should suspect a duplication with a nonexcreting upper pole.

Frequently, the upper pole mass will displace the opacified renal pelvis downwards and laterally, and will flatten its upper convex surface.[13,21] The renal pelvis appears more vertical than usual. Its draining ureter will move into a more lateral position, running obliquely, to reach a more normal position at the L_4–5 level.[21]

The upper pole mass should be differentiated from other renal masses, especially nephroblastoma, and from adrenal masses such as neuroblastoma or hematoma.[1] The radiologist can differentiate between ectopic ureteroceles and renal or adrenal masses by looking for signs of duplication or a filling defect in the bladder.

(2) *The Filling Defect in the Bladder.* The radiolucent mass in the bladder represents the ectopic ureterocele. Because it is filled with nonopacified urine, it contrasts with the opacified urine in the bladder. Large ectopic ureteroceles are usually sphincteric and produce a large central, circular lucency in the bladder which occupies most of the bladder lumen.[18] A thin rim of contrast medium surrounds the central lucency.

Intermediate-sized ectopic ureteroceles can be stenotic or sphincteric.[18] They are eccentrically located and lie on the same side of the bladder as the kidney from which they drain. Sphincteric ectopic ureteroceles usually have an oval shape. A line drawn through the long axis of the oval will point to the affected kidney.[7]

Bilateral ectopic ureteroceles (10 per cent of cases) obstruct the bladder outlet more frequently than unilateral ureteroceles. Usually, they will cause a bilobed filling defect in the bladder.

The radiologist should be wary of the fact that several things resemble the intravesical filling defect of ectopic ureteroceles. Gas in the sigmoid colon or rectum can be troublesome in this way. Steep oblique or lateral views will prove that the gas lies posterior to the bladder. In addition, rhabdomyosarcoma of the bladder causes multiple irregular filling defects. Rarely, rhabdomyosarcoma of the bladder or prostate can produce a single smooth filling defect which will appear identical to an ectopic ureterocele. Cystitis cystica usually results in numerous lucencies in contrast to a single or bilobed defect. A Foley catheter in the bladder superficially looks like an ectopic ureterocele. However, the remainder of the Foley catheter should be visible on the roentgenogram. If in doubt, the radiologist should inspect the patient. Blood clots, since they lie in the lumen of the bladder, are surrounded completely in all projections

by contrast medium and thus may be distinguished from ureteroceles.

(3) *Changes in the Rest of the Urinary Tract.* Acute bladder outlet obstruction and hydronephrosis of either or both kidneys can be appreciated roentgenologically. Acute bladder outlet obstruction in the neonate can cause bilateral hydroureteronephrosis,[21] with delayed contrast medium excretion and a large, usually trabeculated bladder. Although the ipsilateral lower pole can appear normal, more often obstruction or reflux will produce a hydroureteronephrosis. The nephrogram may show typical changes of hydronephrosis; delayed urograms will usually outline the hydroureter. Ectopic ureteroceles can exert a profound effect on the contralateral kidney and ureter. If the ectopic ureterocele extends across the trigone, it can obstruct the contralateral ureteral orifice (or orifices). It may also interfere with the antireflux mechanisms of the opposite side. Either obstruction, reflux or both may cause hydroureteronephrosis on the contralateral side.[18,21]

The following case was chosen because it is a typical example of ectopic ureterocele. It demonstrates classic roentgenologic findings and will serve as a basis for comparison with more unusual cases.

CASE 1. A normal, fullterm female infant had recurrent *Proteus mirabilis* urinary tract infections. We performed excretory urography at the age of 5 months. The 3 minute roentgenogram

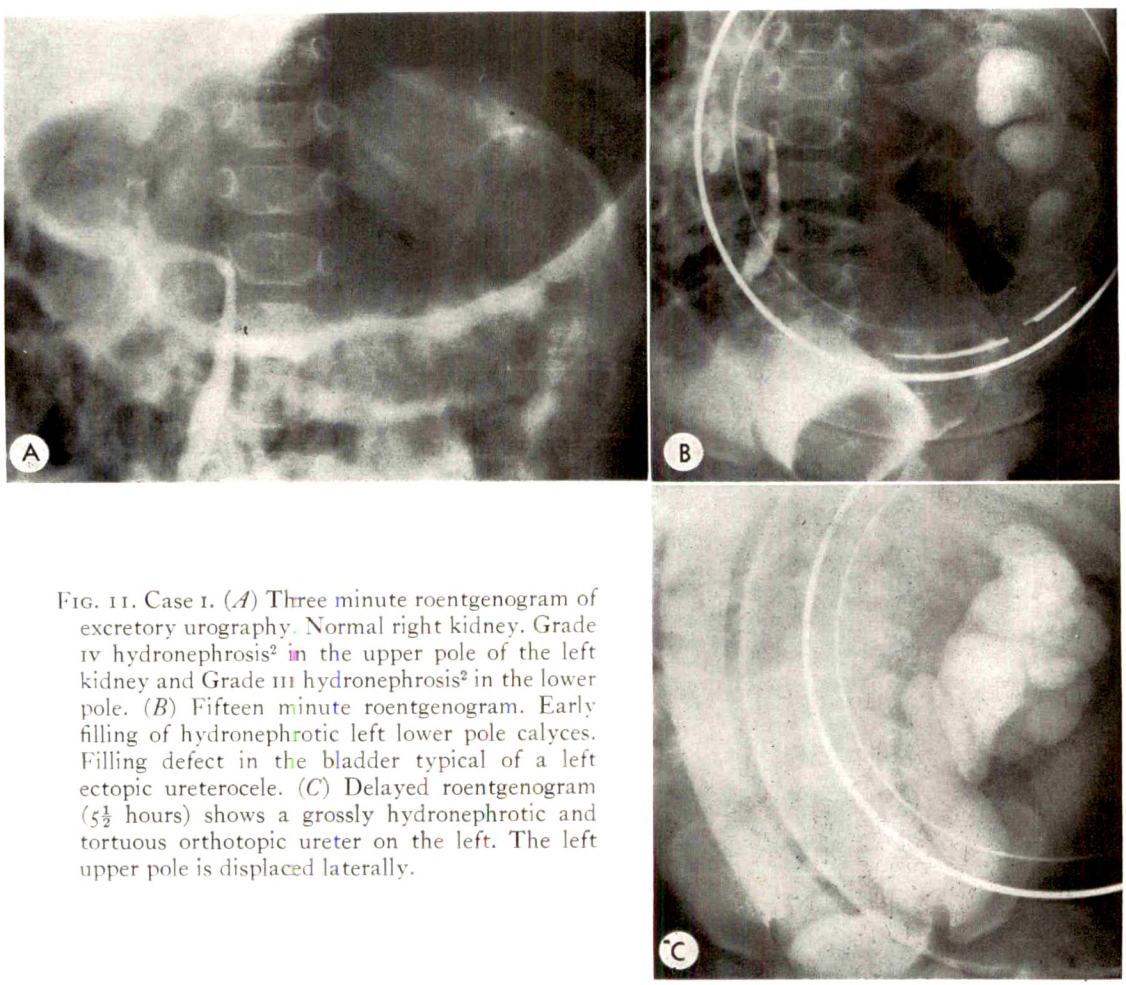


FIG. 11. Case 1. (*A*) Three minute roentgenogram of excretory urography. Normal right kidney. Grade IV hydronephrosis[2] in the upper pole of the left kidney and Grade III hydronephrosis[2] in the lower pole. (*B*) Fifteen minute roentgenogram. Early filling of hydronephrotic left lower pole calyces. Filling defect in the bladder typical of a left ectopic ureterocele. (*C*) Delayed roentgenogram ($5\frac{1}{2}$ hours) shows a grossly hydronephrotic and tortuous orthotopic ureter on the left. The left upper pole is displaced laterally.

showed a normal right side (Fig. 11*A*). On the left, the nephrogram revealed a distinct difference between the upper and lower poles. A faint nephrogram appeared in the left upper pole which exhibited Grade IV hydronephrosis.[2] The parenchyma of the left upper pole measured 1–4 mm. in width. In the lower pole, a rim of parenchymal tissue, 4–6 mm. thick, opacified and exhibited the "Dunbar crescent sign"[5] (Grade III hydronephrosis[2]). Predictably,[2] the lower pole excreted contrast medium into its calyces before the upper pole (Fig. 11*B*). A large, lucent, oval filling defect which displayed typical features of a mural lesion protruded into the bladder lumen. The long axis of the filling defect pointed upward to the left, indicating a diagnosis of ectopic ureterocele arising from the upper pole of the left kidney. A delayed roentgenogram taken at 5½ hours showed gross hydroureteronephrosis and tortuosity of the orthotopic ureter (Fig. 11*C*). The upper pole mass displaced the lower pole laterally thus producing a left-to-right upper pole calyx to pedicle ratio of 2:1. Dilute contrast medium faintly outlined the hugely dilated ureter arising from the upper pole. In this instance the ectopic ureterocele did contain some contrast-laden urine. The upper pole could have excreted the opaque medium which filled the ectopic ureter and ureterocele; opacified urine, on the other hand, could have entered the ureterocele as a result of reflux during voiding.

Cystoscopy revealed that the orifice of the ureter bearing the ectopic ureterocele lay in the midurethra—a sphincteric type. At surgery, the left upper pole and its ureter were excised. Pus filled the ureter and ureterocele, while chronic pyelonephritis had scarred the upper pole. Parenchymal biopsy of the lower pole revealed back pressure atrophy. Postoperative split function studies showed that the remaining lower pole was responsible for 25 per cent of the total renal function.

Comment. This case thus describes typical features of a sphincteric ectopic ureterocele: (1) an oval filling defect in the bladder pointing to the affected side; (2) obstruction of the orthotopic ureter; (3) a mass in the upper pole which displaces the lower pole downwards and laterally.

Factors Affecting Diagnosis: Some Unusual Presentations

Previous reports[15,22] have described misdiagnoses of the disease on the wrong side. In those cases, hydroureteronephrosis was always present bilaterally, so that physicians were uncertain which side had the lesion. We have encountered, however, a unique case in which there was no hydronephrosis of the lower pole of the affected side, while the opposite kidney showed gross hydronephrosis. At first glance, the radiologist might have diagnosed a lesion on the wrong side. Features of duplication with a nonexcreting upper pole on the affected side led to the correct diagnosis.

Case II. A pediatrician palpated a mass in the left flank of an otherwise normal 4 day old male infant. Urinalysis was normal and culture sterile. An excretory urogram demonstrated bilateral duplication, with a nonexcreting upper pole on the left and hydroureteronephrosis of both ureters on the right (Fig. 12). The duplicated right ureters crossed near the ureterovesical junction; thus the ectopic ureter entered the bladder distal to the orthotopic ureter. A large, central, circular, lucent defect, rimmed by contrast medium, filled the bladder.

A left upper pole heminephrectomy was performed. At surgery, a hugely distended left upper pole was found, which transilluminated. Histologically, the upper pole showed cystic dysplasia. The ureteral diameter was huge, 0.7



Fig. 12. Case II. Excretory urogram demonstrates a large, left sphincteric ectopic ureterocele which has obstructed the duplicated right ureters, but has spared the left orthotopic ureter.

cm. The ureter draining the lower pole of the affected side, however, was normal.

Comment. This unique case demonstrates a left sphincteric ectopic ureterocele which obstructed duplicated right ureters, but spared the ureter which drained the lower pole on the affected side.

We have found that if the radiologist does not recognize an ectopic ureterocele and, as a result, surgery is not performed, the upper pole may undergo back pressure atrophy and the ureterocele may shrink as the child grows older. The same thing occurs when a ureterocele ruptures spontaneously, but the ureterocele will then permit free vesicoureteral reflux.[13]

The following unique case documents bilateral back pressure atrophy of the upper poles, due to previously unrecognized bilateral ectopic ureteroceles. The case will show that the radiologist must examine the area of the trigone carefully for a small, lucent mural defect to find a small ectopic ureterocele. Additionally, the radiologist should pay particular attention to any alteration of the course of the ureters, which may indicate duplication.

CASE III. This 1 year old girl first developed recurrent urinary tract infections in the neonatal period. An excretory urogram performed elsewhere at that time disclosed faint excretion and mild bilateral hydronephrosis. In retrospect, careful examination of the urogram revealed bilateral masses in the upper poles which displaced the lower poles downwards and laterally. A voiding cystourethrogram showed bilateral reflux into the lower poles and bilateral hydroureteronephrosis (Fig. 13*A*). Bilateral upper pole masses displaced both lower poles downwards and laterally. An apparent bladder diverticulum projected outside the bladder lumen on the left. The radiologist diagnosed bilateral vesicoureteral reflux due to infection. The child was treated conservatively, but continued to have recurrent episodes of urinary tract infection.

We obtained a second excretory urogram and voiding cystourethrogram at the age of 1 year. The excretory urogram showed calyceal clubbing, parenchymal atrophy, and moderate hydroureter bilaterally (Fig. 13*B*). Examination of the renal outlines did not, however, reveal any evidence of enlargement of the upper poles. The right ureter ran obliquely downwards to the L_5 level, thus suggesting the presence of a duplication on this side. At cystourethrography, using only a small quantity of contrast medium, we noted a small filling defect in the region of the trigone on the right (Fig. 13*C*). As the bladder filled, marked reflux occurred into the left ureter. This ureter had an abnormal oblique course, while the left renal pelvis appeared more vertical and laterally situated than usual, thus pointing to the presence of a duplication on this side (Fig. 13*D*). We diagnosed bilateral duplication and an ectopic ureterocele on the right.

At surgery, bilateral duplication with small bilateral ectopic ureteroceles was found. The upper poles appeared atrophic. Injection of contrast medium through ureteral catheters in the ectopic ureters showed grossly irregular ureters and no evidence of upper pole calyces (Fig. 13*E*). Both upper pole ureters were ligated.

Dynamic events occurring during roentgenography may influence what the radiologist sees or does not see on the roentgenograms. For example, intravenous injection of contrast medium stimulates osmotic diuresis, so that the bladder fills rapidly with contrast medium laden urine (Fig. 10*A*). Distention of the bladder then stimulates reflex voiding in infants (Fig. 10*B*). If the ectopic ureterocele possesses a wide orifice, it will usually permit reflux to occur during voiding. As a result, the ectopic ureterocele fills with contrast medium laden urine of the same radiopacity as urine in the bladder. Since detection of an ectopic ureterocele depends on visualization of a lucent filling defect in the bladder, when the ureterocele fills with contrast medium laden urine, it becomes obscured. Hence, it is a good idea to get a full length 3–5 minute roentgenogram in any infant or child with recurrent urinary tract infections. The following case illustrates how ectopic ureteroceles can virtually disappear from view after voiding.

CASE IV. A female infant was well until the age of 3 months, when she developed fever and

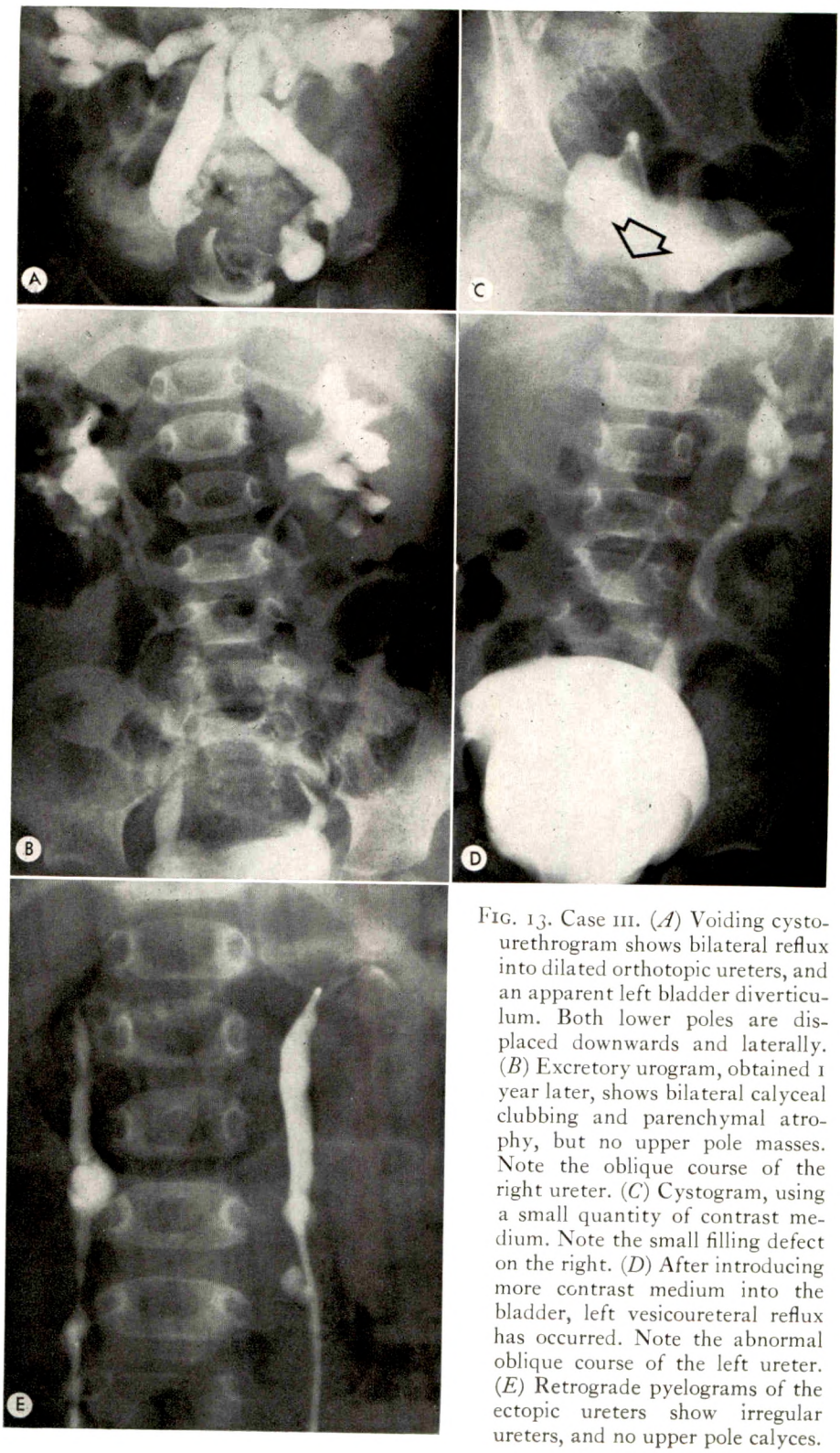


Fig. 13. Case III. (*A*) Voiding cystourethrogram shows bilateral reflux into dilated orthotopic ureters, and an apparent left bladder diverticulum. Both lower poles are displaced downwards and laterally. (*B*) Excretory urogram, obtained 1 year later, shows bilateral calyceal clubbing and parenchymal atrophy, but no upper pole masses. Note the oblique course of the right ureter. (*C*) Cystogram, using a small quantity of contrast medium. Note the small filling defect on the right. (*D*) After introducing more contrast medium into the bladder, left vesicoureteral reflux has occurred. Note the abnormal oblique course of the left ureter. (*E*) Retrograde pyelograms of the ectopic ureters show irregular ureters, and no upper pole calyces.

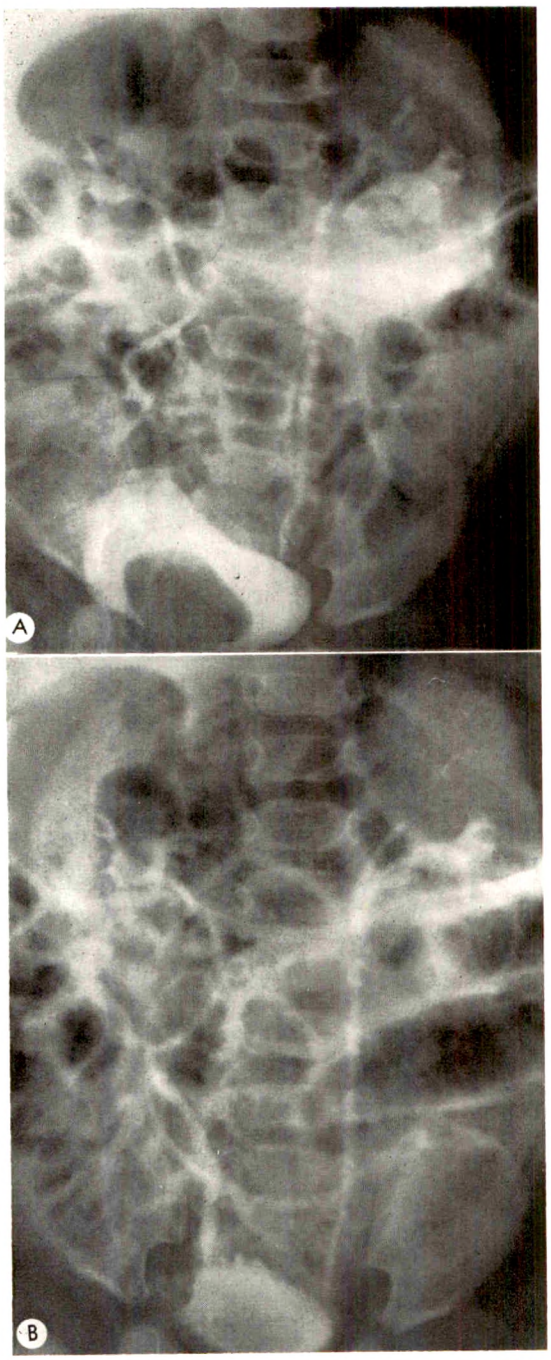


FIG. 14. Case IV. (*A*) Excretory urogram. A 3 minute roentgenogram of pyelography demonstrates bilateral duplication and a typical right sphincteric ectopic ureterocele. (*B*) Six minute roentgenogram. The infant voided shortly before this study was obtained. The ectopic ureterocele has filled with contrast medium refluxed from the bladder and is scarcely visible.

diarrhea. Urine culture revealed an *Escherichia coli* infection. During excretory urography a 3 minute study demonstrated bilateral duplication and a nonexcreting upper pole on the right, and buckling of the right ureter at the points where the orthotopic ureter crossed it (Fig. 14*A*). An oval filling defect projecting into the bladder lumen exhibited the characteristic features of a mural lesion. The long axis of the oval pointed upwards to the right, indicating a diagnosis of right ectopic ureterocele. Shortly before we obtained a second full-length roentgenogram at 6 minutes, the infant voided. This roentgenogram demonstrated that the bladder had partially emptied; the ectopic ureterocele had filled with contrast medium (Fig. 14*B*). We could detect the ectopic ureterocele only because we knew it was there; otherwise we could easily have mistaken it for bowel gas.

If an ectopic ureterocele prolapses into the urethra, especially if the prolapse persists after voiding, most of the ureterocele will lie in the urethra, while the "top of the iceberg" will project into the bladder.

A tense ectopic ureterocele will project further into the bladder lumen, whereas the intravesical pressure will flatten a flaccid ectopic ureterocele. The roentgenologic diagnosis will then be difficult if not impossible. Tension in an ectopic ureterocele is related to 3 factors: (1) the size of the orifice in the ureterocele (ectopic ureteroceles with markedly stenotic orifices are usually tense);[18] (2) the excretory ability of the upper pole; and (3) the amount of drainage and reflux during voiding.

If the patient lies supine, a small amount of contrast medium initially collects posteriorly in the bladder. This initially small amount of contrast medium forms a shallow puddle, which will not completely cover the ectopic ureterocele, which also lies posteriorly. Thus, the ectopic ureterocele will be visible on a roentgenogram. As more contrast medium enters the bladder, the ureterocele will be covered up, and the ureterocele will not be seen. Finally, a distended bladder can compress and empty an ectopic ureterocele, especially if the latter is flaccid. We consider this another valid

reason for obtaining an early full length roentgenogram in infants and children during an excretory urogram.

Because the bladder base plate is higher posteriorly than anteriorly, an anteroposterior projection may not show an ectopic ureterocele when the bladder is full. The radiologist can avoid this problem by always taking a steep oblique or lateral view. We have also found that a Chassard-Lapine view may be a useful adjunct to conventional projections.

B. FACTORS AFFECTING DIAGNOSIS IN VOIDING CYSTOURETHROGRAPHY

During catheterization of the bladder for a voiding cystourethrogram in a patient who has an ectopic ureterocele, the catheter will usually enter the bladder. Occasionally, however, the catheter will slip into a sphincteric ectopic ureterocele, especially if the ureterocele possesses a wide orifice. The following case, in which the tip of the catheter popped into a pseudoectopic ureterocele (a mass of coiled, dilated ureter) created a confusing roentgenographic appearance.

Case v. A 7 month old female infant had a history of recurrent urinary tract infections. An excretory urogram disclosed a normal kidney and pelvicalyceal system on the left, but no excretion on the right (Fig. 15*A*). A large, central lucent filling defect occupied most of the bladder lumen. A rim of contrast medium surrounded the filling defect. A 24 hour roentgenogram demonstrated that a right upper pole mass had forced the grossly hydronephrotic lower pole of the duplicated system downwards and laterally (Fig. 15*B*). A faint filling defect still protruded into the bladder lumen. During catheterization for a voiding cystourethrogram, the catheter entered an unusual coiled structure within the bladder, which was connected to a hugely dilated, tortuous ureter (Fig. 15*C*). A Chassard-Lapine view confirmed these findings (Fig. 15*D*). Under general anesthesia, repeat cystography was performed. A needle was inserted into the "ureterocele" demonstrating that it contained a tortuous ureter (Fig. 15*E*). At surgery, a right ectopic pseudoureterocele was found. A right upper pole heminephrectomy and ureterectomy were performed.

If the catheter enters the bladder, rather than the ureterocele, the study serves 3 purposes: (1) it will demonstrate the presence or absence of vesicoureteral reflux into the ureters not carrying the ectopic ureterocele; (2) it will show the presence or absence of reflux into the ectopic ureterocele; (3) it will confirm the presence of an ectopic ureterocele and occasionally demonstrate an ectopic ureterocele not well shown by other methods.

An ectopic ureterocele can produce a lucent filling defect on the cystogram similar to that seen on an intravenous urogram. A voiding cystourethrogram will frequently not depict an ectopic ureterocele as well as an intravenous urogram for the following reasons: (1) dense contrast medium can obscure the ectopic ureterocele (a steep oblique, lateral, or Chassard-Lapine view will avoid this problem); (2) distention of the bladder can flatten the ectopic ureterocele; (3) an ectopic ureterocele can simulate a diverticulum, especially during voiding[6,12,17] (Fig. 10; 13*A*; and 16*C*).

During voiding, a sphincteric ectopic ureterocele or a stenotic ectopic ureterocele which has a mildly stenotic orifice can contract; the bladder and ectopic ureterocele thus empty together.[17] If the ectopic ureterocele disappears during voiding, and either fills or vanishes at the end of voiding, the radiologist can diagnose a sphincteric ectopic ureterocele with confidence.[17] After voiding, a stenotic ectopic ureterocele usually remains as a globular filling defect in the bladder. During or after voiding, the redundant mucosa of an empty ectopic ureterocele can project outside the general contours of the bladder lumen and simulate a bladder diverticulum[6,12,17] (Fig. 10). Beware of an apparent bladder diverticulum in a patient who has a duplication on the same side in an intravenous urogram! The following case demonstrates how an ectopic ureterocele can simulate a bladder diver-

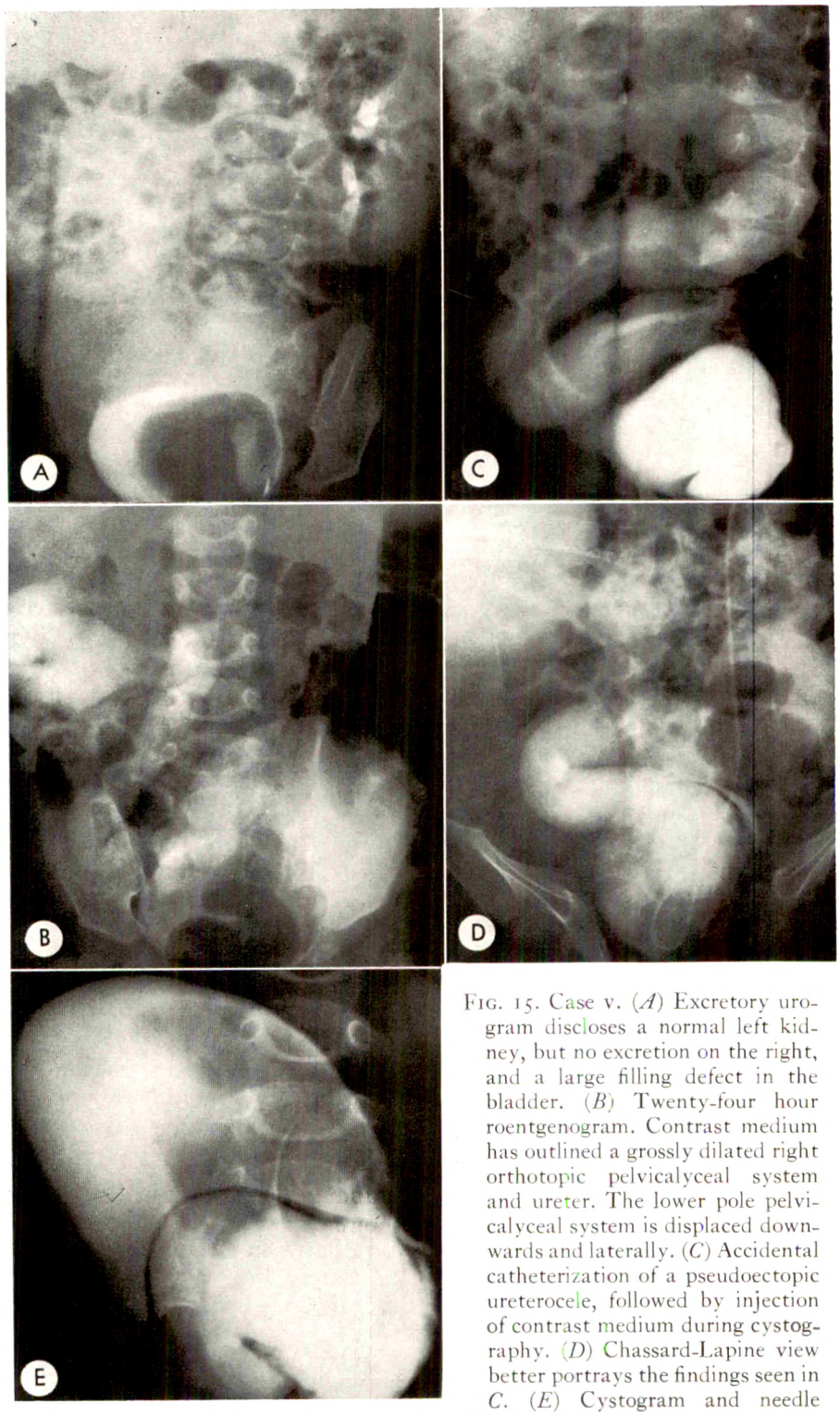


FIG. 15. Case v. (*A*) Excretory urogram discloses a normal left kidney, but no excretion on the right, and a large filling defect in the bladder. (*B*) Twenty-four hour roentgenogram. Contrast medium has outlined a grossly dilated right orthotopic pelvicalyceal system and ureter. The lower pole pelvicalyceal system is displaced downwards and laterally. (*C*) Accidental catheterization of a pseudoectopic ureterocele, followed by injection of contrast medium during cystography. (*D*) Chassard-Lapine view better portrays the findings seen in *C*. (*E*) Cystogram and needle puncture of the pseudoectopic ureterocele performed during surgery.

ticulum during voiding. Additionally, it emphasizes the importance of having some method of rapidly recording events during a voiding cystourethrogram. The case also aroused our interest because a ureteropelvic junction obstruction existed in the lower pole pelvis on the same side. A ureteropelvic junction obstruction occurs more frequently in the lower pelvis of a duplicated kidney,[21] but it has not been previously reported in a case of ectopic ureterocele.

Case VI. A 4 month old male infant, had a mass discovered in the right upper quadrant. Urinalysis was normal, urine cultures sterile.

On an excretory urogram the right kidney failed to visualize (Fig. 16*A*), but a left hydroureter was present. A large central lucent defect, rimmed by contrast medium, filled the bladder. We diagnosed a right ectopic ureterocele.

A voiding cystourethrogram demonstrated a large, lucent, posterior filling defect which exhibited the characteristic features of a mural lesion (Fig. 16*B*). During voiding it flattened (Fig. 16*C*), and finally everted, resembling a diverticulum (Fig. 16*D*).

Retrograde pyelography conducted on the orthotopic ureter revealed a high insertion of the ureter into the pelvis, and gross hydronephrosis, diagnostic of ureteropelvic junction obstruction (Fig. 13*E*). A limited surgical procedure was performed; the ectopic ureterocele was excised and the bladder wall was reconstructed. Fifteen months later, the right kidney and ureter were removed. Severe kinking of the ureteropelvic junction of the lower pole was found; the right kidney was markedly atrophic.

Sometimes, urethral changes constitute the only evidence of an ectopic ureterocele. The urethral changes which the radiologist will usually encounter include: (1) an abnormally wide bladder neck; (2) a lucent posterior filling defect in the posterior urethra, sometimes very faint; (3) prolapse of ectopic ureteroceles (40 per cent of cases) can completely or partially obstruct the urethra. This phenomenon is best depicted on cine or 6 per second 70 mm. films; (4) a cecoureterocele can fill during voiding. As a result of this distention, it can obstruct flow in the urethra.[19]

In boys, several conditions can cause a dilated posterior urethra: (1) posterior urethral valves. When valves are present, the urethral dilatation characteristically terminates in a bullet-nosed distal end. The roentgenograms will also usually depict the valve leaflets; (2) a dilated posterior urethra usually occurs in boys whose abdominal muscles are absent, but the examiner will usually find other physical signs of the syndrome; (3) a normal variation; (4) incompetent internal sphincter and competent external sphincter in patients who have a neurogenic bladder disorder.

SUMMARY

1. A classification of ectopic ureteroceles, modified from Stephens' classification by the addition of 1 category is presented.

2. Stephens' theory of the embryology of ectopic ureteroceles is the only currently adequate theory.

3. Ectopic ureteroceles seen on roentgenograms may display features other than those commonly associated with them. They are consequently difficult to diagnose.

4. The roentgenologic appearance of ectopic ureteroceles may change at any time during the examination because of unpredictable dynamic factors.

5. The excretory urogram is the most useful examination for diagnosing ectopic ureteroceles. Voiding cystourethrograms, however, should also be done to illustrate pathology not seen on the excretory urogram.

6. A full-length roentgenogram should be taken 3–5 minutes after contrast medium injection, before osmotic diuresis has caused the patient to void.

7. Whenever an ectopic ureterocele cannot be ruled out, but cannot be seen on conventional roentgenograms, steep oblique or lateral projections of the bladder should be obtained. Occasionally, a Chassard-Lapine view will also be helpful.

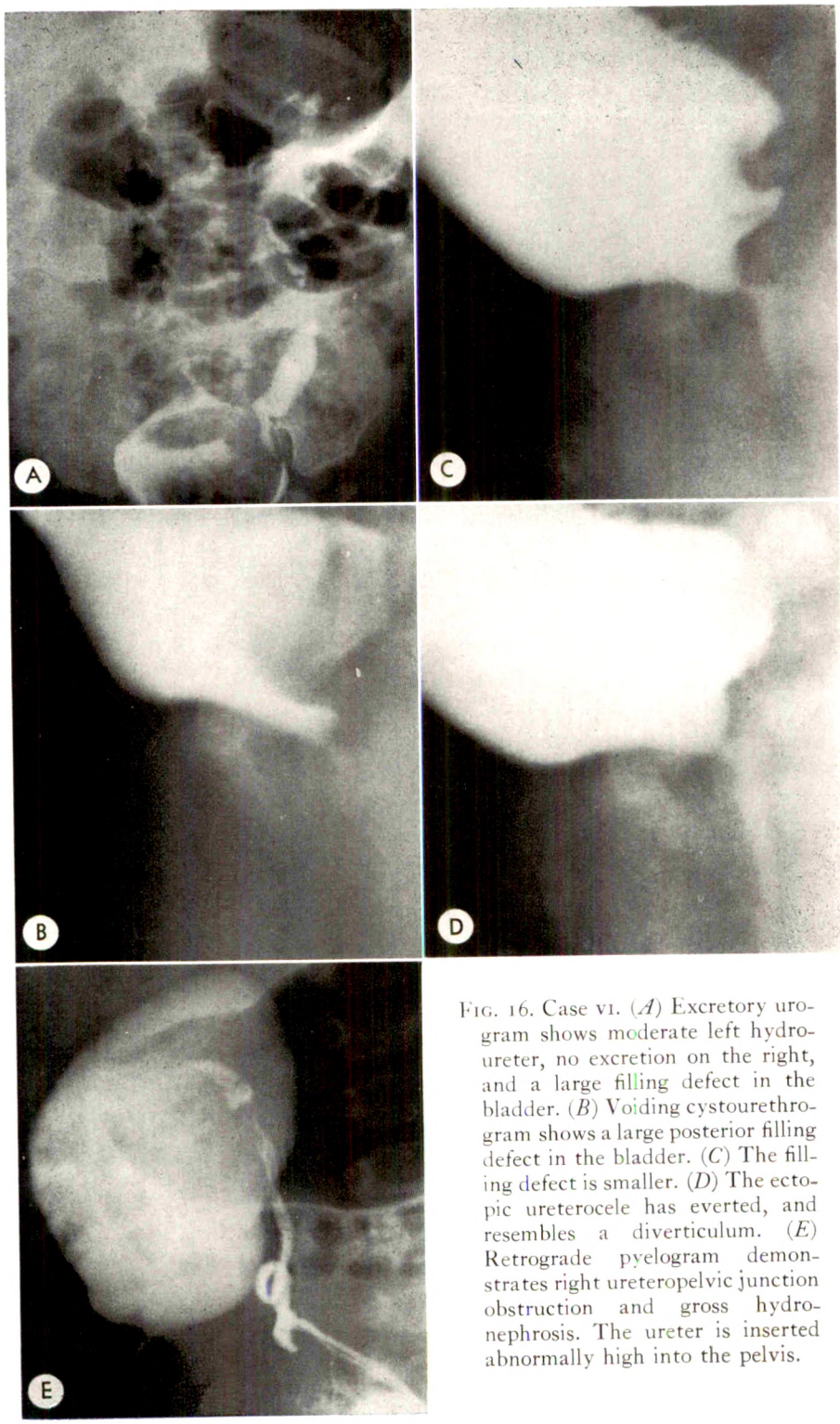


FIG. 16. Case VI. (*A*) Excretory urogram shows moderate left hydroureter, no excretion on the right, and a large filling defect in the bladder. (*B*) Voiding cystourethrogram shows a large posterior filling defect in the bladder. (*C*) The filling defect is smaller. (*D*) The ectopic ureterocele has everted, and resembles a diverticulum. (*E*) Retrograde pyelogram demonstrates right ureteropelvic junction obstruction and gross hydronephrosis. The ureter is inserted abnormally high into the pelvis.

8. When no direct evidence for ectopic ureteroceles exists, the radiologist should ascertain whether or not any indirect evidence exists: change in the axis of the kidney, for example, or in the course of the ureter, or an increase in thickness in the renal parenchyma in the upper pole.

9. The radiologist should search for evidence of ectopic ureteroceles whenever there is, in females *or* males, a history of recurrent urinary tract infection.

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REFERENCES

1. Berdon, W. E., Baker, D. H., Becker, J. A., and Uson, A. C. Ectopic ureterocele. *Radiol. Clin. North America*, 1968, *6*, 205–214.
2. Berdon, W. E., Levitt, S. B., Baker, D. H., Becker, J. A., and Uson, A. C. Hydronephrosis in infants and children: value of high dosage excretory urography in predicting renal salvageability. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *109*, 380–389.
3. Chwalle, R. Process of formation of cystic dilatations of vesical end of ureter and of diverticula at ureteral ostium. *Urol. & Cutan. Rev.*, 1927, *31*, 499–504.
4. Currarino, G., and Allen, T. D. Congenital anomalies of the upper urinary tracts. *Progr. Pediat. Radiol.*, 1970, *3*, 211–217.
5. Dunbar, J. S., and Nogrady, M. B. Calyceal crescent; roentgenographic sign of obstructive hydronephrosis. Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1970, *110*, 520–528.
6. Emmett, J. L., and Witten, D. M. Clinical urography. In: An Atlas and Textbook of Roentgenologic Diagnosis. Volume 3. W. B. Saunders Company, Philadelphia, 1971, pp. 1435–1530.
7. Ericsson, N. O. Ectopic ureterocele in infants and children: clinical study. *Acta chir. scandinav.*, 1954, Suppl. 197.
8. Frazer, J. E. Terminal part of Wolffian duct. *J. Anat.*, 1935, *69*, 455–468.
9. Gyllensten, L. Contributions to embryology of urinary bladder. Part 1: development of definitive relations between openings of Wolffian ducts and ureters. *Acta anat.*, 1949, *7*, 305–344.
10. Hamilton, W. J., Boyd, J. D., and Mossman, H. W. Human embryology. In: Prenatal Development of Form and Function. Williams & Wilkins Company, Baltimore, 1962, pp. 278.
11. Hartman, G. W., and Hodson, C. J. Duplex kidney and related anomalies. *Clin. Radiol.*, 1969, *20*, 387–400.
12. Kjellberg, S. R., Ericsson, N. O., and Rudhe, U. The Lower Urinary Tract in Childhood: Some Correlated Clinical and Roentgenologic Observations. Year Book Publishers, Inc., Chicago, 1957, pp. 114–146.
13. Lundin, E., and Riggs, W. Upper urinary tract duplication associated with ectopic ureterocele in childhood and infancy. *Acta radiol.* (Diag)., 1968, *7*, 13–24.
14. Meyer, R. Normal and abnormal development of ureter in human embryo: mechanistic consideration. *Anat. Rec.*, 1946, *96*, 355–371.
15. Mogg, R. A. Some observations on ectopic ureter and ureterocele. *J. Urol.*, 1967, *97*, 1003–1012.
16. Osathanondh, V., and Potter, E. L. Pathogenesis of polycystic kidneys: Type 4 due to urethral obstruction. *A.M.A. Arch. Path.*, 1964, *77*, 502–509.
17. Stephens, F. D. Ureterocele in infants and children. *Australian & New Zealand J. Surg.*, 1958, *27*, 288–295.
18. Stephens, F. D. Congenital Malformations of the Rectum, Anus and Genito-Urinary Tracts. E&S Livingstone Ltd., Edinburgh, 1963, pp. 153–195.
19. Stephens, F. D. Caecoureterocele and concepts on embryology and aetiology of ureteroceles. *Australian & New Zealand J. Surg.*, 1971, *40*, 239–248.
20. Thornbury, J. R. Roentgen diagnosis of ureterocele in children, Am. J. Roentgenol., Rad. Therapy & Nuclear Med., 1963, *90*, 15–25.
21. Williams, D. I. Paediatric Urology. Appleton-Century-Crofts, Inc., New York, 1968, pp. 195–212.
22. Williams, D. I., and Woodard, J. R. Problems in management of ectopic ureteroceles. *J. Urol.*, 1964, *92*, 635–652.

URETERAL TUMORS*

THE RADIOLOGIC EVALUATION OF A DIFFERENTIAL DIAGNOSIS "THROW-IN"

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THERE is some evidence that primary ureteral tumors are increasing in incidence along with other genitourinary tumors such as bladder carcinoma.[6,38] There has been little written in the radiologic literature about the plain film roentgenographic changes of ureteral tumors despite this suggested increase and reports showing benign ureteral tumors occurring in younger persons, even teenagers.[9]

In patients with hematuria and whose intravenous pyelograms (IVP) show unexplained unilateral hydronephrosis or nonfunction, ureteral tumors are usually "thrown in" at the end of a differential diagnosis preceded by nonapparent or nonopaque calculi, strictures and extrinsic lesions.

In reviewing most major reported series on primary ureteral tumors and our own previously unreported series of 21, we have observed 3 categories of IVP changes which suggest the diagnosis prior to retrograde pyelography or surgery. We feel that these are of help in establishing the diagnosis prior to additional roentgenologic or surgical evaluation.

BACKGROUND

The first description of a tumor involving the ureter was in 1841 by Rayer.[28] The patient had abdominal pain and hematuria. Lebert[20] in 1861 reported the first benign tumor involving the ureter. In 1878 Wising and Blix[36] reported a second malignant tumor involving the ureter. These first 3 reports were in patients who had several genitourinary tract tumors in which the ureteral tumor was only a part.

Davy[10] in 1884 was the first to demonstrate a tumor localized only to the ureter in a patient with pain, hematuria and a left flank mass. Albarran[2] in 1900 made the first preoperative diagnosis of a ureteral tumor.

By 1922 Aschner[3] had found 47 cases. In 1943 Scott[32] reported 59 cases and reviewed 183 cases previously reported in the literature. Mortensen and Murphy[24] in 1950 commented on 322 cases of which 245 were malignant and 77 benign. Abeshouse[1] in 1956 collected 592 cases (454 malignant and 138 benign). Since then, others have reported their experience and compared it to that of these earlier groups. References are made to these authors where indicated later in the discussion.

PATHOLOGY

Primary ureteral tumors are classified in 2 major groups—epithelial and mesodermal.

The benign epithelial tumors include:

1. Papillomas
2. Polyps
3. Adenomas

Malignant epithelial tumors include:

1. Transitional cell carcinoma
2. Squamous cell carcinoma
3. Adenocarcinoma

Fibromas, hemangiomas, myomas and lymphangiomas are benign mesodermal tumors. Sarcomas and angiosarcomas are the malignant mesodermal tumors.

INCIDENCE

Males outnumber females 2 to 1.[1,5,6,15,21–23] The reported age ranges from 24 to 93 years with a mean age in the 7th decade.[1,6,14,15] However, a recent article[9] reports a case of benign ureteral polyp in an 11 year old boy. Multiple series show 75 to 80 per cent of the lesions being malignant.[1,5,15,22,23]

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TABLE I

SUMMARY OF 21 NEW CASES

Case	Age, Race, Sex	Symptoms and History	Treatment	Pathology	Follow-Up
1	73 WF	Painless hematuria	Right ureteronephrectomy	Carcinoma	Recurrence 4 months postoperative, radium implant
2	56 WF	Recurrent bladder tumors, hematuria	Left ureteronephrectomy	Transitional cell carcinoma	Alive and well 3 years postoperative
3	63 WM	Pain, calculus of left ureterovesical junction	Left ureteronephrectomy	Transitional cell carcinoma	Alive and well 9 years postoperative
4	66 WM	Painless, gross hematuria	Right nephroureterectomy	Transitional cell carcinoma	No follow-up
5	69 WM	Painless hematuria	Left ureteronephrectomy	Transitional cell carcinoma	None available
6	59 WM	No urinary tract symptoms	Resection of right ureteral tumor	Papilloma	Alive and well 1½ years
7	77 WM	Pain in left flank	Left ureteronephrectomy	Carcinoma	Death of other cause 3 months postoperative
8	49 WF	Gross and microscopic hematuria	Left ureteronephrectomy	Carcinoma	Alive 5 years postoperative
9	66 WM	Hematuria	Right ureteronephrectomy	Carcinoma	Alive 2 months postoperative, with metastatic lesions
10	69 WM	Painless hematuria	Left ureteronephrectomy	Transitional cell carcinoma	Alive 6 months postoperative with metastatic disease
11	62 WF	Hematuria	Left ureteronephrectomy	Transitional cell carcinoma	Alive 3 months postoperative with recurrent bleeding
12	77 WF	Hematuria, right flank pain, rectal mass	Right ureteronephrectomy, abdominal perineal resection	Carcinoma	Alive and well 12 years postoperative
13	82 WM	None available	Operative confirmation and roentgen therapy	Carcinoma	None available
14	WM	No clinical history	Radiation therapy	Transitional cell carcinoma	Death 4 months postdiagnosis
15	65 WM	No urinary tract symptoms, history of bladder polyp	Right ureteral cystotomy	Transitional cell carcinoma of right ureter, extension to bladder	No follow-up beyond 6 months

TABLE I (*continued*)

Case	Age, Race, Sex	Symptoms and History	Treatment	Pathology	Follow-Up
16	49 WM	No presenting complaint available	Resection of left ureteral tumor with postoperative roentgen therapy	Adenocarcinoma	Death 3 years postoperative
17	66 NM	Intermittent hematuria	Right ureteronephrectomy	Transitional cell carcinoma	Death 10 months postoperative
18	82 WF	No urinary tract symptoms, chronic weight loss	No treatment undertaken	Transitional cell carcinoma	Ureteral carcinoma incidental finding at autopsy
19	50 WM	Hematuria, previous papillary bladder tumor	Right ureteronephrectomy	Transitional cell carcinoma	Death 1 year postoperative
20	65 WM	Pain in left flank	Left ureteronephrectomy	Transitional cell carcinoma	Death 4 months, postoperative radiation therapy
21	67 NF	Hematuria	Left nephroureterectomy	Carcinoma	Postoperative radiation therapy

Between 1 and 2 per cent of genitourinary tumors (kidney, ureter and bladder) are primary ureteral tumors.[6,23,26,30,33]

Autopsy statistics and genitourinary service admission statistics of several large series reveal 14 primary ureteral tumors in 137,000 autopsies and 1 primary ureteral tumor in 3,700 genitourinary admissions.[1,5,6]

Seventy-five per cent of the tumors occur in the lower third of the ureter.[1,5,15,22,23] Some statistics indicate that benign lesions are more likely to occur in the upper portions of the ureter.[7,9,31] Perlmutter *et al.*[27] report 5 synchronous bilateral primary ureteral tumors. Only 1 of our 21 cases had a lesion in the upper part of the ureter.

SUMMARY OF 21 NEW CASES

Table 1 summarizes the information on the 21 previously unreported cases of primary ureteral tumors.

CLINICAL PICTURE

In 8 different series[1,4–6,14,21–23] 70 to 90 per cent of the patients presented with hematuria. Of these, 20 to 35 per cent had associated pain. The hematuria was intermittent and rarely profuse. The duration of hematuria ranged from weeks to months. Macroscopic hematuria was present in most of these patients.

Mass alone due to a hydronephrotic kidney was present in 2 to 5 per cent of the cases; 10 to 20 per cent presented with pain alone. The pain was usually a dull aching pain with a minority of patients having colic.

In our group of 21, 18 good histories were available. There were 11 (61 per cent) patients with hematuria of whom 1 had associated pain. Four (22 per cent) presented with pain alone.

CYSTOSCOPY CHANGES

On cystoscopy, distal ureteral tumors can bulge into the bladder and be seen. Bloody urine may be seen coming from the involved side.

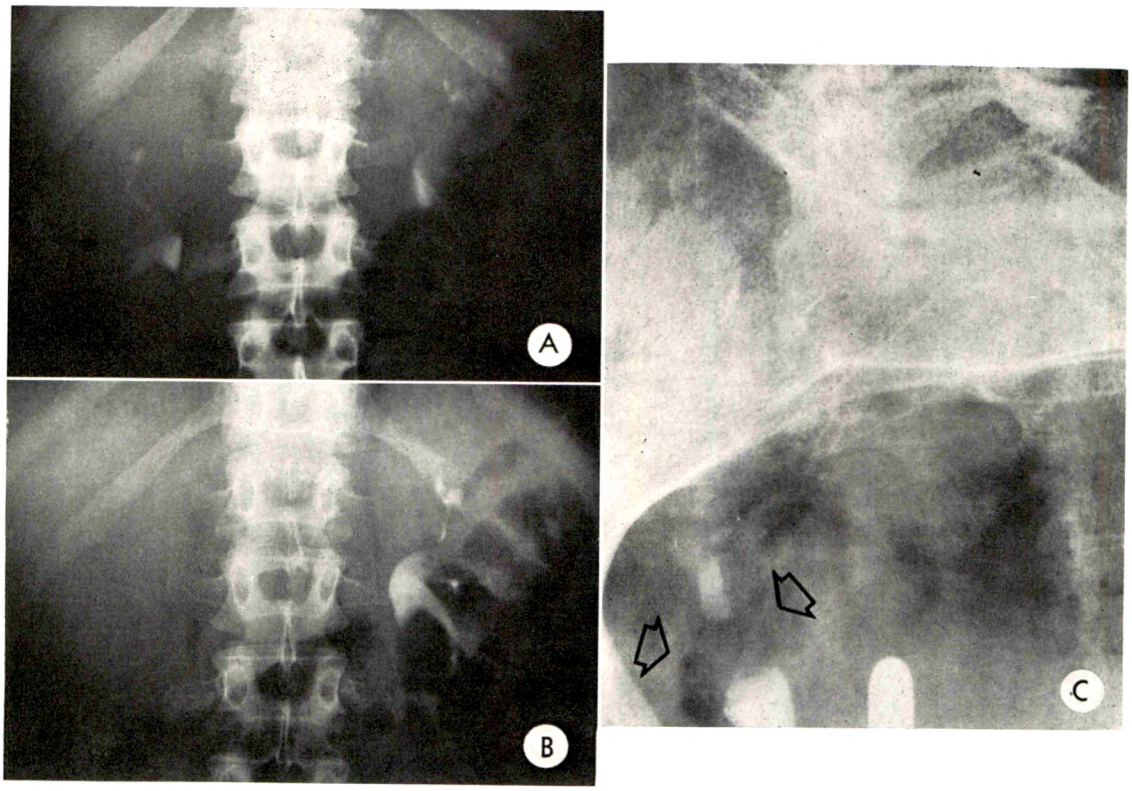


Fig. 1. (*A*) Normal intravenous pyelogram of a 50 year old white male in 1955. (*B*) Intravenous pyelogram 2 years later, shows total unilateral nonvisualization. (*C*) Retrograde pyelogram shows tumor obstructing the lower ureter (open arrowheads).

In 3 reported series[4,15,23] 20 of 93 cases (22 per cent) showed a tumor bulge on cystoscopy. However, Bloom *et al.*[6] saw only 6 per cent in their 100 cases. Only 2 of our 21 cases (9.5 per cent) showed a tumor bulge.

In 2 reported series[4,23] 13 of 76 (17 per cent) had blood coming from one ureter on cystoscopy. Bloom *et al.*[6] reported a 21 per cent incidence. Only 1 of our 21 cases had unilateral bloody urine on cystoscopy.

ROENTGENOGRAPHIC CHANGES

There are 3 categories of IVP changes which are reported by most authors and which we observed in our 21 cases. These are: (1) total unilateral nonvisualization (Fig. 1, *A–C*); (2) unexplained unilateral hydronephrosis (Fig. 2, *A* and *B*; 3, *A* and *B*; and 4, *A–D*); and (3) ureteral defect without hydronephrosis (Fig. 5; 6; and 7, *A* and *B*). Some authors include normal IVP as a fourth category. None of our 21 cases had a normal IVP.

In 6 reported series[4–6,15,22,23] 44 per cent were total unilateral nonvisualization, 33 per cent were unexplained unilateral hydronephrosis, 10 per cent were ureteral defect with no hydronephrosis, and 13 per cent were considered normal.

In our group of 16 available IVPs, 5 of 16 (31 per cent) were total unilateral nonvisualization, 8 of 16 (50 per cent) were unexplained unilateral hydronephrosis, and 3 of 16 (19 per cent) were ureteral defect with no hydronephrosis. None were normal.

In a patient with unexplained hematuria and whose IVP shows apparently normal but only intermittently filled portions of the ureter, early aspects of a ureteral tumor may be overlooked. It is important in these patients to adequately visualize the ureter in its entirety by infusion pyelography or possibly retrograde pyelography.

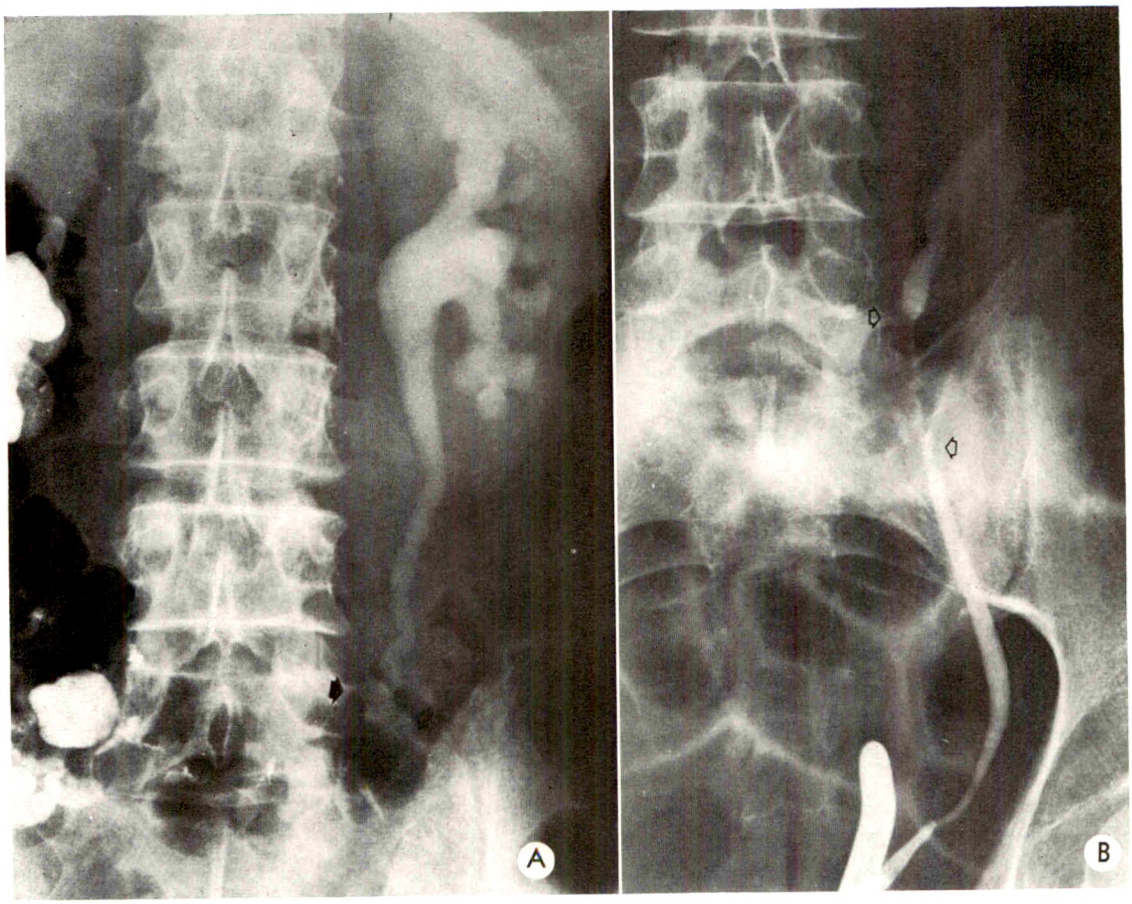


FIG. 2. (*A*) Unexplained unilateral hydronephrosis in a 77 year old white male with hematuria. (*B*) Retrograde pyelogram shows long irregular stricture-like tumor (open arrowheads).

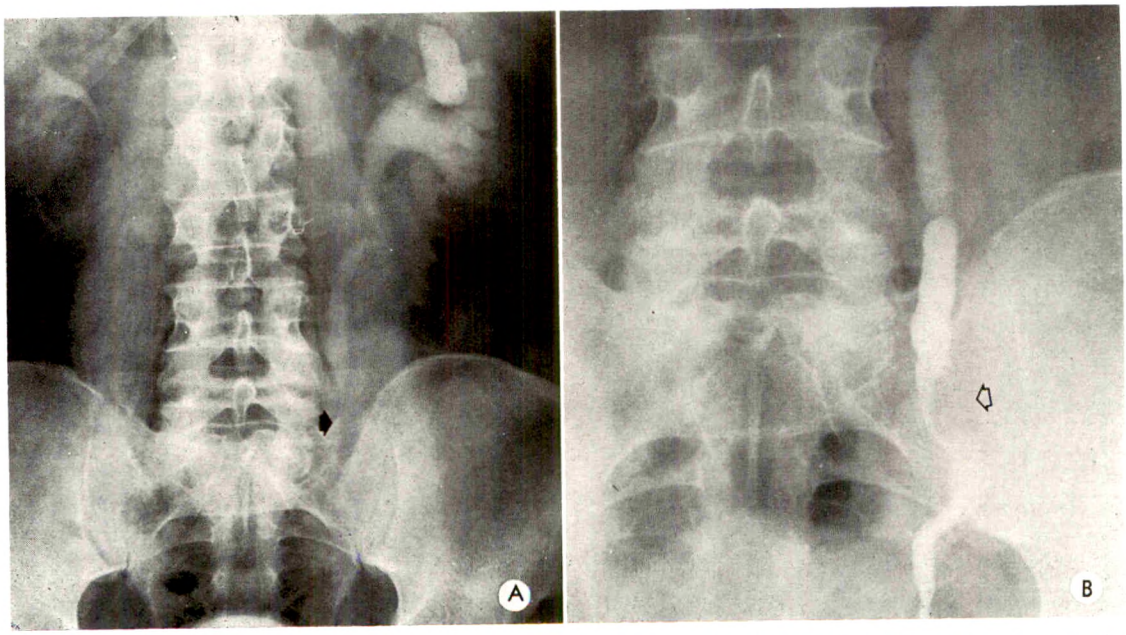


FIG. 3. (*A*) Unexplained unilateral hydronephrosis in a 69 year old white male with painless hematuria. (*B*) Retrograde pyelogram shows dilatation about a filling defect (open arrowhead).

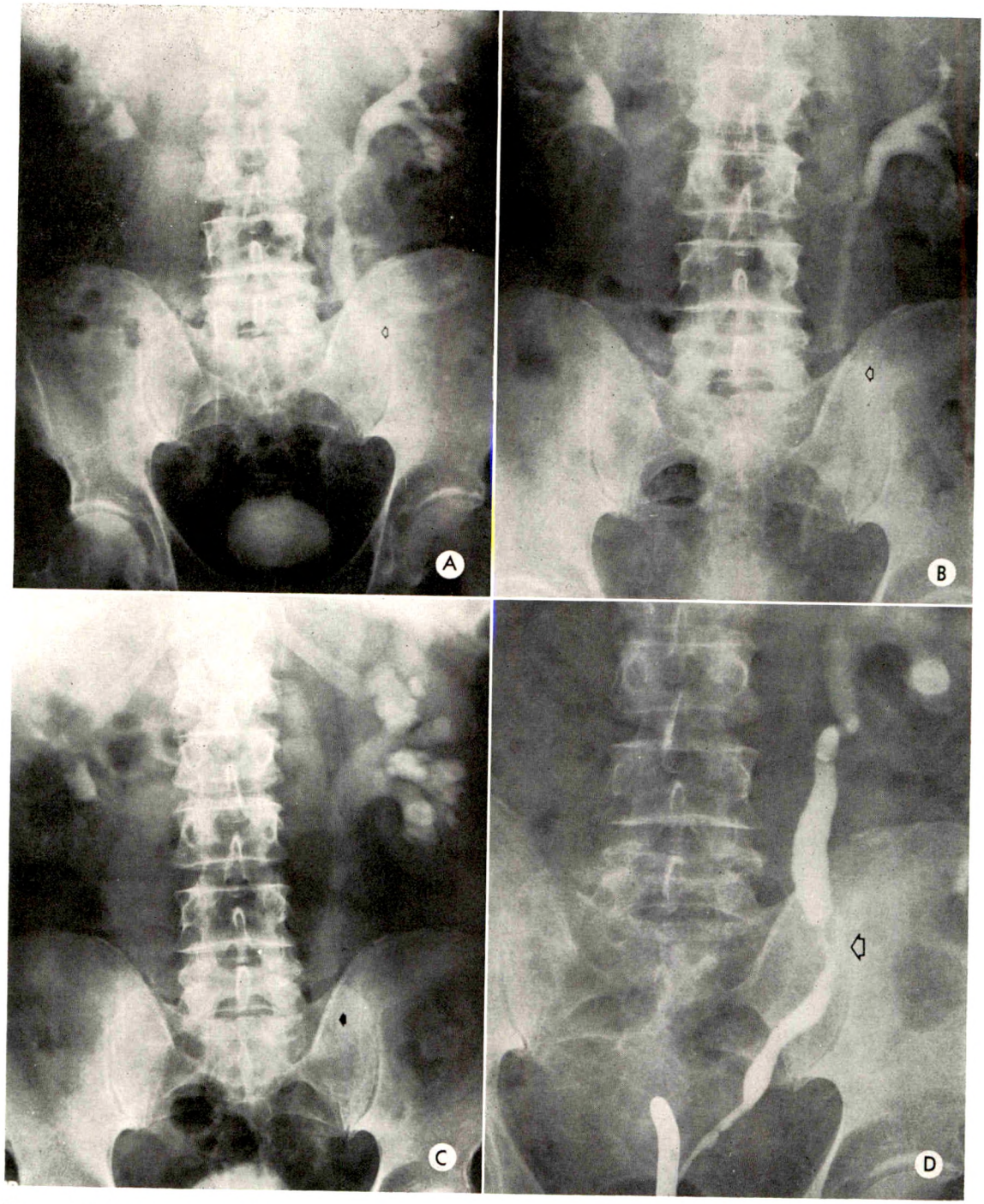


Fig. 4. (*A*) Intravenous pyelogram shows unexplained unilateral hydronephrosis in a 63 year old white male with left backache and past history of left kidney stone. (*B*) Intravenous pyelogram shows less hydronephrosis 2 to 3 months later. (*C*) Six weeks after the second intravenous pyelogram, there is increased obstruction. (*D*) Retrograde pyelogram shows irregular stricture-like tumor (open arrowhead).

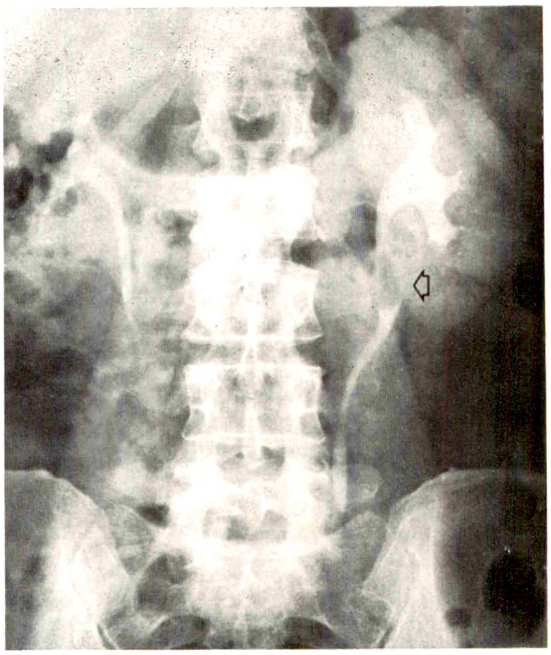
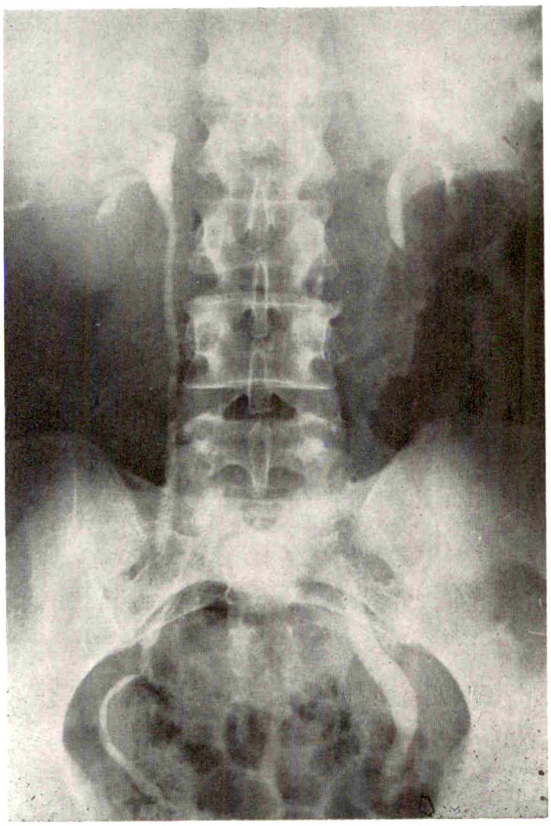


FIG. 5. Intravenous pyelogram shows proximal ureteral filling defect (open arrowhead) without marked hydronephrosis in a 49 year old white female with hematuria.



A variety of appearances of ureteral tumors on retrograde pyelography has been described. These include: (1) catheter hold-up at the tumor site (Fig. 8) (present in 1 of 9 retrograde pyelograms in our 21 cases); (2) dilatation about a filling defect (Fig. 3*B*) (our cases—2 of 9); (3) localized irregular stricture appearing area (Fig. 9) (our cases—4 of 9); (4) ill-defined lower tumor edge with complete block (Fig. 8) (our cases—1 of 9); and (5) sudden well-defined cut-off of contrast at the lower margin of the tumor (our cases—2 of 9).

Bergman *et al.*[5] in 1961 stated that the ureter below a calculus is collapsed, but with an expanding ureteral mass the lumen just below it is wider than normal.

Finby and Begg[11] described a coiling of the ureteral catheter just proximal to the ureteral tumor. They called this the "coiled-catheter sign."

Other described signs are Chevassu-Mock's sign (bleeding from the ureter as the obstruction is passed), and Marion's sign (bloody urine changing to clear urine when the catheter passes proximal to the ureteral tumor).[15]

Some authors suggest that diffuse stricture-like lesions can be clarified by angiography.[17,34] Others do not comment on angiography or feel that angiography plays only a confirmatory role.[4]

Retroperitoneal gas studies can be an adjunct to evaluation of ureteral lesions. The dissectability of retroperitoneal strictures by gas gives an evaluation of lesion resectability.[16]

DIFFERENTIAL DIAGNOSIS

The differential diagnosis includes: (1) nonopaque calculus such as a uric acid stone; (2) ureteral blood clot; (3) renal

FIG. 6. Intravenous pyelogram shows dilated ureter with a filling defect (open arrowhead) and no hydronephrosis in a 56 year old white female with hematuria.

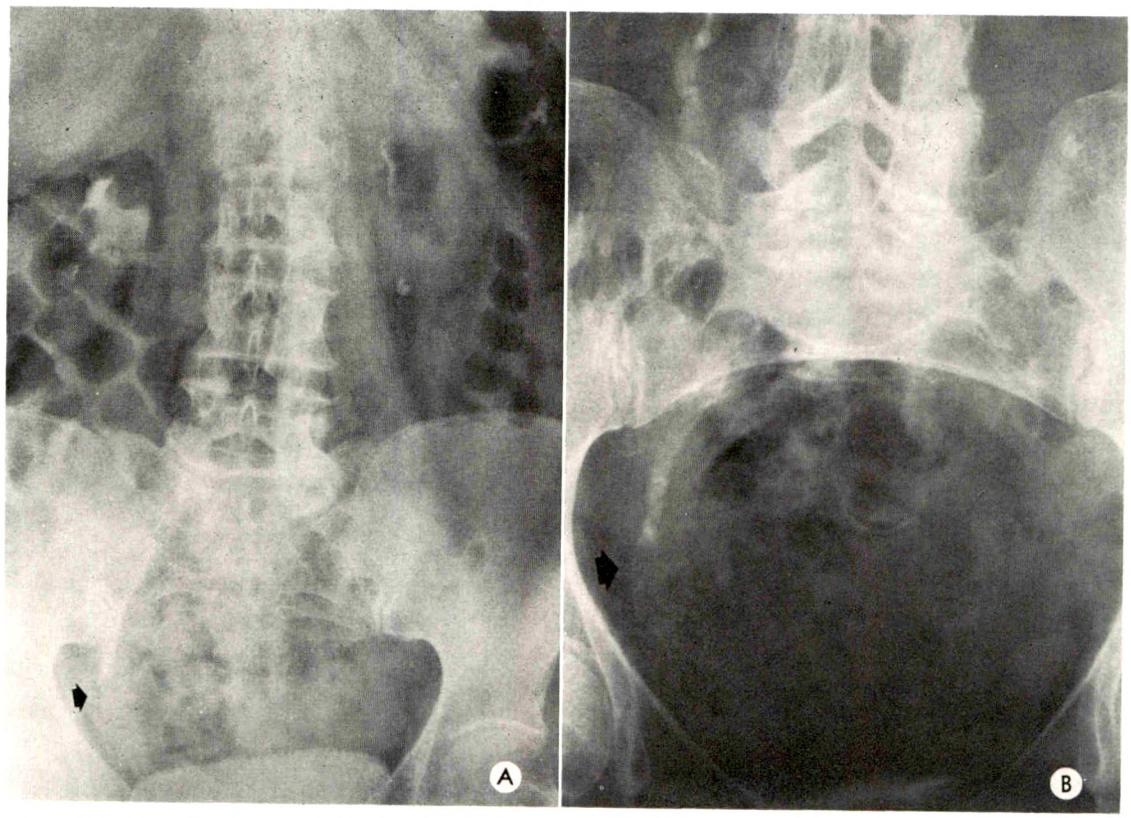


Fig. 7. (*A*) Distal ureteral defect (closed arrowhead) with holdup but no hydronephrosis in a 77 year old white female with hematuria. (*B*) Close-up of distal ureter.

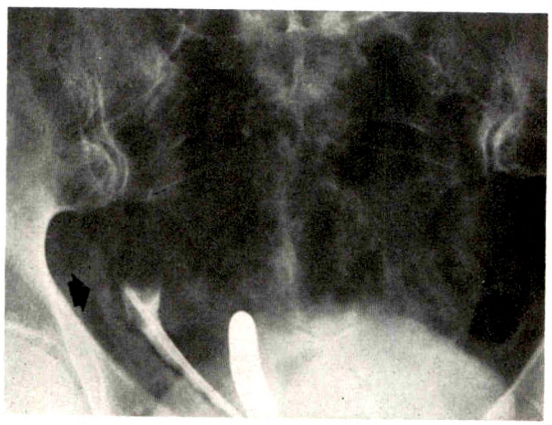


Fig. 8. Complete block (closed arrowhead) with holdup of catheter.

pelvis tumors with secondary ureteral tumors; (4) extrinsic compression due to external malignancies or lymph nodes; (5) benign stricture; (6) ureteritis cystica; (7) air bubbles; (8) rare nonspecific ureteral granulomas; (9) rare ureteral varicosities; and (10) dilated ureteral arteries secondary to a lesion such as renal artery obstruction.

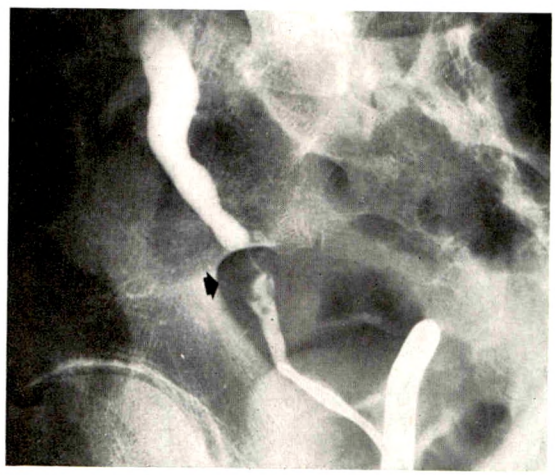


Fig. 9. Irregular stricture-like tumor (closed arrowhead).

TREATMENT

Nephro-ureterectomy with a bladder cuff is the procedure of choice unless there is need to conserve the kidney.

SUMMARY

In the patient with hematuria, intravenous urography suggests primary ureteral tumors as the cause when the IVP shows either total unilateral nonvisualization, unexplained unilateral hydronephrosis or a ureteral defect without hydronephrosis.

The incompletely filled, but apparently normal, ureters can mask a small lesion causing hematuria even in teenage patients. This is especially important since an increasing incidence of genitourinary tumors, including primary ureteral tumors, has been reported.

Our experience with 21 previously unreported cases is summarized and compared with 1,000 previously reported cases of primary ureteral tumors.

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REFERENCES

1. ABESHOUSE, B. S. Primary benign and malignant tumors of ureter· review of literature and report of one benign and 12 malignant tumors. *Am. J. Surg.*, 1956, *91*, 237–269.
2. ALBARRAN, J. (1900). Cited by Scott (1934).
3. ASCHNER, P. W. Primary tumors of ureter. *Surg., Gynec. & Obst.*, 1922, *35*, 749–758.
4. BECK, A. D., HESLIN, J. E., MILNER, W. A., and GARLICK, W. B. Primary tumors of ureter: diagnosis and management. *J. Urol.*, 1969, *102*, 683–688.
5. BERGMAN, H., FRIEDENBERG, R. M., and SAYEGH, U. New roentgenologic signs of carcinoma of ureter. AM. J. ROENTGENOL., RAD. THERAPY & NUCLEAR MED., 1961, *86*, 707–717.
6. BLOOM, N. A., VIDUNE, R. A., and LYTTON, B. Primary carcinoma of ureter: report of 102 new cases. *J. Urol.*, 1970, *103*, 590–598.
7. BRECKENRIDGE, R. L., LYNCH, P. V., and HULFELNER, F. D. Fibrous polyp of ureter. *J. Urol.*, 1963, *90*, 160–162.
8. BULKLEY, G. J., CARROW, L. A., and ESTENSEN, R. D. Endometriosis of ureter. *J. Urol.*, 1965, *93*, 139–143.
9. CRUM, P. M., SAYEGH, E. S., SACHER, E. C., and WESCUTT, J. W. Benign ureteral polyps. *J. Urol.*, 1969, *102*, 678–682.
10. DAVY, R. *Brit. M. J.*, 1884, *2*, 757.
11. FINBY, N., and BEGG, C. F. Carcinoma of ureter: coiled catheter sign. *New York J. Med.*, 1963, 2397–2399.
12. FOORD, A. G., and FERRIER, P. A. Primary carcinoma of ureter: with report of seven cases. *J.A.M.A.*, 1939, *112*, 596–601.
13. HERBUT, D. A. Urological Pathology. Lea & Febiger, Philadelphia, 1952.
14. HOLTZ, F. Papillomas and primary carcinoma of ureter: report of 20 cases. *J. Urol.*, 1962, *88*, 380–385.
15. JONSSON, G. Primary tumours of ureter: report of 17 cases. *Acta chir. scandinav.*, 1963, *126*, 368–378.
16. LANG, E. K. Retroperitoneal gas study in assessment of neoplastic and constricting lesions involving ureter. *Radiology*, 1969, *93*, 99–103.
17. LANG, E. K. Arteriographic diagnosis of primary and secondary tumors of ureter or ureter and renal pelvis. *Radiology*, 1969, *93*, 799–805.
18. LANG, E. K., and NOURSE, M. Roentgenographic diagnosis of obstructive lesions of ureter. *J. Urol.*, 1969, *101*, 812–820.
19. LAZARUS, J. A., and MARKS, M. D. Primary carcinoma of ureter with special reference to hydronephrosis: review of literature and report of unusual case. *J. Urol.*, 1945, *54*, 140–157.
20. LEBERT (1861). Cited by Melicow and Findlay (1932).
21. MACDOUGALL, J. A. Primary tumors of ureter. *Brit. J. Urol.*, 1961, *33*, 160–166.
22. MACKINNEY, C. C., KOHLER, F. P., and UHLE, C. A. Primary tumors of ureter. *J. Urol.*, 1965, *101*, 33–35.
23. MCINTYRE, D., PYRAH, L. N., and RAPER, F. P Primary ureteric neoplasms with report of forty cases. *Brit. J. Urol.*, 1965, *37*, 160–191.
24. MORTENSEN, H., and MURPHY, L. Primary epithelial tumors of ureter: report of 6 cases and literature review. *Brit. J. Urol.*, 1950, *22*, 103–115.
25. NEWMAN, D. M., ALLEN, L. E., WISHARD, W. N., JR., NOURSE, M. H., and MERTZ, J. H. Transitional cell carcinoma of upper urinary tract. *J. Urol.*, 1967, *98*, 322–327.
26. NEWSAM, J. E. Diagnosis of ureteric tumors. *Brit. J. Urol.*, 1966, *38*, 268–273.
27. PERLMUTTER, A. D., RETIK, A. B., and HARRISON, J. H. Simultaneous bilateral carcinoma of ureter present for five years before surgery. *J. Urol.*, 1965, *93*, 582–587.
28. RAYER, P. F. O. "Traité des Maladies des

Reins." Tome III. J. B. Baillière, Paris, 1841, pp. 699.

29. Renner, M. J. Primary malignant tumors of ureter. *Surg., Gynec. & Obst.*, 1931, *52*, 793–803.
30. Riches, E. W., Griffiths, I. H., and Thackray, A. C. New growths of kidney and ureter. *Brit. J. Urol.*, 1951, *23*, 297–356.
31. Robards, V. L., Jr., Thompson, I. M., and Ross, G., Jr. Primary tumors of ureter. *J.A.M.A.*, 1964, *187*, 778–780.
32. Scott, W. W. Review of primary carcinoma of ureter—presenting two cases. *J. Urol.*, 1943, *50*, 45–64.
33. Senger, F. L., and Furrey, C. A., Jr. Primary ureteral tumors with review of literature since 1943. *J. Urol.*, 1953, *69*, 243–258.
34. Siegelman, S. S., Hayt, D. B., Annes, G. P., and Goodgold, M. Angiography in carcinoma of proximal ureter. *Radiology*, 1968, *91*, 925–928.
35. Whitlock, G. F., McDonald, J. R., and Cook, E. N. Primary carcinoma of ureter: pathologic and prognostic study. *J. Urol.*, 1955, *73*, 245–253.
36. Wising, P. J., and Blix, C. (1878). Cited by Rousselot and Lamon (1930).
37. Woodruff, S. R. Secondary tumors of ureter: consideration of various types, with report of two cases. *J.A.M.A.*, 1935, *105*, 925–931.
38. Wynder, E. L., Onderdonk, J., and Mantel, N. Epidemiological investigation of cancer of bladder. *Cancer*, 1963, *16*, 1388–1400.

BENIGN PROLIFERATION PROCESSES PRESENTING AS MASS LESIONS IN THE URINARY BLADDER*

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TWO benign proliferative lesions of the urinary bladder, cystitis cystica and cystitis glandularis, are frequently confused with bladder tumors cystoscopically.[8,14,21] That these lesions may present roentgenographically as masses within the bladder, often indistinguishable from tumor, is less well known. Although mention of scattered cases with positive roentgenographic findings has been made in the urologic literature, only one previous article in the English language radiologic literature describes the roentgenographic appearance of one of these lesions.[4]

Our interest in these conditions was provoked by 2 recent cases in which there were large filling defects in the bladder indistinguishable from tumor (Fig. 1, *A* and *B*; and 2, *A–C*). Brief reports of these 2 patients are presented.

REPORT OF CASES

CASE 1. A 52 year old male presented with a 1 year history of dysuria, frequency, hesitancy, occasional hematuria, and straining at urination. Four months prior to admission, gross hematuria developed. Two bladder biopsies at another hospital showed no tumor, although this was strongly suspected from the cystoscopic appearance. An intravenous urogram obtained 3 months prior to admission was strongly suggestive of a bladder tumor (Fig. 1*A*). Cystoscopy and biopsy of an apparent tumor in the dome of the bladder showed only cystitis cystica histologically. Review of the biopsies from the patient's previous hospitalization also showed only cystitis cystica. However, review of the patient's intravenous urogram strongly suggested extrinsic invasion by tumor. A repeat bladder biopsy revealed mucin-producing adenocarcinoma of the bladder with invasion of the muscle layer. A subsequent

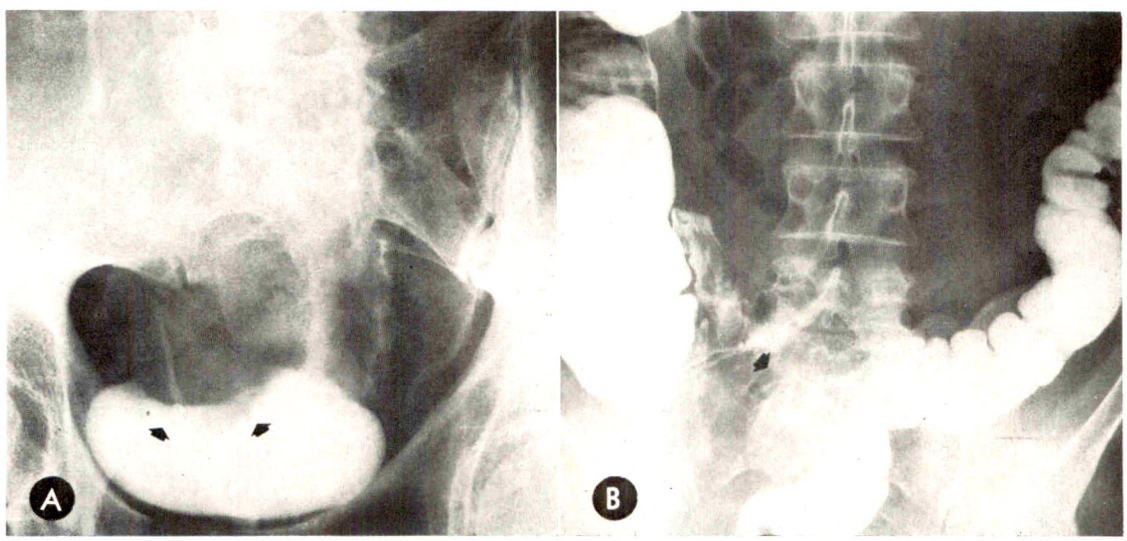


FIG. 1. Case 1. (*A*) There is an irregular filling defect in the right side of the dome of the bladder outlined by arrows. Three biopsies of this mass revealed only cystitis cystica. Not well shown in this reproduction is a contiguous soft tissue mass, suggesting that the findings were due to invasion by an extrinsic tumor. (*B*) A subsequent barium enema examination demonstrates an ulcerated neoplasm of the rectosigmoid in the area of the above soft tissue mass (arrow).

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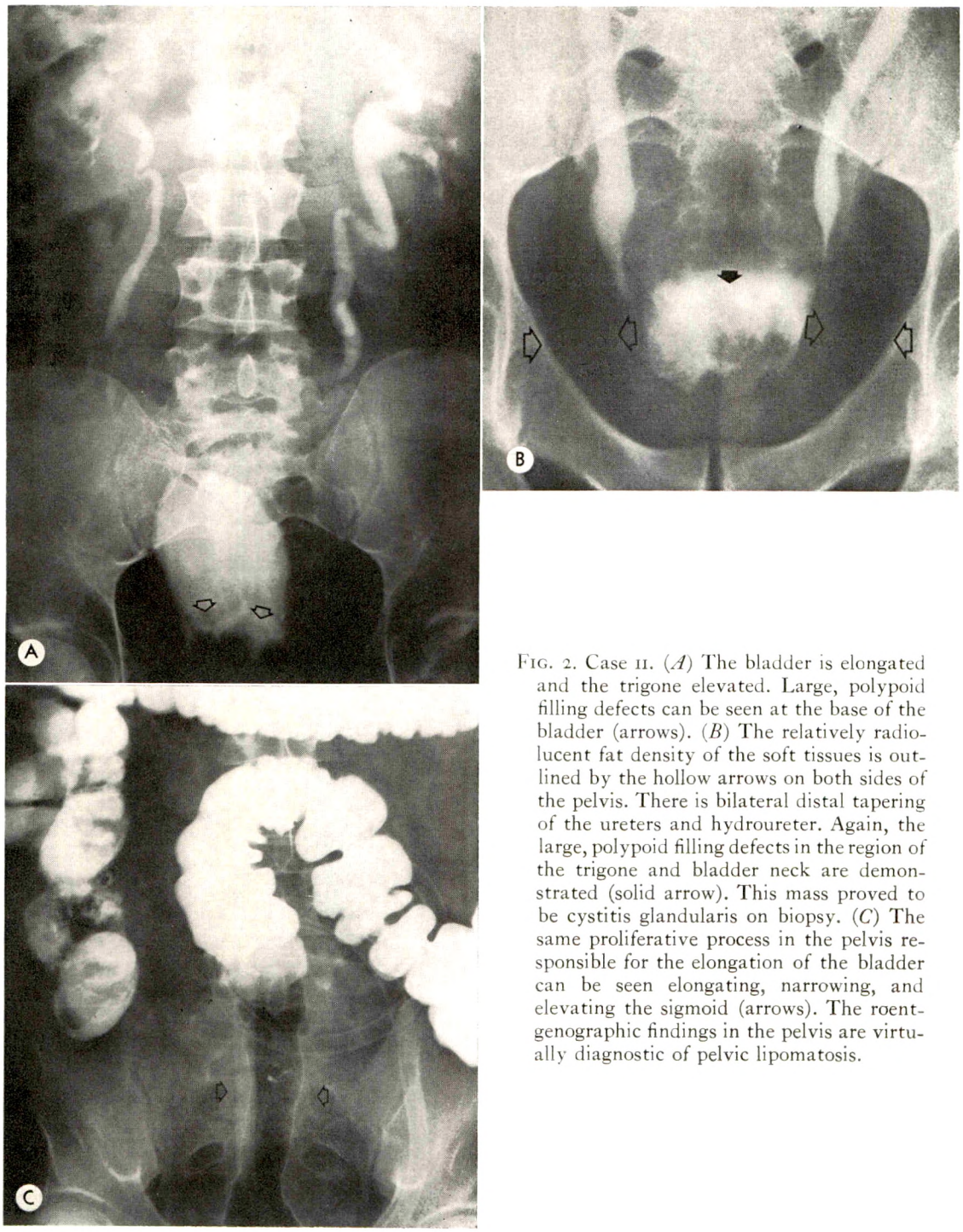


Fig. 2. Case 11. (*A*) The bladder is elongated and the trigone elevated. Large, polypoid filling defects can be seen at the base of the bladder (arrows). (*B*) The relatively radiolucent fat density of the soft tissues is outlined by the hollow arrows on both sides of the pelvis. There is bilateral distal tapering of the ureters and hydroureter. Again, the large, polypoid filling defects in the region of the trigone and bladder neck are demonstrated (solid arrow). This mass proved to be cystitis glandularis on biopsy. (*C*) The same proliferative process in the pelvis responsible for the elongation of the bladder can be seen elongating, narrowing, and elevating the sigmoid (arrows). The roentgenographic findings in the pelvis are virtually diagnostic of pelvic lipomatosis.

barium enema examination (Fig. 1*B*) demonstrated a lesion at the rectosigmoid junction strongly suggestive of a carcinoma of the colon. Subsequent abdominal exploration disclosed a sigmoid adenocarcinoma with direct extension and invasion of the bladder. An anterior resection, partial cystectomy, and uretero-ileostomy were performed.

Comment. Much of the deformity in the above case could be due to invasion of the bladder

TABLE I

ALL PATIENTS

	No. with Symptom or Condition/Total	Per Cent
Cystitis cystica		
Infection	6/15	40
Dysuria	7/14*	50
Hematuria	8/12*	67
Obstruction	5/15	33
Tumor	7/15	47
Exstrophy	1/15	7
Cystitis glandularis		
Infection	6/10	60
Dysuria	3/9*	33
Hematuria	6/9*	67
Obstruction	3/9*	33
Tumor	3/9*	33
Exstrophy	1/10	10

* Documentation of presence or absence of this associated symptom or condition not available from patient records in 1 or more cases.

wall by the colonic adenocarcinoma. However, this apparent tumor was biopsied on 3 different occasions with the histologic finding of cystitis cystica only. This strongly suggests that the bulk of the mass within the bladder is due to this proliferative lesion.

CASE II. A 30 year old male presented for evaluation of a "bladder tumor." The patient was essentially well until 8 years prior to admission, when he experienced the sudden onset of painless hematuria. He was admitted to another hospital, where cystoscopy reportedly showed no abnormality. He did well until 2 months prior to admission, when another episode of gross, painless hematuria occurred. Cystoscopy was again performed at another hospital and a "large bladder tumor" was seen. The patient was referred to the Hospital of the University of Pennsylvania, where intravenous urography demonstrated multiple large masses in the base of the bladder with elevation of the trigone suggesting a bladder tumor (Fig. 2, *A* and *B*). The bladder was also elongated and surrounded by tissue of fat density. An elongation and deformity of the rectum were subsequently demonstrated by barium enema examination (Fig. 2*C*). These roentgenographic changes were felt to be virtually diagnostic of pelvic lipomatosis,[9] although biopsy proof is lacking.

Cystoscopy and biopsy of what was thought to be a large bladder tumor were done with subsequent histologic diagnosis of cystitis glandularis. It was thought that the tumor mass had not been adequately biopsied. An open biopsy 4 days later showed several pedunculated lesions in the region of the bladder neck and trigone. Frozen section again showed only cystitis glandularis. This was confirmed on permanent sections of the biopsied material. The patient was placed on antibiotics and discharged. He has done well subsequently. A repeat urogram 5 months later showed no change in the appearance of the bladder or pelvic viscera.

These 2 cases prompted a review of all patients in whom the diagnosis of either cystitis cystica or cystitis glandularis had been pathologically documented at the Hospital of the University of Pennsylvania over the past 17 years.

MATERIAL AND RESULTS

There were 23 patients in whom the biopsy diagnosis of cystitis glandularis or cystitis cystica was made at this Hospital since 1954: 13 had cystitis cystica; 8 had cystitis glandularis; and 2 had both lesions concurrently. Of the total of 23 patients, 21 had excretory urograms available for review. Of these, 7 (33.3 per cent) had positive findings consisting of filling defects in the bladder, due entirely, or at least in part, to cystitis glandularis or cystitis cystica. Of the 7 patients in whom there were filling defects attributable to these lesions on excretory urography, 3 had cystitis glandularis, 3 had cystitis cystica, and 1 had both conditions simultaneously. This incidence of positive roentgenographic findings was, to us, surprisingly high.

A summary of the frequency of associated conditions and symptoms in our patients is given in Table I. A similar summary, restricted to the patients in whom there were positive findings on urography, is presented in Table II. Although the numbers are too small to be statistically significant, it is obvious that there is no apparent difference in the frequency of associated symptoms and conditions between those patients with, and those without, positive roentgenographic findings.

DISCUSSION

Both of these lesions are felt to represent metaplastic changes in the bladder epithelium induced by a wide variety of irritable or noxious stimuli.[6,8,12,13,15,16,18,21,25] A minority of authors feel that they may occasionally derive from embryonic rests.[19,21,23] An outline summary of the supposed pathogenesis of these lesions and their relationship to other metaplastic changes of bladder epithelium, is presented in Figure 3. Many authors feel that cystitis glandularis represents a further degree of metaplasia than, and can evolve from, cystitis cystica.[12,13,18,25] Both conditions are frequently associated with a variety of symptoms and conditions including dysuria, hematuria, infection, obstruction, and tumor.[2–4,12,21] Both lesions are frequently found in association with exstrophy of the bladder.[7] As mentioned previously, both conditions, particularly cystitis glandularis, can be, and often are, mistaken for bladder tumors at cystoscopy. Cystitis glandularis can even be confused histologically with adenocarcinoma of the bladder.[23] Although there exist many similarities between the 2 conditions, there are important differences.

Cystitis cystica appears cystoscopically as multiple sharply circumscribed, rounded, translucent submucosal beads, varying in color from pearly to yellow-brown.[5,11,25,27] These may vary in size from 1 mm. to 3 cm.[1] Cystitis cystica most often occurs in the trigone and/or bladder neck, but may occur anywhere in the bladder.

TABLE II

PATIENTS WITH POSITIVE EXCRETORY UROGRAMS

	No. of Patients with Symptom or Condition/Total	Per Cent
Cystitis cystica		
Infection	1/4	25
Dysuria	1/4	25
Hematuria	2/3*	67
Obstruction	1/4	25
Tumor	1/4	25
Cystitis glandularis		
Infection	2/4	50
Dysuria	1/4	25
Hematuria	3/3*	100
Obstruction	1/4	25
Tumor	1/4	25

* Documentation of presence or absence of symptom or condition not available from patient records in 1 or more cases.

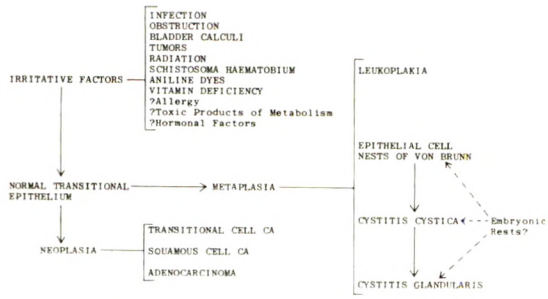


FIG. 3. Outline summary of the pathogenesis of various forms of bladder epithelial metaplasia and neoplasia. The degree of metaplasia increases from leukoplakia through cystitis glandularis.

Cystitis glandularis has a distinct cystoscopic appearance which varies with location. The majority of these lesions also occur in the vesical neck and trigone. Here the lesions are irregular mamillated elevations separated by deep ridges which lack the translucency of cystitis cystica and are sharply demarcated from the surrounding normal mucosa.[4,23,25] The second appearance, characteristic of those lesions occurring high in the dome of the bladder, is that of fleshy, villous-like proliferations, which may be confluent or be separated from each other by normal mucosa.[4,23] These lesions may reach a size of up to several centimeters.

Cystitis cystica has an extremely high (90 to 95 per cent) association with infection, obstruction, tumor, bladder calculi, or ureteral obstruction or stasis.[17] Approximately 50 per cent have either current evidence of, or a history of, infection.[27] Kaplan and King[11] felt, on the basis of personal experience, that the presence of cystitis cystica in children identifies patients who are likely to have infections which are difficult therapeutic problems. Although cystitis glandularis also has a high association with these irritant factors, the association with infection is appreciably less than is the case with cystitis cystica.[4,14,23] Only cystitis glandularis has been reported to cause uni-

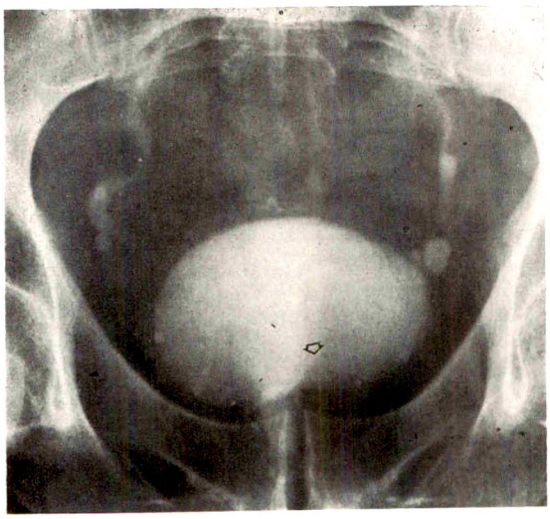


FIG. 4. Close-up of excretory urogram from a 72 year old male with a chief complaint of hematuria. There is a filling defect in the left trigonal area which was thought to be a bladder tumor (arrow). Biopsy showed cystitis cystica.

lateral or bilateral hydronephrosis.[2–4,12,21] Cystitis glandularis may present as urinary obstruction in otherwise healthy young males without any associated pathology.[14] It may, as in our Case 11, also present as painless hematuria in this same group of patients.[4]

Many authors feel that both lesions are potentially premalignant.[2,16,21,22] Although cystitis cystica is theorized to be premalignant, this has never been proven. However, there have been 2 well documented cases of cystitis glandularis developing into an adenocarcinoma of the bladder.[17,24] Many authors have reported the histologic appearance of adenocarcinoma developing adjacent to areas of cystitis glandularis.[6,13,20,22,26] Both cystitis glandularis and adenocarcinoma may present with the unique symptom of constant elimination of mucus during, and/or following, voiding.[6,23]

Because cystitis glandularis is potentially premalignant, and because it may cause ureteral obstruction, many authors feel that this lesion should be treated by fulguration and/or resection. Patients should be followed closely by both cystoscopy, and, when indicated, repeated biopsy. Occasionally, cystectomy may be needed for relief of ureteral obstruction. However, the usual treatment for both cystitis cystica and cystitis glandularis is the specific treatment of the underlying infection and/or obstruction.

ROENTGEN APPEARANCE

Both cystitis cystica and cystitis glandu-

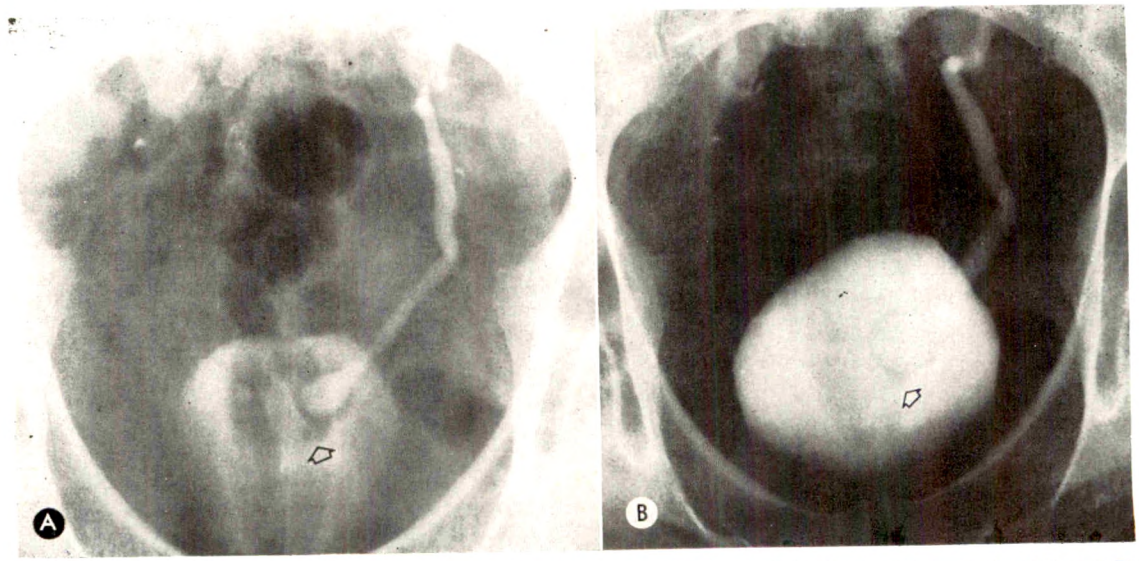


FIG. 5. A 56 year old male with a chief complaint of urinary hesitancy, dribbling, and nocturia. (*A* and *B*) Excretory urograms demonstrate a left ureterocele with a filling defect attached to the lower pole of the ureterocele (arrows). This lesion proved on biopsy to represent both cystitis cystica and cystitis glandularis.

laris may present as filling defects in the urinary bladder on excretory urography. Several cases, demonstrating such findings, have been published in the urologic literature.[2,4,5,14,17,24] Deformity and irregularity of the trigone have also been demonstrated.[3] Grieve[10] reported 2 cases of cystitis cystica associated with active infections. Both had cystic changes in the bladder wall mimicking a bladder tumor on excretory urography. These completely disappeared, both cystoscopically and roentgenographically, with successful treatment of their infections. The roentgenographic appearance of these lesions has varied from a single large filling defect, as in our Case 1 (Fig. 1*A*), to multiple small, rounded filling defects similar to our Case 11 (Fig. 2, *A* and *B*).

Only cystitis glandularis has been reported as a cause of obstructive uropathy.[2–4,12,14,21] These lesions occurred at or near the trigone causing either unilateral or bilateral hydroureter and pyelocaliectasis. This appearance, plus frequent association with hematuria, explains why the lesion is readily mistaken for a bladder tumor.

Brogdon *et al.*[4] made the first mention of either disease in the radiologic literature in 1965. They described roentgenographic changes in the urinary bladder due to cystitis glandularis. Of 14 cases, only 3 permitted evaluation of the roentgenographic findings: 3 patients had multiple rounded filling defects in the bladder; 2 had unilateral hydroureter and hydronephrosis.

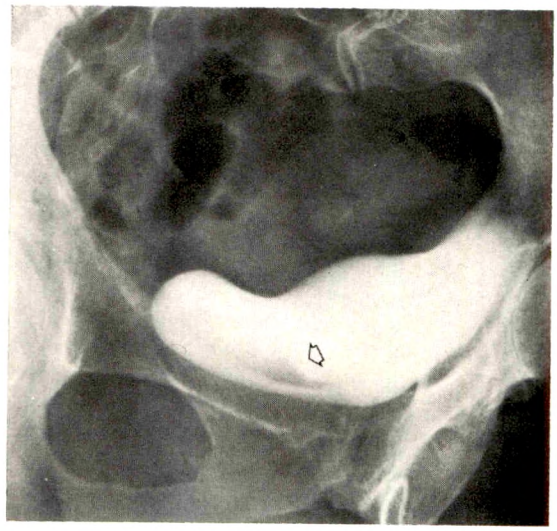


Fig. 6. A 71 year old female with bladder infection. There is a filling defect at the right base of the bladder which was thought to represent a bladder tumor on excretory urography (arrow). Cystoscopy and biopsy of this lesion demonstrated cystitis glandularis.

All 7 of our patients had filling defects in the bladder clearly attributable to one or both lesions. These were either within the lumen of (Fig. 5, *A* and *B*; 7, *A* and *B*; and 8), or protruding from one wall of (Fig. 1*A*; 2, *A* and *B*; 4; and 6) the bladder. The majority were in the region of the trigone or ureteral orifices, although lesions also occurred elsewhere. In addition to tumor, the lesions might mimic radiolucent

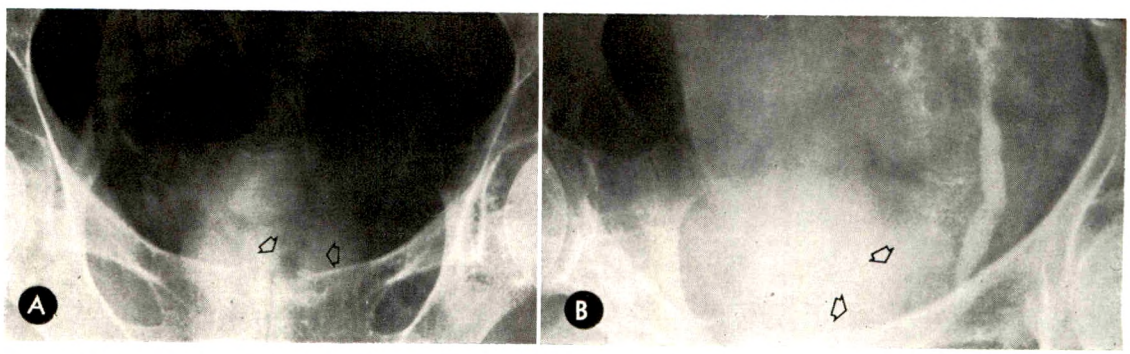


Fig. 7. Patient with bladder infection and pelvic prolapse. There are multiple rounded filling defects in the left base of the bladder in both the anteroposterior (*A*) and oblique (*B*) views (arrows). Cystoscopy demonstrated a pedunculated tumor approximately 2 cm. in size to the left of the trigone. Biopsy showed cystitis glandularis.

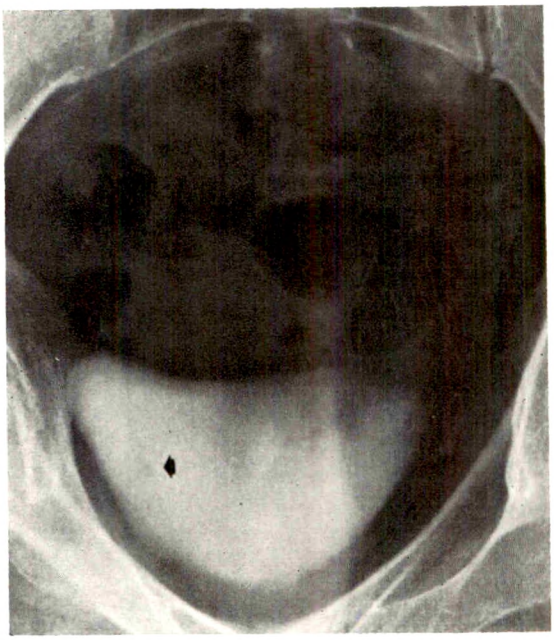


FIG. 8. A 66 year old female complaining of frequency and urgency of 6 months' duration. There is a small filling defect in the right side of the bladder (arrow). A "papilloma" was found in this same area at cystoscopy and excised. The pathologic diagnosis was cystitis cystica.

stone, blood clot, bullous cystitis, or foreign body. Our Case 11 is the first known reported instance of coincidental cystitis glandularis and pelvic lipomatosis—another benign condition which mimics neoplasm.[9]

Except when ureteral obstruction exists, cystitis glandularis appears identical to cystitis cystica. It should be emphasized that both lesions may occur in otherwise healthy young patients, not in the usual cancer age group. The finding of a "tumor" in such a patient should alert the radiologist as to the possibility that this could instead represent one of these benign bladder lesions. Cystoscopy and biopsy are required for definitive diagnosis.

SUMMARY

Two benign, proliferative lesions of the urinary bladder—cystitis cystica and glandularis—are briefly discussed.

These lesions can mimic bladder tumors and other filling defects roentgenographically and cystoscopically.

Seven of 21 cases had filling defects of the bladder on excretory urography. One represents the first known report of a coincidental occurrence of cystitis glandularis and pelvic lipomatosis.

Because of their frequent association with bladder tumors, careful follow-up of patients with these lesions is necessary. The radiologist should be aware of the roentgenographic appearance and clinical significance of these lesions.

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REFERENCES

1. ANDERSON, W. A. D., and PESSIN, S. B. Lower Urinary Tract and Male Genitalia Pathology. Edited by W. A. D. Anderson. C. V. Mosby Company, St. Louis, 1966, pp. 660.
2. BELL, T. E., and WENDEL, R. G. Cystitis glandularis: benign or malignant? *J. Urol.*, 1968, *100*, 462–465.
3. BLUM, J. A. Cystitis glandularis causing bilateral urinary tract obstruction. *Chicago Med. School Quarterly*, 1966, *25*, 250–252.
4. BROGDON, B. G., SILBIGER, M. L., and COLSTON, J. A. C., JR. Cystitis glandularis. *Radiology*, 1965, *85*, 470–473.
5. CINGELL, J. C., and BURN, J. I. Cystitis glandularis—case report. *Brit. J. Urol.*, 1970, *42*, 446–449.
6. COPPRIDGE, W. M., ROBERTS, L. C., and CULP, D. A. Glandular tumors of bladder. *J. Urol.*, 1951, *65*, 540–549.
7. CULP, D. A. Histology of exstrophied bladder. *J. Urol.*, 1964, *91*, 538–548.
8. EMMETT, J. L., and MCDONALD, J. R. Proliferation of glands of urinary bladder simulating malignant neoplasm. *J. Urol.*, 1942, *48*, 257–265.
9. FOGG, L. B., and SMYTH, J. W. Pelvic lipomatosis: condition simulating pelvic neoplasm. *Radiology*, 1968, *90*, 558–564.
10. GRIEVE, J. Contrasting cases of cystitis cystica. *Brit. J. Urol.*, 1965, *37*, 157–159.
11. KAPLAN, G. W., and KING, L. R. Cystitis cystica in childhood. *J. Urol.*, 1970, *103*, 657–659.
12. KITTREDGE, W. E., and BRANNAN, W. Cystitis glandularis. *Tr. Am. A. Genito-Urin. Surgeons*, 1958, *50*, 136–147.
13. KITTREDGE, W. E., COLLETT, A. M., and MORGAN, C., JR. Adenocarcinoma of bladder as-

sociated with cystitis glandularis: case report. *J. Urol.*, 1964, *91*, 145–150.

14. Lowrey, E. C., Hamm, F. C., and Beard, D. E. Extensive glandular proliferation of urinary bladder resembling malignant neoplasm. *J. Urol.*, 1944, *52*, 133–138.
15. Morse, H. D. Etiology and pathology of pyelitis cystica, ureteritis cystica and cystitis cystica. *Am. J. Path.*, 1928, *4*, 33–50.
16. Mostofi, F. K. Potentialities of bladder epithelium. *J. Urol.*, 1954, *71*, 705–714.
17. Parker, C. Cystitis cystica and glandularis: study of 40 cases. *Proc. Roy. Soc. Med.*, 1970, *63*, 239–242.
18. Patch, F. S., and Rhea, L. S. Genesis and development of Brunn's nests and their relation to cystitis cystica, cystitis glandularis and primary adenocarcinoma of bladder. *Canad. M. A. J.*, 1935, *33*, 597–606.
19. Pund, E. R., Yount, H. A., and Blumberg, J. M. Variations in morphology of urinary bladder epithelium: special reference to cystitis glandularis and carcinomas. *J. Urol.*, 1952, *68*, 242–251.
20. Rohman, M., and Sommers, S. C. Double primary carcinoma of urinary bladder. *J. Urol.*, 1954, *72*, 1174–1177.
21. Roll, W. A. Cystitis glandularis as cause of hydronephrosis. *J. Urol.*, 1960, *84*, 76–78.
22. Salm, R. Neoplasia of bladder and cystitis cystica. *Brit. J. Urol.*, 1967, *39*, 67–72.
23. Sauer, H. R., and Blick, M. S. Cystitis glandularis: consideration of symptoms, diagnosis and clinical course of disease. *J. Urol.*, 1948, *61*, 446–458.
24. Shaw, J. L., Gislason, G., and Imbriglia, J. E. Transition of cystitis glandularis to primary adenocarcinoma of bladder. *J. Urol.*, 1958, *79*, 815–822.
25. Stirling, C., and Ash, J. E. Chronic proliferative lesions of urinary tract. *J. Urol.*, 1941, *45*, 342–359.
26. Swersie, A. K., Solomon, C., and Rayos, B. O. Urachal adenocarcinoma of urinary bladder. *J. Internat. Coll. Surgeons*, 1959, *31*, 169–173.
27. Warrick, W. D. Cystitis cystica: bacteriological studies in series of 28 cases. *J. Urol.*, 1941, *45*, 835–843.

MALACOPLAKIA OF THE URINARY TRACT*

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MALACOPLAKIA was recognized as a new entity microscopically, long before suitable procedures were available for clinical diagnosis.[18] The smooth, yellowish-white, hemispheric mammillations may stud surfaces of the genitourinary tract, usually appearing in persistent coliform infections. They are composed exclusively of unusual foam cells[11] (Hansemann cells). Their characteristic is the presence of round bull's eye, or target inclusions in cytoplasm. These mimic a second nucleus very closely in size, except that they have adsorbed phosphate and iron, which are demonstrable histochemically (Michaelis-Gutmann bodies). The transitional epithelium remains intact over these hemispheric accumulations, which may show a rim of superficial hemorrhage in this early phase and bleed. Then they soften slowly, healing individually over a period of months, and dimple centrally. The lesions gradually become thin flat plaques, with a very characteristic ringed "moon crater" appearance on cystoscopy. It is this late phase which is best known clinically.

Although there are only approximately 120 individual instances of malacoplakia reported,[9,21] we believe that the condition is not rare. One was recognized in our large genitourinary service before 1970, but 4 have been found since. As there are few illustrations of the radiology,[4,5,13] and discussions of radiologic appearances are rare,[8] we reviewed these 5 instances in retrospect for differential diagnostic features.

THE EVOLUTION IN COMPARATIVE RADIOLOGIC AND CYSTOSCOPIC FEATURES

In malacoplakia of bladder the *waxing phase* produces smooth, discrete, domed filling defects, which are usually multiple. While these are not more than 5 mm. in diameter, their radiologic appearances are inseparable from those of cystitis cystica. Characteristically, malacoplakia is yellow on cystoscopy, and the overlying epithelium remains intact. Malacoplakic nodules may grow to 2.5 cm. in diameter. Should they be apparently solitary or few in number, they may then mimic neoplasm, which lacks the yellow color on cystoscopy (Fig. 1–3).

The only other nodules we know which



FIG. 1. Case 1. The excretory urogram shows moderate dilatation of the proximal and distal right ureter. Multiple scalloped and nodular filling defects distort the whole bladder surface.

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are yellow are localized amyloid plaques of bladder.[15] Generally these are single or very few in number, and evolve by ulcerating as their vessels are affected. Otherwise, they are distinguishable only by biopsy.

Malacoplakia has a tendency to involve the lower ureter and adjoining trigone. This is also a site of predilection for endometrioma of bladder.[7] The covering epithelium remains intact over this lesion also, but it is usually lobulated and pallid. In addition, spontaneous resolution occurs only after the menopause. Clinically, both lesions are associated with presenting symptoms of terminal hematuria and obstructive hydronephrosis. In our Case II, malacoplakic infiltrates of the lower ureter made insertion of a catheter very difficult. Mucosal filling defects were finally shown in the distal two-thirds of the ureter (Fig. 4 and 5). The terminal ureteric segment was resected, with the adjoining trigone, producing relief. However, 2 years later the chronic pyelonephritic process had progressed and the nonfunctioning right kidney was then removed. Only a solitary 3 mm. malacoplakic nodule remained in the ureter at this time.

We note here that prominent scarring or stenosis is not to be expected as a residuum of malacoplakia. This lesion remains characteristically nonfibrogenic and superficial. When it heals, the foam cells resolve completely.

Where malacoplakic filling defects in bladder are multiple, and reach a large size,

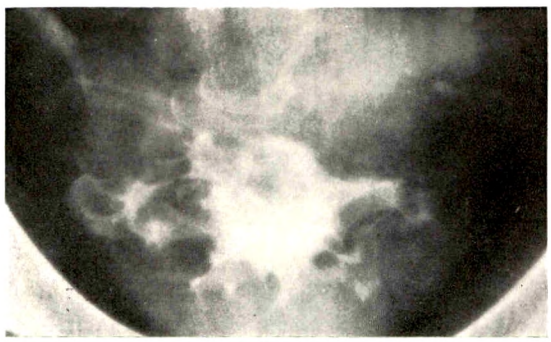


Fig. 2. Case I. Postvoiding excretory urogram shows multiple smooth nodular filling defects reaching 2.5 cm. in diameter.

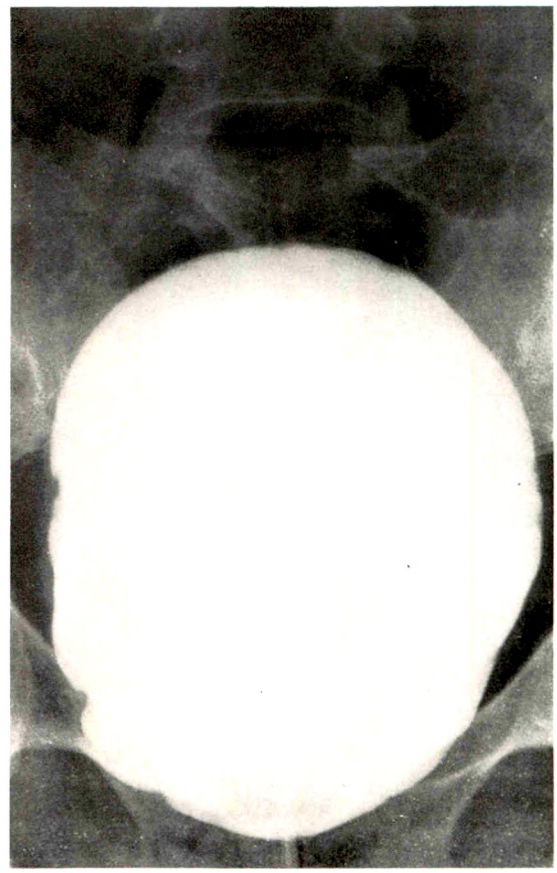


Fig. 3. Case I. Cystogram shows asymmetric outline and smooth filling defects on the right side.

their radiologic appearances are identical with those of bullous cystitis. This latter condition is likely to occur temporarily in the bladder dome, rather than the base. It is often associated with inadvertent injury from deeply inserted catheters, over which the decompressing bladder "tents." When associated with acute cystitis, the trigonal epithelium is the most firmly attached and least likely to yield to bullous edema. With bullous cystitis of either type, the clinical resolution, with treatment, is rapid. Large malacoplakic filling defects may occur anywhere in the bladder, and the largest have been ballotable with the cystoscope.[4] Healing occurs spontaneously, but slowly over a period of months, when the nodules become soft, are flattened, while still remaining nonulcerated, and finally they umbilicate. We are not aware of any demonstration of

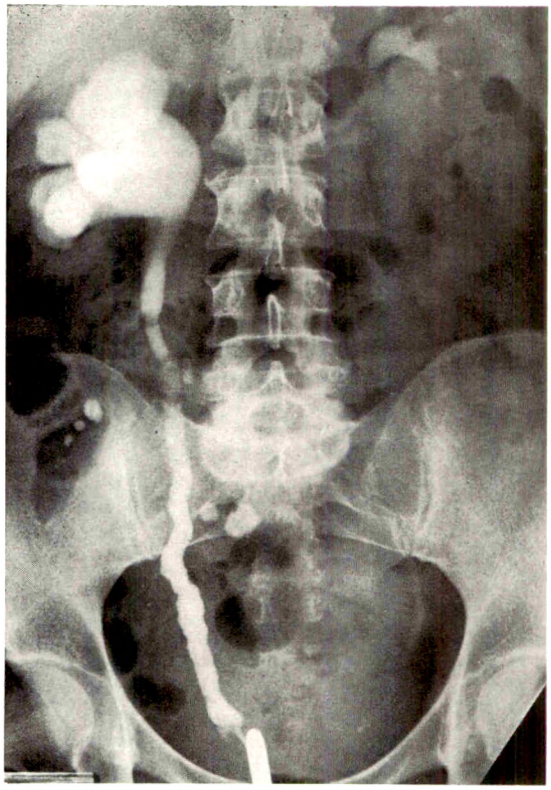


FIG. 4. Case II. Retrograde pyelogram shows multiple contiguous shallow filling defects in the distal two-thirds of the right ureter. Distinct right hydronephrosis and mild hydroureter are present.

the radiologic counterpart to this serial cystoscopic evolution. Cystograms in numerous oblique views, and double contrast techniques could improve radiologic assessment in estimating the stage in the natural history of the condition. However, cystoscopy and biopsy are usually sufficient.

The later umbilicated lesions, which have been characteristic (Fig. 11) for so long on cystoscopy, become so flat that they may not be demonstrable radiologically. While the condition is *focally self-limiting*, healing in one area is often accompanied by the appearance of nodules in adjoining sites. In our Case IV, where there was persistent coliform bacilluria, 5 crops of nodules were seen in differing locations on cystoscopic examinations repeated over a 2 year period. Case V showed 3 similar recurrences over 9 months of observation. Cowie and Whitaker[4] reported 1 instance, where there had been persistence, and passage of pseudotumor fragments, over 8 years.[4]

Eventual spread of malacoplakia to the ureters and renal pelves has been reported in a number of cases. There are very few radiologic appearances published, but in all the nodules have been small and multiple giving a most characteristic appearance (Fig. 5–8). The rounded filling defects may stud the entire pelvicalyceal and ureteric surfaces. They are somewhat flatter than ureteritis cystica and not so sharply defined. Pyeloureteritis cystica differs chiefly in that the filling defects remain hemispheric, with intervening normal mucosa. In this situation the malacoplakic nodules tend to coalesce, as can be seen on micros-



FIG. 5. Case II. A magnification of the distal right ureter shows the shallow filling defects to better advantage. Note that their contour is not as hemispherical as in pyelitis cystica.

copy. Kolodny[13] mentioned the similarity in the 2 appearances radiologically, but we think that they can be differentiated.

The most important radiologic differential diagnosis is exclusion of papillary transitional cell carcinomatosis, which often has a similar distribution. The papillary lesions vary considerably in size, and as many are very fine fronds interspersed with larger nodules of several millimeters in diameter, their margin of filling defect appears rougher, irregular or mottled. It may be indistinguishable (Fig. 6).

The longitudinal mucosal folds, which may be seen in upper urinary tract infections, ureteric reflux, and upon decompression of hydronephrosis, are more regular in distribution and are longitudinally arrayed.[23] Pyeloureteritis emphysematosa can be easily distinguished by the demonstration of gas in the tissues seen on preliminary roentgenograms and by the bizarre distortion of the renal pelvis which this produces.[3] While tissue necrosis may occur, especially in diabetics, this is not a feature of malacoplakia. Tuberculous ureteritis can produce granular filling defects, but these are usually associated with multiple strictures and some shortening of the ureter. Leukoplakia is not expected to present any differential diagnostic problem, for this is usually a large plaque-like lesion with linear wrinkling rather than a multiplicity of small nodules. The other temporary, nonmucosal, intraluminal defects due to gas bubbles, blood clots, and nonopaque calculi are excluded by repeat examination. Proximal ureteric compression by dilated ure-

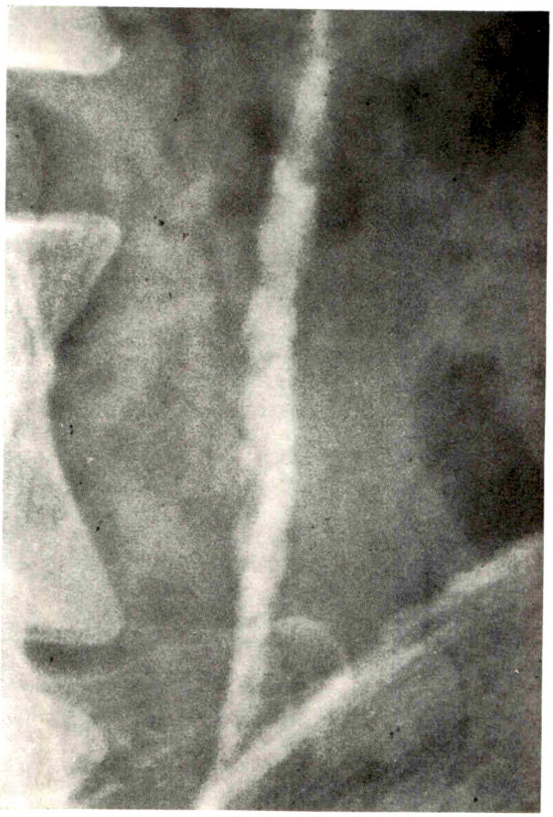


Fig. 6. Case III. Retrograde pyelogram of left ureter shows feathery irregularity due to proven malacoplakia simulating papillary transitional cell carcinoma.

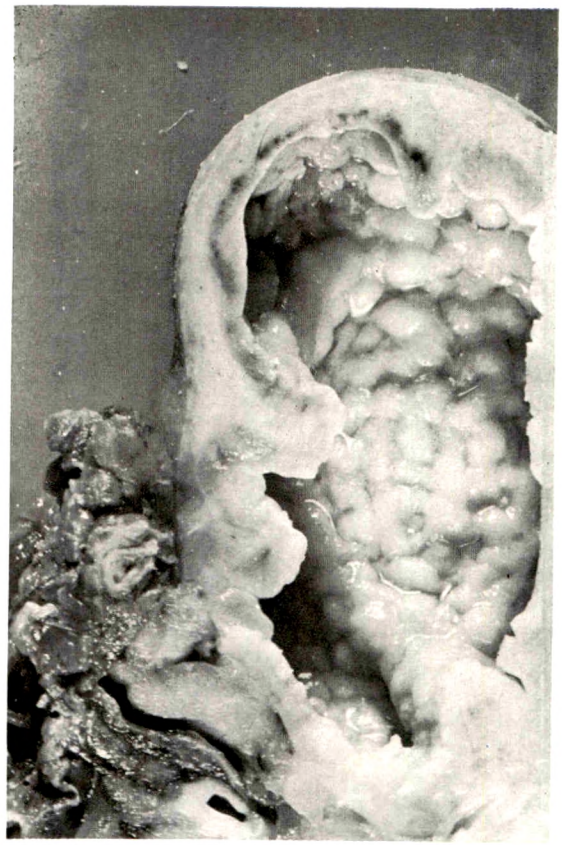


Fig. 7. Case III. Mammillated subepithelial malacoplakic nodules stud the surfaces of upper dilated renal pelvis. The epithelium is characteristically intact throughout. These lesions are early. Only one shows the umbilication of healing.



FIG. 8. Case III. Retrograde pyelogram shows calyectasis. Small smooth scalloped filling defects stud the calyces and the pelviureteric region.

teral arteries causes multiple sharp extrinsic marginal defects easily differentiated from those of malacoplakia.

PATHOLOGIC CORRELATIONS

The first description of malacoplakia was given by Michaelis and Gutmann[18] in 1902, as umbilicated, whitish-yellow, foam cell plaques in bladder with characteristic sidero-calcific inclusions on microscopy. This is recognizable now as the late phase. Clinically, the patients' histories of dysuria are not distinctive; 2 of our cases involving bladder had an initial complaint of painless, terminal hematuria.

While most reports show good photomicrographic illustrations, there are very few of gross appearances. The late healing phase with characteristic "moon crater" flat plaques is by far the best illustrated (Fig. 11) by Morison.[19] Before 1957, of 60 reported cases 45 were discovered at autopsy and reported chiefly in the German literature.[17] By 1956 "a case presented at a Pathology Seminar was recognized only by five out of one-hundred-fifty-one participants, . . . and eighty-one regarded it as a carcinoma."[24] Awareness of the condition was stimulated by an Armed Forces Institute of Pathology slide study set #L6864. This deals with malacoplakia of the urinary tract. In Smith's series of 24 cases, compiled at the Institute,[24] one quarter of the lesions were firm and therefore presumably early phase, not soft as the term "malaco" implies.

The lesions are typically of surfaces, but similar foam cells with Michaelis-Gutmann bodies have been detected in 1 case with extension of the foam cell reaction from renal pelvis into a perinephric abscess.[22] Malacoplakia occurs in bladder, ureter, renal pelvis, prostate and testis,[12] in that order of frequency.

The condition is sometimes confused with renal xanthogranuloma in the literature, because both conditions contain foam cells. While they may overlap infrequently,[1] they differ in many respects. None of our last 10 cases of renal xanthogranuloma, on which

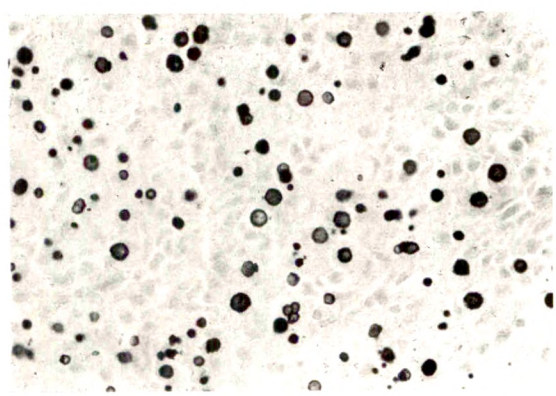


FIG. 9. Case V. Malacoplakia stained by von Kossa method for phosphate. Far more calco-spherocytes (Michaelis-Gutmann bodies) show than on H&E stain.

we made a comparison, showed any Michaelis-Gutmann bodies. This granuloma produces massive, hard scarring chiefly in perinephric fat, fixing kidney to inferior vena cava or aorta and mimicking renal cortical carcinoma. There are no radiologic similarities.[6]

As might be expected from the close clinical association with coliform and *Aerobacter aerogenes* infections,[2] some 20 instances are reported in nongenitourinary sites.[9] The first were associated with ureteric fistulas, but the foam cell reaction has been seen confined entirely to the intestinal tract. It must then be distinguished histochemically from Whipple's lipodystrophy.[27] For example, it has been reported in an inflammatory polyp of the colon with extension into a pericolic abscess,[9] confined to submucosa of stomach and large intestine,[27] and in an extension of a pelvic abscess to groin.[20] Gupta *et al.*[10] reported an unusual case which seemed to have disseminated from the genitourinary tract by the blood stream, producing tumor-like masses in both lungs and vertebrae.

The origin of the concentrically laminated bull's eye Michaelis-Gutmann inclusions was investigated by Terner and Lattes.[25] The concretions appeared to be polysaccharide "not compatible with known human mucin . . . but compatible with a capsular polysaccharide, lipopolysaccharide, or endotoxin of micro organisms." Loele,[14] and later Bleich and Konikov,[2] "showed that similar calcific spheroids could be produced on incubating *E. coli* with urine and blood." The latter 2 media do not seem now to be essential components.[9,25] Michaelis-Gutmann bodies have been found outside foam cells, but more frequently inside cell cytoplasm. The foam cells may contain bacterial debris and red cells. However, electron microscopic studies, while supporting origin from phagocytosed material, tend to exclude red cell debris as the source of the inclusions, as their ferritin content is minimal.[9,26]

The so-called "characteristic basophilia" of the sidero-calcific inclusions on hematoxylin stain is due to its affinity for the iron and perhaps calcium eventually adsorbed to the acid bacterial lipid polysaccharide substances. Other ions are also absorbed. For example, Gomori reported that the von Kossa procedure used for demonstration of calcium was "in reality a method for phosphates which is virtually

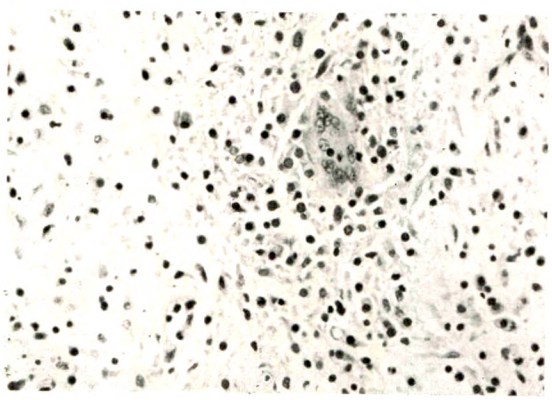


Fig. 10. Case III. A histiocytic granuloma with fusion giant cell in a pseudotubercle adjoining a minute focus of calcification in malacoplakia of renal pelvis.



Fig. 11. Late malacoplakia of bladder showing the characteristic umbilicated yellow plaques, from a 6 year old patient with recurrent pyelonephritis. (Reproduced with permission from the *Journal of Pathology and Bacteriology*.[19])

the only form inorganic calcium takes in mammalian tissues."[25] In diagnostic practice the spheroids are difficult to see on H and E stain, and far more are detectable histochemically (Fig. 9 and 10).

Varying associations with other conditions are reported. All appear to have been coincidental. We think that confusion in the past with tuberculosis or sarcoidosis[5] was very understandable. In malacoplakia dense histiocytic clusters, simulating tuberculoid granulomata, do appear containing scattered giant cells and microscopic calcification. Tubercle bacilli are not found in these on search using sensitive fluorescent techniques (Fig. 10). Malacoplakia may concur with genitourinary tuberculosis as in our Case III, who had successful chemotherapy for tuberculous epididymitis, temporary tuberculous bacilluria, and a subsequent persistent coliform cystitis. Ingenious suggestions were made in the past concerning the Michaelis-Gutmann inclusion bodies; for example, that they were cryptococcal, coccidioidal, granular cell myoblastoma,[8,17] due to the action of coliform bacilli stimulating growth in pre-existing adrenocortical rests,[16] etc. The difficulties of excluding these exotic possibilities have probably reduced the reasonable frequency of reporting of malacoplakia.

As persistent coliform infections of the genitourinary tract are common, the problem still remains why few seem to provoke this unusual localized and persistent subepithelial foam cell reaction. The intact transitional epithelium presumably protects the foam cells from dispersion. Only minimal puncture of a nodule by a ureteric catheter is sufficient to accelerate local resolution from our experience.

SUMMARY

Malacoplakia appears as dome-shaped yellow nodules which stud the surfaces of the genitourinary tract. Throughout their evolution the overlying epithelium remains intact. In early phases they simulate pyelocystitis cystica, which is usually far more discrete. The malacoplakic nodules grow to 2.5 cm. diameter in bladder and then resemble bullous cystitis, or if single they simulate tumor or endometrioma. Only isolated amyloid nodule of bladder, however, is also yellow.

As malacoplakic nodules heal, they do so individually, and have an umbilicated moon-crater appearance which is quite characteristic. Crops of lesions may appear over several years of observation. Final healing is without scarring.

The various radiologic features and cystoscopic and pathologic correlations are reviewed from studies on 5 instances.

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REFERENCES

1. ANGELL, J. C., and SMITH, I. Renal malacoplakia with perinephric extension. *Brit. J. Urol.*, 1968, *40*, 429–432.
2. BLEICH, V. R., and KONIKOV, N. F. Malacoplakia of urinary bladder. *A.M.A. Arch. Path.*, 1952, *54*, 388–397.
3. BLIZNAK, J., and RAMSEY, J. Emphysematous pyelonephritis. *Clin. Radiol.*, 1972, *23*, 61–64.
4. COWIE, A. G. A., and WHITAKER, R. H. Malacoplakia of bladder with recurrent passage of pseudo-tumour fragments over eight years. *Brit. J. Surg.*, 1970, *57*, 883–885.
5. CURTIS, W. R., BOZZELL, J. D., and GREEN, C. L. Malacoplakia of bladder: report of case successfully treated with anti-tuberculous medical therapy. *J. Urol.*, 1961, *86*, 78–81.
6. ELLIOTT, G. B., JOHNSON, H. W., and BALFOUR, J. A. Xanthogranulomatous pyelonephritis and perirenal xanthogranuloma. *Brit. J. Urol.*, 1968, *40*, 548–555.
7. ELLIOTT, B. G., CHRISTENSEN, R. M., and ELLIOTT, K. A. Invasive endometriosis of intestine: report of 21 cases. *Canad. J. Surg.*, 1970, *13*, 387–395.
8. FRERICHS, J. B., GIBSON, T. E., and BARETA, J. Pyelonephritis with pseudoxanthomatous re-

action malacoplakia. *Cancer Seminar*, 1956, *2*, 12–14.

9. Finlay-Jones, L. R., Blackwell, J. B., and Papadimitrios, J. M. Malacoplakia of colon. *Am. J. Clin. Path.*, 1968, *50*, 320–329.
10. Gupta, R. K., Schuster, R. A., and Christian, W. D. Autopsy findings in unique case of malacoplakia: cyto-immunohistochemical study of Michaelis-Gutmann bodies. *A.M.A. Arch. Path.*, 1972, *93*, 42–48.
11. von Hansemann, D. Ueber Malacoplakie der Harnblase. *Virchow's Arch. Path. Anat.*, 1903, *173*, 302–308.
12. Haukohl, R. S., and Chinchinian, H. Malacoplakia of testicle. *Am. J. Clin. Path.*, 1958, *29*, 473–478.
13. Kolodny, G. M. X-ray Seminar #62. Ureteral dilatation with pyuria. *J.A.M.A.*, 1966, *197*, 577–578.
14. Loele, W. Ein Beitrag zur sog. Malacoplakie der Harnblase. *Beitr. path. Anat.*, 1910, *48*, 205–220.
15. Malek, R. S., Greene, L. F., and Farrow, G. M. Amyloidosis of urinary bladder. *Brit. J. Urol.*, 1971, *43*, 189–200.
16. McDonald, S., and Sewell, W. T. Malacoplakia of bladder and kidneys. *J. Path. & Bact.*, 1914, *18*, 306–318.
17. Melicow, M. M. Malacoplakia: report of case, review of literature. *J. Urol.*, 1957, *78*, 33–40.
18. Michaelis, L., and Gutmann, C. Ueber Einschlusse in Blasentumoren. *Ztshr.f. Klin.Med.*, 1902, *47*, 208–215.
19. Morison, J. E. Malacoplakia of urinary bladder. *J. Path. & Bact.*, 1944, *56*, 67–72.
20. Rao, N. R. Malacoplakia of broad ligament, inguinal region and endometrium. *A.M.A. Arch. Path.*, 1969, *88*, 85–88.
21. Schneiderman, C., and Simon, M. A. Malacoplakia of urinary tract. *J. Urol.*, 1968, *100*, 694–698.
22. Scott, E. V. A., and Scott, W. F. Fatal case of malacoplakia of urinary tract. *J. Urol.*, 1958, *79*, 52–56.
23. Silber, I., and McAlister, W. H. Longitudinal folds as indirect sign of vesicoureteral reflux. *J. Urol.*, 1970, *103*, 89–81.
24. Smith, B. H. Malacoplakia of urinary tract—study of 24 cases. *Am. J. Clin. Path.*, 1965, *43*, 409–417.
25. Terner, J. Y., and Lattes, R. Malacoplakia of colon and retroperitoneum. *Am. J. Clin. Path.*, 1965, *44*, 20–31.
26. Waisman, J., and Rampton, J. B. Malacoplakia of testes and epididymis. *A.M.A. Arch. Path.*, 1968, *86*, 431–437.
27. Yunis, E. J., Estevez, J. M., Pinzon, G. J., and Moran, T. J. Malacoplakia; discussion of pathogenesis and report of three cases including one of fatal gastric and colonic involvement. *A.M.A. Arch. Path.*, 1967, *83*, 180–187.

COWPER'S GLANDS IN THE URETHROGRAM*

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THE bulbourethral or Cowper's glands[1,3,7,9,11,12] are a pair of pea-sized bodies situated behind and on each side of the membranous urethra between the 2 fascial layers of the urogenital membrane among the fibers of the membranous urethral sphincter (Fig. 1). They are of the compound tubulo-alveolar type and are made up of a number of lobules, each drained by a ductule. The lobular ductules promptly unite to form 2 single main ducts, 1 for each gland. The 2 main ducts are directed forward and slightly medially, and in their rather long course—3 to 4 cm. in the adult—they first pierce the inferior layer of the urogenital membrane and then continue for 1 to 1.5 cm. through the substance of the bulb of the corpus spongiosum. They then enter the ventral wall of the perineal bulbous urethra, where they course submucosally for 2 to 2.5 cm. to exit in the urethra just in front of the triangular ligament by 2 very small openings located one on each side of the midline. The bulbourethral glands arise during the 9th week of fetal life as 2 epithelial sprouts from the posterolateral wall of the urogenital sinus at the beginning of the future anterior urethra. They are the homologue of the Bartholin's glands in the female, and are considered as accessory male sex glands. During ejaculation they discharge a clear fluid which acts as a lubricant and as a vehicle for the spermatozoa, and which is said to contribute to semen coagulation.

Opacification of these structures by voiding or retrograde urethrography is an uncommon finding, even during forceful retrograde filling of the urethra. Only a few references to this phenomenon could be found in the literature. Some of the cases are recorded under the heading of Cowper's ducts and glands, others are classified as congenital urethral diverticula.

Brodney and Robins[2] reported an instance of opacification of an infected right Cowper's duct and gland by retrograde urethrography in an adult, with persistence of the contrast material (lipiodol) within the channel for 4 years. Gaca,[5] and Hodson and Edwards[6] each published an excellent example of urethrographic opacification of the left Cowper duct and gland in adult males who had a more distal urethral stricture. Two other cases of opacification of Cowper's ducts, but not of their glands, also in adults with a more distal urethral stricture, have been illustrated.[10,13]

In a urethrographic study of "congenital urethral diverticula," Edling[4] reported on 24 patients, mostly children, who had a channel or pouch originating from the floor of the bulbous urethra which he interpreted as dilated Cowper's ducts and glands. He divided the lesions into 2 main groups with

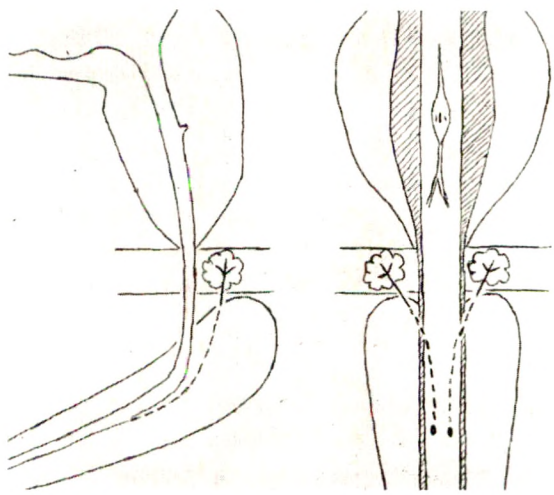


FIG. 1. Diagram of the location of the normal Cowper glands and ducts, lateral and anteroposterior aspect.

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transitional forms: the *globular type* consisting of rounded or oval cavities up to 3×6 cm. in size, originating from the ventral surface of the bulbous urethra commonly as a wide neck; and the *tubular type* consisting of tubular structures or channels originating from the same area of the urethra and coursing backward directly below and parallel to the urethra. Most of the channels were located on either side of the midline. Some were only slightly larger than normal Cowper's ducts, while others reached considerable size. Kjellberg *et al.*,[8] in a discussion of urethral diverticula, described 7 children with urethrographic findings similar to those reported by Edling[4] and came to the same conclusion as to etiology. In addition, both Edling[4] and Kjellberg *et al.*[8] suggested that most of the so-called diverticula originating from the ventral surface of the bulbar urethra probably represent dilated Cowper's ducts and glands, usually on a congenital basis. The same opinion is shared by Williams and Retik[14] in a more recent paper on congenital diverticula of the urethra, where some observations similar to those reported by Edling[4] and by Kjellberg *et al.*[8] are described.

To the above reported material we wish to add 10 similar examples of opacification of Cowper's ducts discovered as an incidental finding during routine voiding cystourethrography. These patients will be described and illustrated in order of increasing size of the urethral "diverticulum."

REPORT OF CASES

CASE I. J.B., a 7½ year old male, had a long history of recurrent urinary tract infections. He underwent a complete urologic survey including excretory urography, voiding cystourethrography and cystourethroscopy. No abnormalities were demonstrated, except for minimal reflux into the lower left ureter and the urethrographic findings shown in Figure 2*A*. Identical findings were observed 1 year later.

CASE II. D.K., a 5 year old male, was investigated because of persistent enuresis. A urologic work-up including excretory urography, voiding cystourethrography and cystourethroscopy showed nothing unusual, except for the urographic findings illustrated in Figure 2*B*.

CASE III. M.W., a 13 year old male, was investigated following an episode of hematuria. All the findings were within normal limits, except for the urethrogram shown in Figure 2*C*.

CASE IV. A.S., a 7 year old male, was investigated for persistent enuresis with day-time frequency and urgency. A urologic work-up, including urinalysis, excretory urography, voiding cystourethrography and cystourethroscopy showed no abnormalities, except for right vesicoureteral reflux, a patulous right ureteral orifice and the urethrographic finding illustrated in Figure 2*D*.

CASE V. J.N., a 6½ year old male investigated because of persistent enuresis, had a normal excretory urogram, voiding cystourethrogram and cystourethroscopic findings, except for the changes illustrated in Figure 2*E*. Identical findings were demonstrated 1 year later.

CASE VI. E.L.P., a 31 year old male with many episodes of epididymitis in the past, was found to have a retrovesical mass and a nonfunctioning left kidney by excretory urography. A voiding cystourethrogram showed no abnormalities, except for the findings illustrated in Figure 2*F*. Further investigation and surgical exploration disclosed a dysplastic left kidney with the left ureter drained ectopically into the left seminal vesicle.

CASE VII. K.T., a 10½ year old male, underwent a urologic investigation because of enuresis. All of the results were within normal limits, except for the urethrographic findings shown in Figure 2*G*.

CASE VIII. R.M., a 7½ year old male, was admitted to this hospital because of cervical lymphadenopathy of 3 weeks' duration which was subsequently found to be due to Hodgkin's disease. The excretory urogram showed some deviation of the ureters due to enlarged paraaortic lymph nodes, and a voiding cystourethrogram was normal, except for the findings illustrated in Figure 2*H*. A voiding cystourethrogram 2 years later showed similar findings.

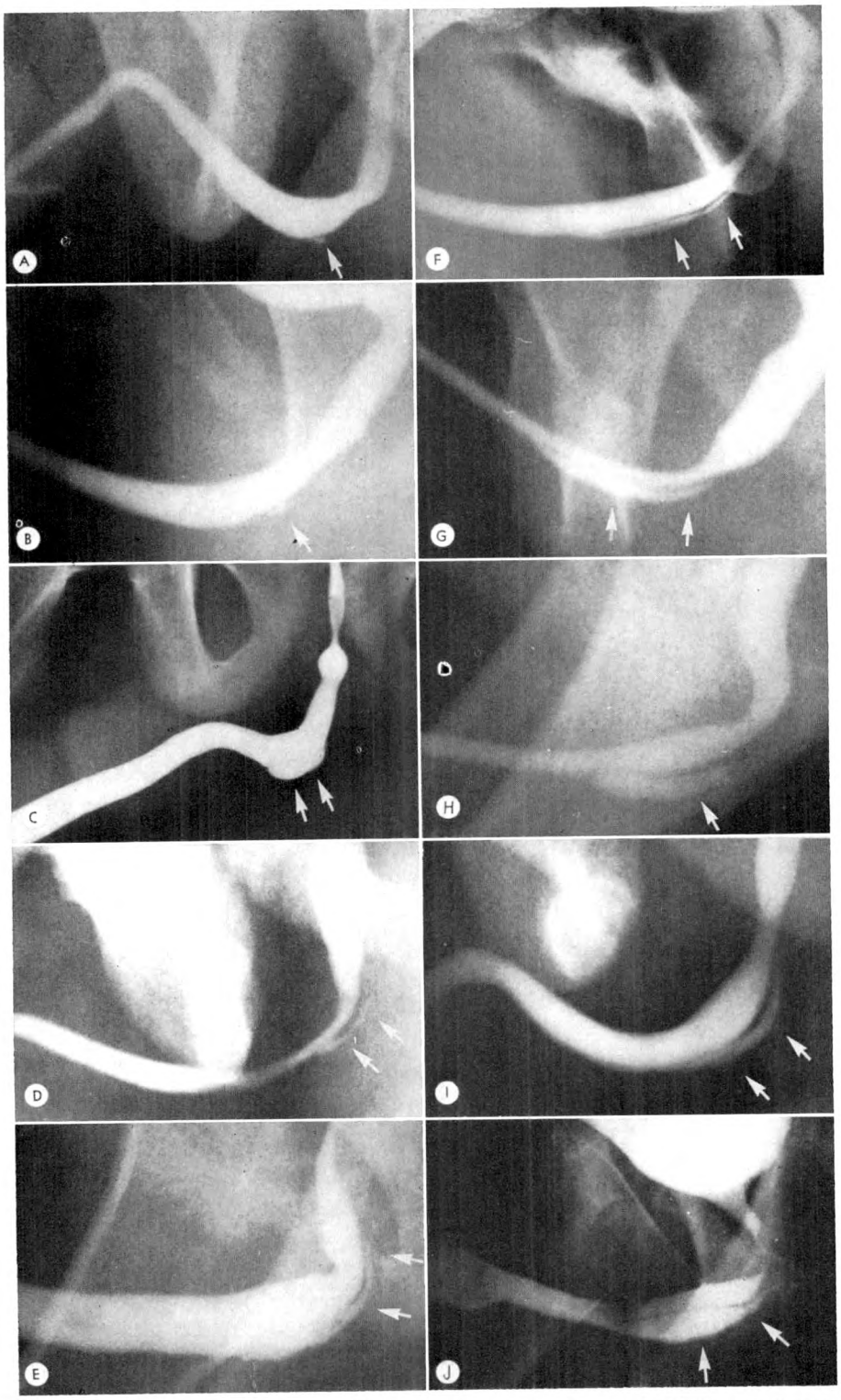


FIG. 2. (*A–J*) Urethrograms of Cases I–X. The arrows point to the urethral diverticula interpreted as dilated ducts of Cowper's glands.

Case IX. P.B., a 10½ year old male, has had recurrent urinary tract infections during the preceding 3 years. A urologic investigation including excretory urography, a cystourethrography and cystourethroscopy, showed decreased renal function bilaterally, considerable bilateral hydronephrosis and hydroureter, and trabeculated bladder without vesicoureteral reflux or lower urinary tract obstruction, plus the urethrographic findings illustrated in Figure 2*I*. A neurologic examination and myelography showed normal results.

Case X. G.C. was first seen in this institution at the age of 15½ years for a left orchiopexy. He was known to have a neurogenic bladder secondary to a myelomeningocele for which he had worn a condom catheter for several years. A urologic survey prior to the orchiopexy revealed a normal renal function and normal upper tracts by excretory urography. The voiding cystourethrogram showed a mildly trabeculated bladder without ureteral reflux, moderate spasm of the external sphincter, a wide neck diverticulum in the ventral surface of the scrotal urethra and an additional diverticulum-like channel originating off the ventral surface of the bulbous urethra (Fig. 2*J*). Identical findings were demonstrated 1 year later.

DISCUSSION

This series of patients includes 10 children ranging from 5 to 15½ years of age, and a 31 year old man. In all instances the voiding cystourethrogram showed a similar diverticulum-like structure originating from the bulbous urethra. All the pouches were of the tubular type, when seen in the lateral projection. In the oblique views of the urethra they generally tended to appear more globular due to foreshortening. No true globular cavities similar to those described by previous authors[4,8,14] were encountered. The channels originated from the ventral surface of the perineal portion of the bulbous urethra, but not all from the same level. Their length varied from a few millimeters to 6 cm. and their width from 1 mm. to 6–8 mm. They coursed backward and very close to the undersurface of the urethra. In 6 cases they extended backward and around the bulb as far as what was thought to be the urogenital diaphragm.

Although the channels apparently ended in a branched duct system in 2 instances, in no case was a definite opacification of glandular parenchyma demonstrated.

In 5 patients, additional anteroposterior or very slightly oblique views of the urethra were obtained, demonstrating a single channel in 3 patients (2 were on the left and 1, the largest of all, was in the midline), and a double tract in another patient (1 tract on each side of the midline).

Retrograde urethrography was performed in 5 patients with essentially the same findings, although the channel was seen less advantageously with this method apparently because of overdistention of the anterior urethra.

A cystourethroscopy was performed in 6 patients and in all cases the urethra was found to be "normal." The openings of the ducts were not visualized, even when a special search for them was made, except in 2 patients in whom they were described as dilated.

The finding under discussion was thought to be unrelated to the presenting clinical problem and to be of no clinical importance at this time.

Although the nature of these channels is not absolutely certain, their site of origin and course suggest that they may represent dilated Cowper's ducts. (In 3 instances the channels were very large and appeared to be in the midline relative to the urethra, but this position can be explained by the size of the structures.) Because of the age of the patients and absence of a more distal urethral stricture or actual urethral infection, the anomaly is presumed to have been of congenital origin with the exception perhaps of Case x who had worn a condom catheter for several years because of his neurogenic bladder.

Other causes are thought to be improbable. Partial urethral duplication is an unlikely possibility in that this anomaly occurs characteristically on the dorsal aspect

of the normal urethra. A persistence of Müllerian duct derivative or male vagina is also unlikely, as the channels in these patients originated more distally than might be expected of a vaginal primordium, as already pointed out by Kjellberg *et al.*[8] In addition, none of the patients had hypospadias or ambiguous genitalia. An incomplete bulborectal fistula does not occur in the absence of imperforate anus, or is extremely rare. Contraindicating an acquired fistulous tract is the absence of previous history of trauma or instrumentation in most cases. A fortuitous extravasation of contrast medium in the corpus spongiosum or between the urethra and the corpus spongiosum is not expected to assume a smooth tubular configuration, as was observed here.

CONCLUSION AND SUMMARY

The normal anatomy of the bulbourethral glands and ducts and previous roentgenographic demonstrations of these structures by urethrography are reviewed.

Ten additional instances of this uncommon finding are reported, without an obvious acquired cause in most cases.

From this and previous studies, it appears that the finding may result from urethral strictures or infections or, more commonly, from a congenital dilatation of the ducts.

The clinical significance of the anomaly seems negligible, except perhaps in more advanced cases where stone and infection could conceivably complicate the picture.

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REFERENCES

1. AREY, L. B. Developmental Anatomy: A Textbook and Laboratory Manual of Embryology. Seventh edition. W. B. Saunders Company, Philadelphia, 1965.
2. BRODNEY, M. L., and ROBINS, S. A. Urethrography in bulbo-urethral adenitis. *J. Urol.*, 1940, *43*, 844–850.
3. Editorial: Cowper's glands. *Urol. & Cutan. Rev.*, 1938, *42*, 231–232.
4. EDLING, N. P. Radiologic appearances of diverticula of male cavernous urethra. *Acta radiol.*, 1953, *40*, 1–8.
5. GACA, A. Die Cowperische Drüse im Röntgenbild. *Urol. internat.*, 1962, *14*, 125–127.
6. HODSON, C. J., and EDWARDS, D. In: A Text-Book of X-Ray Diagnosis by British Authors. Edited by S. C. Shank and P. Kerley. Fourth edition. Volume 5. W. B. Saunders Company, Philadelphia, 1970, pp. 507.
7. JOHNSON, F. P. Later development of urethra in male. *J. Urol.*, 1920, *4*, 447–502.
8. KJELLBERG, S. R., ERICSSON, N. O., and RUDHE, U. The Lower Urinary Tract in Childhood: Some Correlated Clinical and Roentgenologic Observations. Year Book Publishers, Inc., Chicago, 1957, pp. 273.
9. LOWSLEY, O. S., and KIRWIN, T. J. Clinical Urology. Third edition. Williams & Wilkins Company, Baltimore, 1956, pp. 280.
10. NEY, C., and FRIEDENBERG, R. M. Radiographic Atlas of the Genitourinary System. J. B. Lippincott Company, Philadelphia, 1966, pp. 618 and 644.
11. PATTEN, B. M. Human Embryology. Third edition. Blakiston Company, New York, 1968.
12. TESTUT, L., and LATARJET, A. Traité d'Anatomie Humaine. Ninth edition. Volume 5. Gaston Doin et Cie, Paris, 1949, pp. 380.
13. VOGLER, E. In: Roentgen Diagnosis. Second American edition. Volume 5. Edited by H. R. Schinz, W. E. Baensch, W. Frommhold, R. Glaumer, E. Uehlinger and J. Wellauer. Grune & Stratton, Inc., New York, 1967, pp. 703.
14. WILLIAMS, D. I., and RETIK, A. Congenital valves and diverticula of anterior urethra. *Brit. J. Urol.*, 1969, *41*, 228–234.

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CORRELATION OF ULTRASOUND AND RADIOISOTOPE PLACENTOGRAPHY*

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ACCURATE localization of the placenta is of considerable importance in the management of obstetric patients with third trimester bleeding. In addition, information may be provided to assist in the choice of an optimal site for aminocentesis. Two of the easily applied placentography techniques which are currently available employ the use of radioisotopes[8] and ultrasound.[2,6]

The purpose of this study is to compare the results of these 2 methods with each other as well as the findings at cesarean section.

METHOD

A group of 41 patients were examined by both ultrasound and radioisotope placentography techniques for this prospective study. Twenty-one of these were referred to obtain information relative to a specific clinical situation, predominantly third trimester bleeding. The remaining 20 patients were scheduled for elective cesarean sections and received both examinations strictly for the purpose of the study.

The B-scan ultrasound examinations were performed with a synchronized manual scanner using a 2 MHz transducer. This was applied to the patients' abdomen and scanned with a compound arc-sector motion. The image was displayed on a storage type of oscilloscope and photographed for a permanent record. Throughout the examinations the patients were maintained in a supine position with their heads slightly elevated. Multiple scans were obtained in each patient in both the longitudinal and transverse directions with appropriate angulation from the scanning axis to optimally display the internal interface of the placenta.

TABLE I

CORRELATION OF ULTRASOUND WITH RADIOISOTOPE PLACENTA SCANS

(41 patients)

	High-Mid-Low	Anterior-Posterior	Right-Left	Total
Good	35	34	34	103
Fair	6	5	7	18
Poor	0	2	0	2

The radioisotope scannings were performed with 2.0–4.0 millicuries In^{113m} bound to serum transferrin,[4,5] except for an occasional early case in the series in which Tc^{99m} labeled human serum albumen was used. The patients were examined by a dual probe rectilinear scanner using 8 inch crystals in both the supine and lateral positions. Therefore, in each patient 4 views of the abdomen and pelvis were obtained (right and left lateral, anterior and posterior).

RESULTS

The 2 groups of scans were evaluated independently of each other. The parameters selected correlated the cranial-caudad position of the placenta relative to the uterus (scored as High, Mid, or Low), and the axial position (scored as predominantly Anterior, Posterior, Right or Left). The correlation was considered as "Good" if the parameters were scored in the same manner; "Fair" if adjacent regions were submitted (*i.e.*, High *vs.* Mid); and "Poor" if there was a greater divergence (Table 1). A well correlated example is illustrated in Figure 1, *A–D*.

In the 20 cases with information from cesarean sections, the ultrasound correla-

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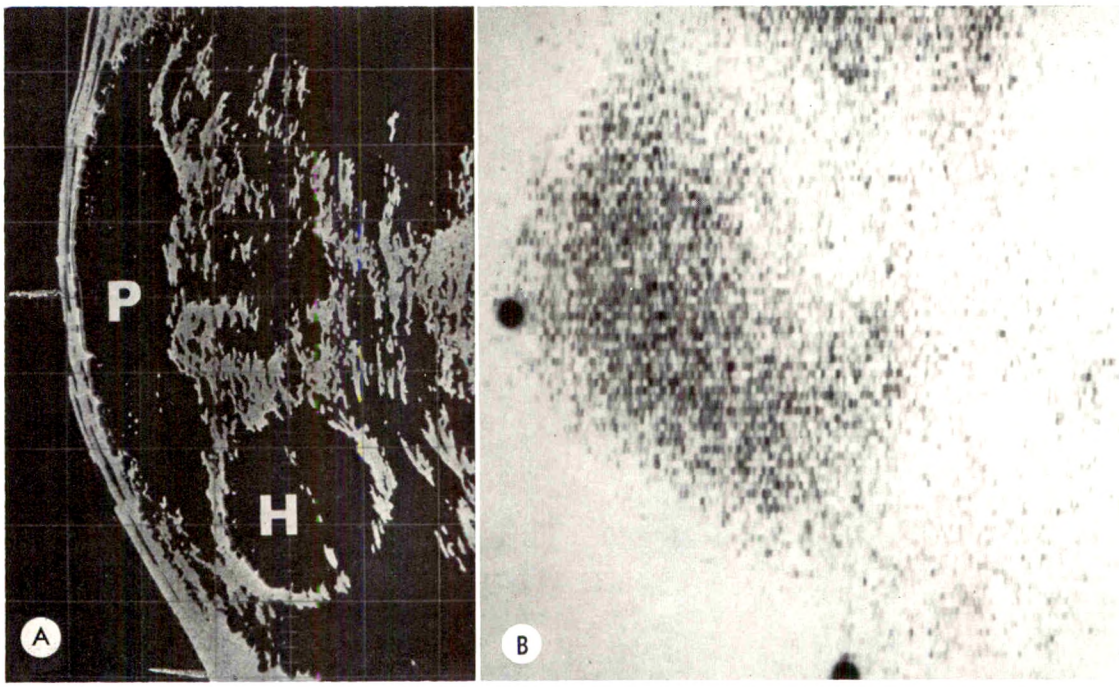


FIG. 1. (*A*) A midline, sagittal plane ultrasonogram is oriented in a manner analogous to the (*B*) right lateral radioisotope placentagram. The levels of the umbilicus and symphysis are indicated by the horizontal lines and large dots respectively. The placenta (P) is well displayed in both examinations. In *A* however, the inferior margin is more precisely defined and can be related to the presenting fetal head (H).

tion was slightly better than the radioisotope (Table II). In the instance in which it did not correlate, the ultrasound examination failed to identify an accessory lobe. This was detected in the radioisotope scan. Of the 5 cases where the radioisotope scan did not conform to the surgical findings, 2 were discrepancies in superior-inferior location, and 3 in the axial location. By our criteria these would all have been scored as "Fair" correlations.

The over-all group included 5 proved placenta previas. These were all indicated by the ultrasound examination, although the degree of previa was not accurately stated in 2 instances. In 1 case the possibility of previa was suggested, but not substantiated on a double set-up vaginal examination. The patient subsequently had an uneventful delivery.

The radioisotope examinations identified 4 of these placenta previas, 1 of which varied in degree with the surgical findings. The placenta previa which was missed was identified on the ultrasound study. Another was suggested, but not verified, by vaginal examination or the subsequent

TABLE II

A. CORRELATIONS OF ULTRASOUND AND RADIOISOTOPE PLACENTA SCANS WITH FINDINGS AT CESAREAN SECTION
(20 patients)

Good correlation with ultrasound	19 patients
Good correlation with radioisotopes	15 patients
Good correlation with both	14 patients

B. CORRELATIONS WITH CESAREAN SECTION WHEN ULTRASOUND AND RADIOISOTOPE SCAN CORRELATED FAIR OR POOR WITH EACH OTHER IN AT LEAST ONE PARAMETER
(8 patients)

Ultrasound correlates better	5 patients
Radioisotopes correlate better	1 patient
Specific information not given for parameter in question	2 patients

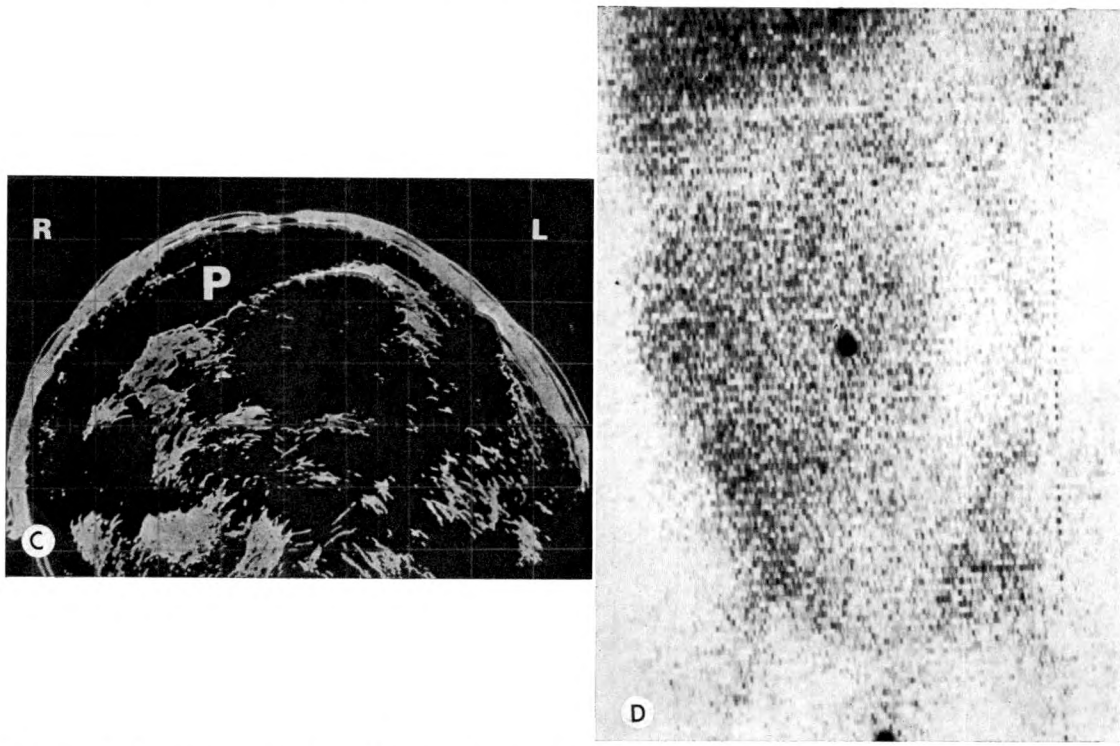


Fig. 1. (*C*) A transverse section ultrasonogram at the level of the umbilicus as well as the (*D*) anterior radioisotope scan further localize the placenta toward the right side of the uterus.

clinical course. The single false positives that occurred, respectively, with each type of scan involved different patients.

DISCUSSION

Both types of placentography generally correlated well with each other as well as the observations at cesarean section. The slightly better results with ultrasound could be attributed to the more precise definition of the edges of the placenta and the concurrent visualization of the presenting part, thereby allowing the sometimes critical relationships between these 2 structures to be observed. On the other hand, the diminishing blood pool at the tapering edge of the placenta creates a gradient of scintillation activity which blends with that of the uterine wall on the radioisotope scan. Therefore, although the main portion of the placenta is well displayed, the placenta edge is not clearly delineated. This difficulty can be intensified when pressure from the presenting part in the pelvis displaces additional blood from the placenta and further diminishes that portion which is detectable. This was well demonstrated in 1 of our patients.

An additional advantage of the ultrasound over the radioisotope method is that it employs no radiation. Although the dosage to the fetus is small with currently used radioisotopes, even this should be avoided[10] if an alternative method with at least comparable accuracy is available. Energy is also transmitted to the patients by diagnostic ultrasound, but ionization does not occur and, at the extremely low levels delivered by available pulsed systems, neither somatic nor teratogenic effects have been detected.[3] However, since the possibility of some as yet unknown harmful effect cannot be completely discounted, continued investigation should be pursued.

Some of the axial discrepancies of the radioisotope scan, which are of significance

only in the consideration of a site for amniocentesis, are probably related to the fact that the lateral views are obtained with the patient lying on the side by necessity of the instrument design. This would predispose to some rotation of the uterus that might not be present during the supine ultrasound examination or cesarean section.

Our findings are in general agreement with those of the large comparative series performed at University College Hospital in London,[2,7,11] where Tc^{99m} HSA was used for their radioisotope placentograms and a contact scanner for the ultrasound studies. By performing the latter examination with a fluid distended bladder, these investigators felt that they could identify the internal os of the cervix and thereby more precisely relate the inferior margin of a low lying placenta to it. Although both methods performed well, they favored the ultrasound technique because of some of its inherent advantages. A similar conclusion was reached by Aiers *et al.*[1] However, their radioisotope method did not employ a scan technique, but consisted of obtaining counts at multiple points plotted over the uterus. In a smaller comparative series in which the radioisotope scannings were performed with In^{113m}, Niehoff *et al.*[9] found a greater accuracy with the radioisotope, but a placenta previa missed with this method was detected on the ultrasound scan.

SUMMARY AND CONCLUSIONS

Ultrasound and radioisotope placenta scans were compared in a group of 41 patients, 20 of whom were also correlated with the results of cesarean section.

The discrepancies in localization between the two modalities were minimal.

The ultrasound, however, has the inherent advantages of more precise delineation of the placenta margin, the concurrent visualization of the presenting part, and the lack of radiation exposure. For these reasons it is favored as the primary method for placentography.

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REFERENCES

1. Aiers, M., Evered, D. C., and Smith, A. H. Placental localization by use of ^{132}I-human serum albumen and by ultrasonic scanning: comparative study. *J. Obst. & Gynaec. Brit. Commonwealth*, 1969, *76*, 220–224.
2. Campbell, S., and Kohorn, E. I. Placental localization by ultrasonic compound scanning. *J. Obst. & Gynaec. Brit. Commonwealth*, 1968, *75*, 1007–1013.
3. Freimanis, A. K. Biological effects of medically applied ultrasound and their causes. *Crit. Rev. Radiol. Sc.*, 1970, *1*.
4. Huddelston, J. E., Mishkin, F. S., Carter, J. E., Dubois, P. D., and Reese, I. C. Placenta localization by scanning with indium 113m. *Radiology*, 1969, *92*, 587–590.
5. Johnson, P. M., Chao, S., and Goodwin, P. N. Initial evaluation of indium 113m as agent for blood pool imaging. *Radiology*, 1969, *92*, 625–626.
6. Kobayashi, M., Hellman, L. M., and Fillisti, L. Placenta localization by ultrasound. *Am. J. Obst. & Gynec.*, 1970, *106*, 279–285.
7. Kohorn, E. I., Secker Walker, R. H., Morrison, J., and Campbell, S. Placental localization. *Am. J. Obst. & Gynec.*, 1969, *103*, 868–877.
8. Nelp, W. B., and Larson, S. M. Placenta. In: Clinical Scintillation Scanning. Edited by L. M. Freeman and P. M. Johnson.
9. Niehoff, R. D., Hendee, W. R., and Brown, D. W. Placenta scanning with ^{113m}In. *J. Nuclear Med.*, 1970, *11*, 15–18.
10. Schwarz, G. S. Radiation hazards to human fetus in present-day society. *Bull. New York Acad. Med.*, 1968, *44*.
11. Secker Walker, R. H., Kohorn, E. I., and Morrison, J. Placental localization by isotope and ultrasound scanning. *Proc. Roy. Soc. Med.*, 1969, *62*, 446–448.

THE WIDENED LOOP SIGN OF LIPPE'S LOOP PERFORATIONS*

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PERFORATION of the uterus, although one of the least common complications associated with the use of intrauterine contraceptive devices, is one of the most serious. The incidence of perforation by the Lippe's loop has been reported as 1 per 1,000 to 1 per 2,500.[6,10] Plain film roentgenography has previously been discounted as a means for identifying the extrauterine position of the device.[3,5,9]

This paper presents a new sign for determining perforation by a Lippe's loop without resorting to contrast studies.

REPORT OF CASES

CASE I. S.C., a 28 year old white female, gravida II, para II, was admitted to the hospital on August 29, 1971. She had had a Lippe's loop inserted on August 15, 1971, but on a routine pelvic examination 2 weeks later, no strings from the intrauterine device (IUD) were seen. The patient was asymptomatic and had no physical signs. On plain film roentgenographic examination of the pelvis, the IUD was in the vicinity of the uterus. The loops of the IUD were widely separated, with the bulbous tip approximately 1 cm. from the second loop. A hysterosalpingogram confirmed the extrauterine location of the device (Fig. 1). A transabdominal surgical approach was initially considered, until a pelvic pneumogram showed the loop deep in the cul-de-sac (Fig. 2). A posterior colpotomy was performed instead, and the loop removed without difficulty.

Comment. The patient was asymptomatic and a diagnosis of extrauterine IUD was made on the plain film roentgenogram and confirmed by a hysterosalpingogram. In this case, the surgical approach was aided by precise localization of the device by means of a pelvic pneumogram.

CASE II. S.R., a 20 year old white female, gravida I, para I, was admitted to the hospital on September 2, 1971. She had had an IUD inserted on June 1, 1971 and 5 months later, on a routine visit, no strings from the IUD were seen. Roentgenogram of the pelvis showed a widened Lippe's loop in the region of the uterus and a hysterosalpingogram revealed the loop to be extrauterine in location (Fig. 3; and 4). A posterior colpotomy approach for removal of the device was considered, but rejected when an intravenous pyelogram showed the IUD to be anterior to the bladder (Fig. 5). Therefore, an exploratory laparotomy was performed and the extrauterine IUD removed.

Comment. The patient was asymptomatic and the roentgenogram of the pelvis showed the loop to be in the region of the uterus. However, the widening of the loop was presumptive evidence of perforation and, therefore, the hysterosalpingogram was obtained to definitely document this. Once again the surgical approach was aided by precise roentgenographic

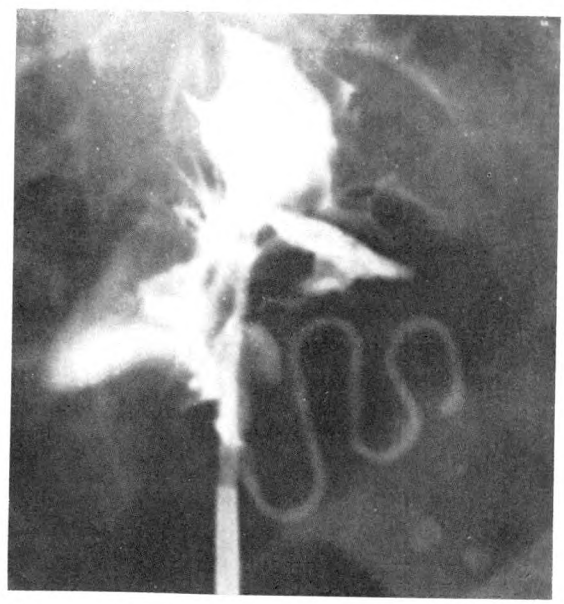


FIG. 1. Case I. Hysterosalpingogram shows the widened Lippe's loop, with the bulbous tip separated by 1 cm. from the second loop, to be clearly extrauterine.

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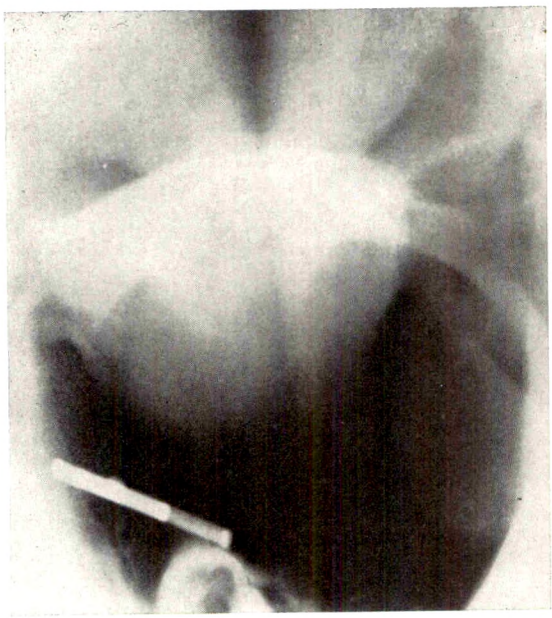


Fig. 2. Case I. Pelvic pneumogram also shows the intrauterine device (IUD) to be in an extrauterine position, lying in the cul-de-sac.

localization, in this case by an intravenous pyelogram showing the loop to be anterior to the bladder and therefore requiring a laparotomy to remove it.

Case III. P.G., a 37 year old white female, gravida VI, para VI, was admitted to the hospital on September 16, 1970. She had had an IUD inserted on a postpartum check-up and, 15 months later, no strings from the IUD were seen on a routine examination. The patient was asymptomatic. The roentgenogram of the pelvis showed a widened Lippe's loop lying in a horizontal plane in the region of the uterus (Fig. 6). The extrauterine location was confirmed with a hysterosalpingogram (Fig. 7). An exploratory laparotomy was performed and the extrauterine IUD was removed.

Comment. The patient was asymptomatic and the widened Lippe's loop as well as its horizontal orientation on the roentgenogram of the pelvis suggested a uterine perforation, proven by a hysterosalpingogram and subsequent surgery.

Case IV. N.M., a 17 year old white female, gravida I, para I, was admitted to the hospital on September 15, 1971. She had had an IUD inserted in December 1970, but on a routine check-up no strings from the IUD were found. The device was presumed to have been expelled from the uterus and another was inserted. The patient had slight bleeding from the IUD, which was then removed, and she was placed on birth control pills. She had back pain for several months and was seen in the Orthopedic Department, where a roentgenogram of the pelvis revealed a widened Lippe's loop in the vicinity of the uterus. On the hysterosalpingogram, this was seen to lie in an extrauterine position. A posterior colpotomy was performed and the IUD was removed.

Comment. The patient had several months of back pain, the only 1 of our 4 cases to have any symptoms. When the second IUD was inserted, presumably no roentgenograms were obtained. This suggests the necessity of taking routine roentgenograms after presumed IUD expulsion, to rule out the possibility of the IUD lying within the peritoneal cavity in an extrauterine position.

DISCUSSION

Perforation of the uterus by the Lippe's loop, seen in 1 per 1,000 to 1 per 2,500 cases, is a potentially serious complication associated with the use of the intrauterine contraceptive device. The perforation may be partial or complete. With the far more common complete perforation, the device is



Fig. 3. Case II. There is a markedly widened Lippe's loop in the vicinity of the uterus on this early hysterosalpingogram.

fully extruded into the peritoneal cavity, while in the partial perforation, a portion is embedded within the uterine wall. Most authors state that perforations occur at the time of insertion and that the device is placed directly into the peritoneal cavity.[3,7] The hypothesis of erosion of the uterine wall has been discounted by most observers. This was previously based on the fact that the devices were confirmed to be *in utero* by a plain roentgenogram of the pelvis and were later proven to have caused partial perforations. These perforations were most likely present at the initial examination and a contrast study at that time would have proven this fact.[3]

Perforation at the time of insertion is often asymptomatic and unrecognized.[5,7,9] One of the early symptoms of uterine perforation is marked abdominal pain immediately after the insertion. There is a high incidence of abdominal cramps after normal insertion of the device, but this finding should lead to an examination to determine the exact position of the device. Postinsertion pregnancy, with no history of expulsion of the device, has also been a frequent sequel to perforation.[5]

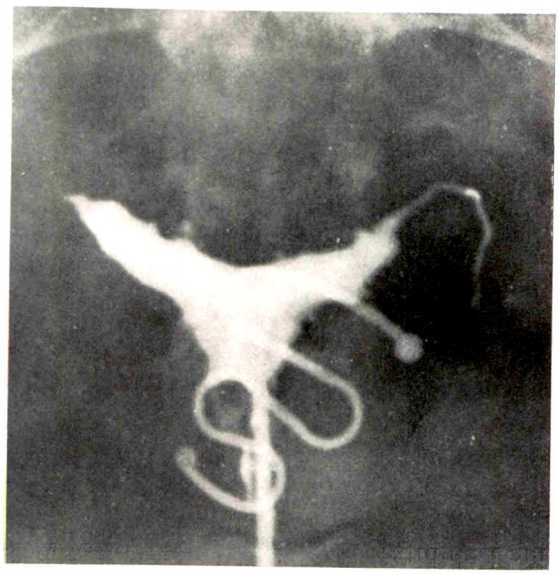


Fig. 4. Case II. The uterus is well filled with contrast material and the IUD with widened loop is clearly seen to have perforated.

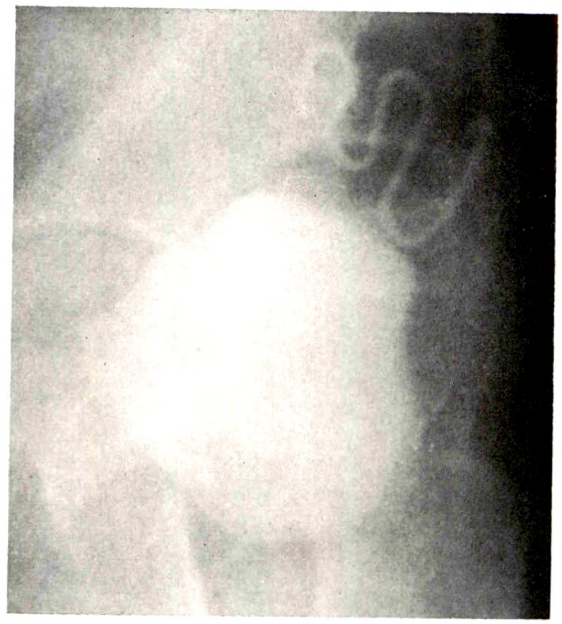


Fig. 5. Case II. On the intravenous pyelogram, the IUD is seen to be anterior and superior to the contrast material filled bladder, thus requiring a transabdominal approach for its removal.

The plain film roentgenographic diagnosis of uterine perforation has been discounted by all authors. Indeed, one author claims that roentgenograms "fostered a false sense of complacency by showing the IUD to be in the vicinity of the uterus."[5] These authors state that hysterosalpingography or roentgenograms made with a metallic sound in the uterus is the only reliable way to diagnose perforation. In our series, plain anteroposterior roentgenograms of the pelvis, showing widening of the Lippe's loop, were sufficient to make a diagnosis of perforation. In each case, the findings were proven by hysterosalpingography and subsequent surgery. In addition, in reviewing illustrations of examples of Lippe's loop perforations previously published,[1,2,7] the same plain film roentgenographic findings were uniformly present. No false negative results were obtained in our series.

The uterus is a thick-walled muscular organ, approximately 7.5 cm. in length, 5 cm. in width, and nearly 2.5 cm. in thickness.[4] Therefore, the internal cavity of the

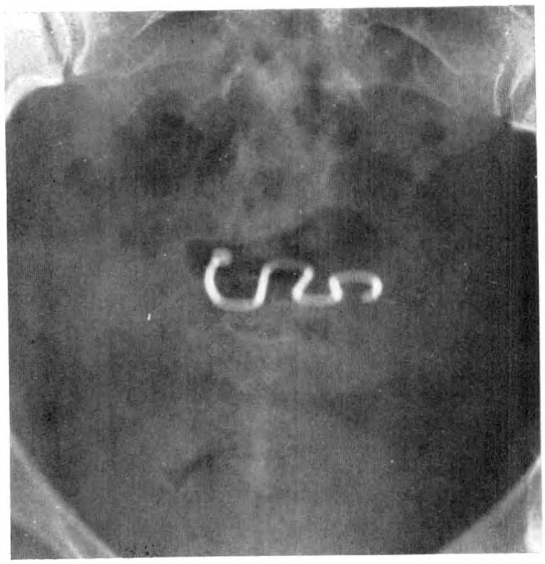


FIG. 6. Case III. The IUD is seen to be lying in a horizontal plane with widening of the loop on this plain roentgenogram of the pelvis.

uterus is quite small. While in the inserter, the Lippe's loop is completely straight. As it is ejected from the inserter and put into the patient, the intrauterine device takes its natural double S curve with the width of any given loop approximately 1 cm. However, the confines of the uterus are too small to accommodate this size, and thus the space between the loops is greatly diminished to 0.5 cm. or less. In our series of uterine perforations, the distance from the bulbous tip at the end of the loop to the second loop was approximately 1 cm. in every case. In a series of normal controls with IUDs known not to have perforated, the distance was usually 2 or 3 mm. and never greater than 5 mm. (Fig. 8, *A* and *B*). Therefore, we consider the widening of the Lippe's loop on a roentgenogram of the pelvis to imply perforation.

A hypothesis to account for this finding is suggested. In the intact uterus, the cavity is so small that it prevents the assumption of the natural widened shape of the loop. However, once the loop has perforated into the peritoneal cavity, it no longer has the restraint of the uterine cavity and can assume its "normal" state (Fig. 9). Thus the widened Lippe's loop should be taken as presumptive evidence of perforation and hysterosalpinography should be performed to definitely document this. If the loop is closed, this should be taken to imply that the IUD is within the uterine cavity.

Uterine perforation of the Lippe's loop can first be suspected by the widened appearance of the loop in a plain film roentgenographic examination. The extrauterine position is confirmed by hysterosalpingography. A pelvic pneumogram can be of assistance in determining whether the loop is situated deep in the cul-de-sac and, therefore, can be removed by posterior colpotomy. The pelvic pneumogram also has the capacity to demonstrate adherence or entanglements of the IUD with loops of bowel or mesentery in the cul-de-sac, although no such case was encountered in our limited series. In this instance, the relatively blind removal by posterior colpotomy would seem less desirable because of the possibility of iatrogenic trauma to adherent structures. The intravenous pyelogram can similarly localize the loop in relation to the bladder, and, if anterior, indicate a transabdominal approach for removal.

In 1 case, the IUD was presumed to be expelled, while it had actually perforated

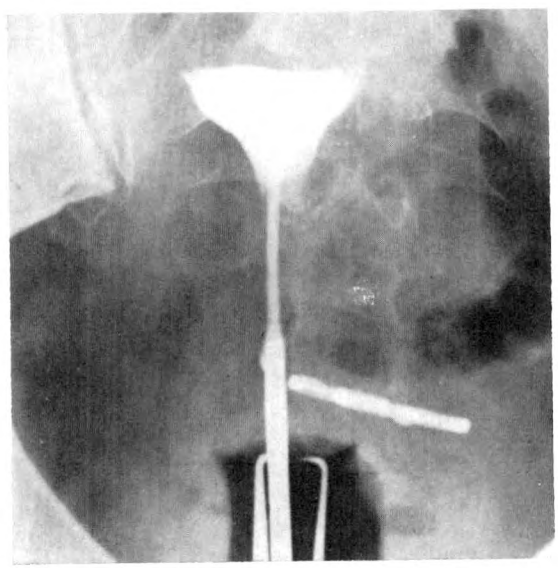


FIG. 7. Case III. With the uterus filled with contrast material, the loop is seen to be obviously extrauterine in location.

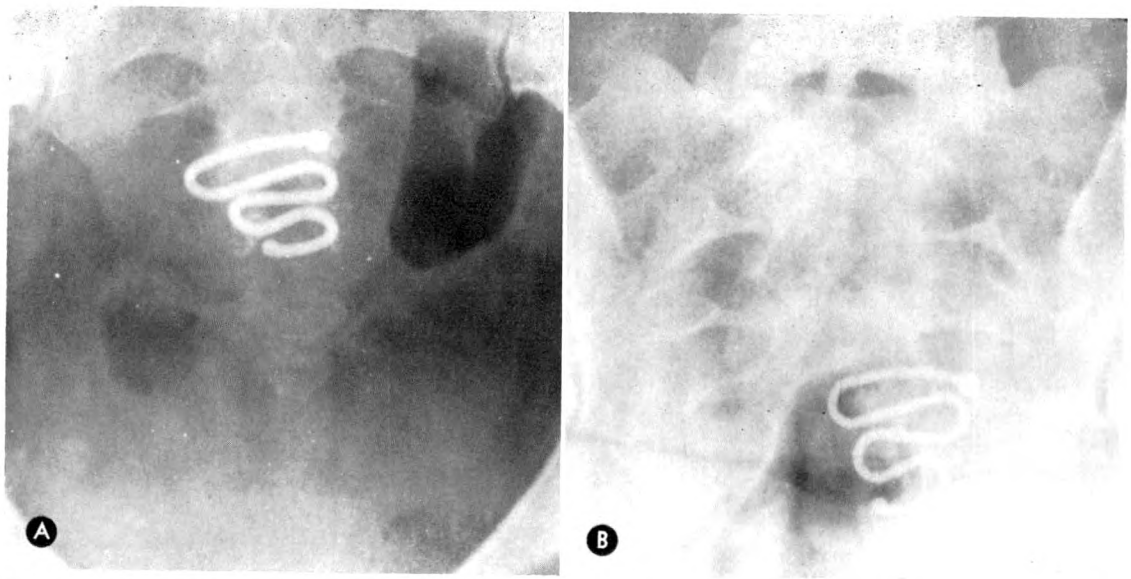


Fig. 8. (*A* and *B*) Two examples of well positioned Lippe's loops in patients who had roentgenograms of the pelvis for unrelated reasons. Note the compactness of the loop and the small distance between the bulbous tip and the second loop. Compare these with the previous examples of uterine perforations.

the uterus and was lying in the cul-de-sac. Therefore, routine roentgenograms should be taken after presumed IUD expulsion to rule out the possibility of the IUD lying within the peritoneal cavity in an extrauterine position.

SUMMARY

Four cases of perforation of the uterus by a Lippe's loop are reported along with normal controls.

A widened loop was seen in all cases of perforation and should be considered presumptive evidence of uterine perforation on the roentgenogram of the pelvis and documented by hysterosalpingography.

The value of pelvic pneumography and intravenous pyelography to assist in choosing the correct surgical approach is discussed.

Routine roentgenographic examination of the pelvis for suspected expulsion of the IUD is recommended.

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The author would like to express his appreciation to Doctors Robert Sabatelle and Salvatore Rini for use of their cases and Mrs. Mary Price for her secretarial work.

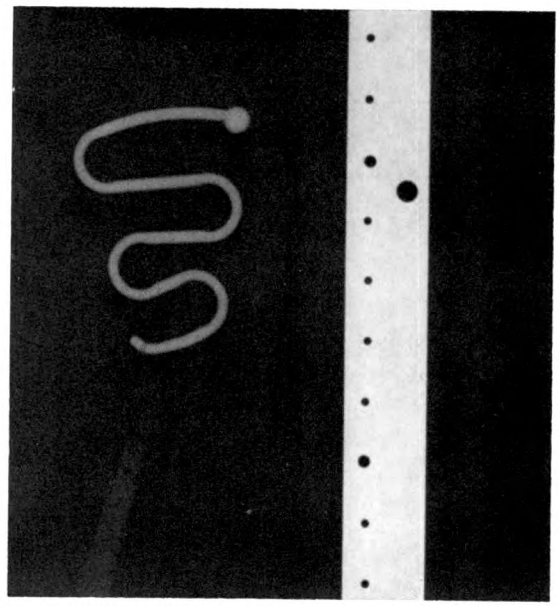


Fig. 9. This is an example of an IUD which has just been pushed through the inserter and is lying free. Note the 1 cm. distance between the bulbous tip and the second loop. This is an example of the "natural" shape of the IUD, also the appearance of the loop lying free in the peritoneal cavity when it is out of the confines of the uterine cavity.

REFERENCES

1. ALBERTS, P. S. Uterine perforation by Lippe's loop. *Obst. & Gynec.*, 1970, *36*, 164–166.
2. DHALL, K., DHALL, G. I., and GUPTA, B. B. Uterine perforation with Lippe's loop; detection by hysterography. *Obst. & Gynec.*, 1969, *34*, 266–270.
3. ESPOSITO, J. M. Perforation of uterus, secondary to insertion of IUD. *Obst. & Gynec.*, 1966, *28*, 799–805.
4. GRAY, H. Anatomy of the Human Body. Twenty-seventh edition. Lea & Febiger, Philadelphia, 1965.
5. HALL, R. E. Reappraisal of intrauterine contraceptive devices. *Am. J. Obst. & Gynec.*, 1967, *99*, 808–813.
6. HALL, R. E. Comparative evaluation of intrauterine contraceptive devices. *Am. J. Obst. & Gynec.*, 1966, *94*, 65–71.
7. LEDGER, W. J., and WILLSON, J. R. Intrauterine contraceptive devices: recognition and management of uterine perforations. *Obst. & Gynec.*, 1966, *28*, 806–811.
8. MCLEOD, P. A. Uterine perforation by IUD. *J.A.M.A.*, 1969, *210*, 728.
9. SHIMKIN, P. M., SIEGEL, H. A., and SEAMAN, W. B. Radiographic aspects of perforated intrauterine contraceptive devices. *Radiology*, 1969, *92*, 353–358.
10. TIETZE, C. Comparative Statistical Project of the Evaluation of Intrauterine Contraceptive Devices. 6th Progress Report, Part I, National Committee on Maternal Health, New York, December 31, 1965.

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Fifty-fifth Annual Meeting: Broadmoor Hotel, Colorado Springs, Colorado, April 22–26, 1973.

EDITORIAL

THE SEVENTY-THIRD ANNUAL MEETING OF THE AMERICAN ROENTGEN RAY SOCIETY

The Annual Meeting of the American Roentgen Ray Society was held for the eleventh time in beautiful Washington, the Nation's Capital, in our time the Center of the World. To quote the chronicler: "In every age, there has been one city which has seemed to be the Center of the World, which the Fates have chosen to be the Guardian for the hopes of all men, to hold and control their aspirations, to determine the probability of their glory, or their happiness, or their misery, their bondage or their freedom. That world city in our time is Washington."

The Seventy-third Annual Meeting was again held at the Washington Hilton Hotel from Tuesday, October 3, to Friday, October 6, 1972.

At 8:30 A.M. Tuesday, President Thomas M. Fullenlove of San Francisco, California, called the meeting to order. Immediately thereafter outgoing President Fullenlove, assisted by Dr. Sidney W. Nelson, Columbus, Ohio, Chairman of the Executive Council, installed Dr. J. Scott Dunbar, Vancouver, B. C., Canada, as the new President of the Society. In so doing, outgoing President Fullenlove presented to Dr. Dunbar the symbolic Pfahler gavel, fashioned from a mastodon's tooth recovered from Alaskan glacial ice, and the working Erskine gavel made from memorabilia of Röntgen, Coolidge and Snook. Concluding the ceremony, Dr. Fullenlove pinned on Dr. J. Scott Dunbar the traditional gold emblem of the Society engraved "President of the American Roentgen Ray Society."

President Dunbar, as his first function, remitted to Dr. Thomas M. Fullenlove the Past Presidents' Book, inscribed with his name and containing a list of all Past Presidents, as well as color photographs of the historic Pfahler and Erskine gavels. He then gave his scholarly Inaugural Address on "The Patient as a Person," a very timely subject, which is published *in extenso* as the leading article in this issue of the JOURNAL.

The First Scientific Session began at 8:55 A.M. Tuesday with President J. Scott Dunbar in the Chair. Paper 1 was the *Award Paper of the Society for Pediatric Radiology*, which was selected the preceding day by the Society for Pediatric Radiology as being of greatest interest to all radiologists. Its authors were Martin H. Reed, M.D., and N. Thorne Griscom, M.D., of Boston, Massachusetts and its title was "The Complexity of Hydrometrocolpos in Infancy." In this Award Paper the authors stated that in 25 infants with hydrometrocolpos the dilatation sometimes involved the vagina alone, rarely the uterus alone, and sometimes both organs. The lesions obstructing the genital tract included imperforate hymen, vaginal diaphragm, varying degrees of vaginal atresia, and atresia of the cervix. Sixteen of the patients had major congenital anomalies in addition to the genital obstruction. Imperforate anus with rectovaginal fistula, bicornuate uterus, and renal agenesis or hypoplasia were especially common. The authors concluded that the complexity of these abnormalities requires individualization of preoperative radiologic investigation. The merits of plain film roentgenograms in various positions, intravenous urography, cystography, genitography, and direct injection of the mass were discussed. The first 2 of these should be employed whenever hydrometrocolpos or any

other abdominal mass is suspected; the latter 3 often add to the preoperative anatomic delineation, which should be as complete as the child's condition allows. This Award Paper was followed by 12 additional papers, of which 4 dealt with pediatric subjects, 4 with gastrointestinal problems, 1 with urographic study, and 3 with therapeutic irradiation.

On Tuesday evening the Caldwell Lecture was given by Alexander R. Margulis, M.D., Professor and Chairman, Department of Radiology, University of California, San Francisco, California. The title of his Lecture was "Radiobiologic Lessons in Diagnostic Roentgenology." Doctor Margulis, who is one of the foremost Radiologists of the world, gave an excellent Lecture delivered with clarity and profusely illustrated.

Dr. Margulis was introduced by an equally world renowned Radiologist, Leo G. Rigler, M.D., Professor of Radiology, University of California Los Angeles, Los Angeles, California.

At the conclusion of the Lecture, President Dunbar presented to Dr. Margulis the Caldwell Gold Medal, the highest recognition of the Society for meritorious scientific achievement.

The Second Scientific Session was started at 8:30 A.M. Wednesday, with Dr. Benjamin Felson, Cincinnati, Ohio, First Vice-President, in the Chair. At this session 14 papers were given on various subjects. These subjects included: 7 papers on intrathoracic lesions; 2 papers on gastrointestinal lesions and 1 on positive contrast peritoneography; 2 papers on tomography; 1 paper on chronic temporomandibular joint syndrome; and 1 paper on the radiologic aspects of an abortion program.

Wednesday noon at the Twenty-Third Annual Past Presidents Luncheon, Dr. John P. Medelman, White Bear Lake, Minnesota, in the name of all living Past Presidents presented, in a standing ovation ceremony, to Clifford L. Sherratt, Honorary President of the Society, a Past Presidents' book, with the added special engraving: "Clifford L. Sherratt, Honorary President, American Roentgen Ray Society."

Wednesday at 1:30 P.M. the American College of Radiology, following tradition of the past, presented this year "Three Programs for Radiologists from the American College of Radiology." These programs were initiated in response to the expressed wishes of College members. Time had been allowed for questions and comments about these and other College programs. The Moderator was Seymour F. Ochsner, M.D., President, American College of Radiology. The 3 Programs were: College Accreditation Activities, by Stanley M. Wyman, M.D., Chairman, Committee on Accreditation, ACR; Medical Malpractice and Radiology, by J. Richard Raines, M.D., Chairman, Committee on Professional Liability, ACR; and Purposes and Results of the Continuing Education Series, by Milton Elkin, M.D., Chairman, Commission on Diagnostic Radiology, ACR.

The Third Scientific Session began Thursday morning at 8:30 A.M. with Dr. Sydney W. Nelson, Columbus, Ohio, Chairman of the Executive Council presiding. At this Session 13 papers were delivered. Paper 23 was the *Award Paper of the American Society of Neuroradiology*. Its authors were, P. Braunstein, M.D., I. Kricheff, M.D., J. Korein, M.D., K. Corey, Ph.D., and N. Chase, M.D., New York, New York, and its title was "A Simple Bedside Evaluation of Cerebral Blood Flow in the Study of Cerebral Death." In this Award Paper the authors stated that modern advances in medicine have created a need to identify total brain death in patients who maintain a heartbeat. The combinations of clinical and electroencephalographic criteria presently in use for making this assessment are in themselves unduly prolonged and unreliable. Absence of cerebral circulation has been demonstrated in such cases angiographically and by intracarotid injection of radionuclides; these techniques are, however, inappropriate for general application. The authors have shown the feasibility of demonstrating this cerebral circulatory def-

icit at the bedside, using a portable scintillation probe system to monitor the passage of an intravenously injected radioisotopic bolus through the head. There were 12 other papers on such varied topics as: 4 papers on renal subjects; 2 on nuclear medicine; 3 on blood and lymph vessels; 2 on mammography; and 1 on the hard palate.

On Thursday evening at 6:30 P.M. a Cocktail Party preceded the Annual Banquet which was held at 7:30 P.M. in the Hilton International Ballroom. At this Banquet, which always is the highlight of the Social Activities of the Annual Meeting, President Dunbar, after introducing the newly elected officers and their consorts, presented the greatly appreciated Awards to the Scientific Exhibit winners. This brief ceremony was followed by an elaborate floor show and a pleasant evening of dancing.

On Friday morning, the Fourth and last Scientific Session, which again was started at 8:30 A.M., was presided over by Dr. Robert N. Cooley, Galveston, Texas, 2nd Vice-President of the Society. At this Session 14 papers were presented. Paper 42 was the *Award Paper of The American Radium Society*. Its authors were Henry M. Lemon, M.D., F.A.C.P., Omaha, Nebraska, John F. Foley, M.D., Ph.D., Omaha, Nebraska, Frederick F. Paustian, M.D., Omaha, Nebraska, Anne Kessinger, M.D., Omaha, Nebraska, James Delevan, M.D., Grand Rapids, Michigan, and Charles W. McLaughlin, Jr., M.D., Omaha, Nebraska. Its title was, "Toward Improved Palliation of Pancreatic Carcinoma." The authors stated that pancreatic carcinoma is now the fourth commonest cause of cancer death in the U. S.; their study was based on 250 cases, comparing the surgical results with radiation and fluorouracil therapy. Abnormal blood loss measured by fecal excretion of Cr^{51} tagged red blood cells occurs in 90 per cent of patients, and as in other intestinal cancers, probably often precedes the morphologic changes observed by barium studies. A patient-operated toilet monitor automatically recording fecal radioactivity on a teletype print-out has been used for these studies and was described. After diagnosis by celiotomy, 4,000 rads to the pancreatic area combined with 12 mg./kg. fluorouracil weekly have resulted in a survival comparable to that seen after radical pancreatico-duodenectomy, with probable primary tumor eradications in 3 cases. The other papers included: 3 on radiation therapy; 2 on technology; 3 on gastrointestinal lesions; 2 on urographic problems; and 2 on rare bone and joint lesions; and the last paper was on the radiographic manifestations of pulmonary opportunistic infections.

The Section of Instruction had again Dr. Harold O. Peterson, with his long experience, as Director and Drs. Richard G. Lester and Seymour H. Levitt, as Associate Directors. They assembled an outstanding Faculty of distinguished Instructors who conducted the courses on the latest developments in radiology in a rather informal manner. There were 49 courses: 15 each on Tuesday, Wednesday and Thursday, and 10 on Friday. The arrangements for the courses was excellent and they attracted large audiences.

The Scientific Exhibits this year constituted the highlight of the Annual Meeting. A surprisingly large number complemented the Scientific Essays on the same subject. The Society is greatly indebted to Dr. James F. Martin and his able Committee for their untiring effort in assembling these excellent Exhibits which, together with the splendid Technical Exhibits, formed the most attractive part of the Annual Meeting. A detailed description of these Scientific Exhibits is given on the following pages of this issue of the JOURNAL.

The Technical Exhibits, which were arranged under the expert direction of Dr. James C. Cook, Manager and Dr. George A. Kling, Associate Manager of the Annual Meeting, were most instructive and superbly displayed. This issue of the JOURNAL also gives an account of these exhibits with a detailed list of the exhibiting firms.

As 3 years ago, the Chairman of the Committee on Local Arrangements was Dr. Charles E. Bickham, Jr., with charming Mrs. Charles E. Bickham acting as Chairman of the Ladies' Committee. The social activities prepared for this year's Annual Meeting were as follows:

Monday: *Seeing Beautiful Washington.* A completely lectured riding tour to the Lincoln Memorial, Arlington National Cemetery (including Tomb of the Unknowns and the Kennedy gravesites), Jefferson Memorial, Capital Hill, the White House, Washington Monument, the Federal Triangle and others.

Tuesday: *Morning at the Galleries.* Guided tour of the National Gallery of Art and of the Renwick Gallery (the latest addition in the family of the Smithsonian); followed by luncheon at the Port O'Georgetown on the C&O Canal in Old Georgetown, with an after lunch walk along the canal, or shopping in the square; followed by a visit to 2 of the leading Embassies, where the visitors were made welcome by high ranking officials of each Embassy.

Wednesday: *Day at the Theatre.* Lunch at the Watergate—home of Senators, Congressmen and many top Washington officials and diplomats; then a tour of the magnificent Kennedy Center; after that, attendance to a new musical comedy entitled "Pippin" at the Opera House in the Kennedy Center.

Thursday: *Shopping Expedition.* A trip to Landover Mall, one of the largest enclosed regional shopping centers on the East Coast, 4 major department stores (Woodward & Lothrop, Sears, Hecht, and Garfinkle), 125 specialty shops and 6 theatres.

To Dr. and Mrs. Charles E. Bickham, Jr., and the members of their Committees, the Society is very grateful and extends its profound thanks for arranging this most enjoyable program.

On Monday, preceding the meeting, the Annual Golf Outing for Members of the Society and their Guests was held at the Indian Spring Country Club. Dr. Ralph M. Caulk, Past President of the Society and ardent golf enthusiast, with the aid of Drs. Albert J. Miele, and Gregory T. Henesy made all the necessary arrangements. Under sunny skies on a beautiful day the Golf Play was for the Willis F. Manges Trophy (low net) and the Exhibitors' Trophy (low gross). This was a "shot-gun" tournament starting at 10:00 A.M. simultaneously from all tees. The players finished at the same time in midafternoon and a late buffet luncheon was served.

The Committee on Press Relationship, with Dr. Homer L. Twigg, Jr., as Chairman, ably assisted by Mr. Charles Honaker, Director of Public Relations of the American College of Radiology, conducted the press and public relation activities, with skill and good judgment.

The Society expresses its sincerest thanks to the Washington Convention Bureau and to the Management and Staff of the Washington Hilton Hotel, for the efficient registration, the excellent service, and the many other courtesies gracefully rendered throughout the meeting.

The Seventy-third Annual Meeting in *beautiful Washington, the Nation's Capital and in our time the Center of the World* was an outstanding success. President Dunbar merits the unbounded thanks and admiration of the Society.

Traian Leucutia, M.D.

THE SCIENTIFIC EXHIBITS

THE SCIENTIFIC EXHIBITS of the Seventy-third Annual Meeting of the American Roentgen Ray Society, held at the Washington-Hilton Hotel, Washington, D. C., October 3–6, 1972 were displayed in the spacious and conveniently located Convention Hall. They were the largest in the Society's history, of superb visual and documentative quality, and a surprisingly large number of them served to complement the Scientific Essays on the same subject presented at the Scientific Sessions.

The secret Committee on Awards, in judging the excellence and evaluating the scientific contributions of these Exhibits, must have had a very difficult task in selecting the most meritorious ones for the well deserved awards. This year, one Gold Medal, one Silver Medal, one Bronze Medal, and 20 Certificates of Merit were awarded.

The following is a list of the Awards granted and a brief description of the beautiful individual exhibits:

GOLD MEDAL

Radiologic and Related Studies of Cerebrospinal Fluid Formation. D. Gordon Potts, M.D., Michael D. F. Deck, F.F.R., and Vishnu Deonarine, R.T. Cornell University Medical College, New York, New York.

The mechanisms and sites of cerebrospinal fluid (CSF) formation were reviewed and the evidence for ependymal and extraventricular formation of CSF was discussed.

The rate of fluid formation in the lateral ventricle of the dog may be measured using lipiodol ascendant, pantopaque and air, or air alone. The formation proximal and distal to the aqueduct may be measured by the stereotactic introduction of an aqueductal block using microfil, a plastic material that is solidified after introduction into the aqueduct. Total CSF formation may be measured proximal to a cisternal block created by the introduction of lipiodol ascendant into the posterior fossa subarachnoid cisterns.

These techniques were demonstrated and compared with physiologic methods that have been used to measure the rate of CSF formation. The pneumographic changes due to CSF formation were displayed. These changes may be differentiated from those caused by absorption of the gas introduced into the ventricles.

SILVER MEDAL

Renal Scleroderma: A Combined Radiologic–Physiologic Study. William J. Casarella, M.D., Bentley A. Hollander, M.D., David B. Case, M.D., Paul J. Cannon, M.D., and E. Carwile LeRoy, M.D., Columbia-Presbyterian Medical Center, New York, New York.

Rapidly progressive renal failure is a frequent cause of death in scleroderma. Patients with renal scleroderma were studied by magnification arteriography, xenon 133 washout analysis, renal vein renin determinations and detailed roentgenograms of resected specimens.

Correlation of these parameters has demonstrated a possible pathophysiologic mechanism of rapid progressive cortical necrosis secondary to functional vasospasm or a renal "Raynaud's phenomenon."

In a series of 210 cases, 78 (37 per cent) had clinical evidence of renal involvement; however, 73 per cent of autopsied cases had microscopic evidence of renal disease and in 12 of 40 cases (30 per cent) 1 renal failure was considered to be the cause of death.

The exhibit displayed the anatomic-physiologic correlates of the study and the radiologic techniques employed.

BRONZE MEDAL

The Abnormal Brain Scan: Sensitivity and Specificity of Descriptive Parameters. Dennis D. Patton, M.D., Edward V. Staab,

M.D., and David R. Brill, M.D., Vanderbilt University Medical Center, Nashville, Tennessee.

Several signs in brain scans are said to be "typical" of certain central nervous system (CNS) disorders; *e.g.*, the wedge sign for cerebrovascular accident (CVA). In interpreting such a sign one should rather know its specificity (how many patients showing a wedge have CVA) than its sensitivity (how many patients with CVA show a wedge).

To evaluate the specificity and sensitivity of a number of "signs" the Exhibitors analyzed the lesions in 300 true-positive brain scans using 78 parameters describing the size, shape, number, location, density, homogeneity, sharpness, frontal lucency sign, symmetry, bolus findings, and reproducibility.

The exhibit summarized the specificity and sensitivity of each of the parameters for 21 CNS disorders.

CERTIFICATES OF MERIT

Bronchial Brush Biopsy in Cavitary Lung Disease. Robert Thomas Bramson, M.D., John Vincent Forrest, M.D., and Ronald Gene Evens, M.D., Mallinckrodt Institute of Radiology, St. Louis, Missouri.

Bronchial brush biopsy was performed 26 times in 25 patients with a wide variety of cavitary lung disease which could not be diagnosed by other methods short of surgical exploration. Fifteen of these 26 brush biopsies provided diagnostic material, including 12 of 16 cases in which the cavity was entered by the brush. Bronchial brush biopsy is a highly effective method of diagnosing cavitary lung disease, especially if the cavity can be entered.

A wide variety of cavitary lung disease, including primary and secondary neoplasm, bacterial lung abscess, tuberculosis, fungal infection, and Wegener's granulomatosis was demonstrated and the brush biopsy results presented.

Pleuropulmonary Manifestations of Collagen Diseases. Robert H. Dann, M.D., Peter H. Arger, M.D., and James R. Schwade, M.D., Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania.

The "specific" pleuropulmonary manifestations of 5 collagen diseases—rheumatoid arthritis, scleroderma, systemic lupus erythematosus, polyarteritis nodosa, and Wegener's granulomatosis—were enumerated and discussed with examples shown. The relative incidence and distinguishing clinical and laboratory features were considered. Frequent nonspecific manifestations were discussed, and examples shown, where appropriate.

Ophthalmic Angiography in the Tumors of the Orbit. D. Dilenge, M.D., Centre Hospitalier Universitaire, Sherbrooke, P.Q., Canada.

Angiography of the ophthalmic artery has been perfected to such an extent that its use has become crucial in the study of intraorbital tumors. A better definition of the ophthalmic artery and its branches has thus permitted to identify most of the changes typical of each intraorbital pathology. As these tumors are rare and their angiography poorly known, the Exhibitors have gathered in this exhibit the most typical angiographic patterns selected from their personal series of 37 cases, where angiography played a decisive diagnostic role. This series included benign tumors and 3 metastatic lesions.

The Intraosseous Ganglion. Frieda Feldman, M.D., and Austin D. Johnston, M.D., Columbia-Presbyterian Medical Center, New York, New York.

Intraosseous ganglia have been rarely recorded. This apparent rarity may be partially ascribed to confusion in terminology, as well as to unfamiliarity with an entity which has been variously labelled as "synovial, bursal or degenerative cyst."

The purpose of this exhibit was to review an experience with 33 ganglia which occurred within the bone rather than subperiosteally or adjacent to bone, to discuss theories as to their pathogenesis and to describe their histologic and roentgen findings.

Roentgenology of Branchial Arch Anomalies. Edmund A. Franker, Jr., M.D., and Heun Y. Yune, M.D., Indiana University Medical Center, Indianapolis, Indiana.

The embryology, classification, and pertinent roentgenology of the various branchial arch anomalies were illustrated.

An attempt was made to clarify the roentgen approach to these lesions by presenting the developmental relationships of various structures of the head and neck which are involved in these conditions.

Structural anomalies of the facial region are best understood when related to embryologic branchial arches. First arch disorders include mandibular, facial, external auditory canal, and ossicular anomalies. Persistence of branchial grooves and/or pharyngeal arches 2 and 3 results in sinus, cyst, or fistula in the neck.

Vascular Catheters, Clots and Clot Prevention. Cesare Gianturco, M.D., James Anderson, Ph.D., Sidney Wallace, M.D., Hector Medellin-Lastra, M.D , David deJongh, M.D., and Gerald Dodd, M.D., M. D. Anderson Hospital & Tumor Institute, Houston, Texas.

The authors have utilized light and scanning electron microscopy to investigate the various complications associated with vascular catheterization. Methods of reducing thromboembolic complications utilizing systemic heparinization and heparin surface coating agent were presented. The topographic features of angiographic catheters and both stainless steel and teflon coated guide wires were illustrated along with microscopic evidence of vascular intimal injury associated with their use.

The following conclusions were made: (1) no significant difference has been noted between stainless steel and teflon coated guide wires with respect to clot formation and vascular trauma; (2) surface clotting is found in significant amounts on guide wires and vascular catheters kept within the arteries or veins for 10 or more minutes; (3) to minimize clot formation the following precautions are recommended: (a) careful manipulation of the catheters and guide wires to obviate vascular trauma, (b) use of the smallest possible catheter consistent with an adequate study, (c) reduction of the time required per procedure, (d) frequent intermittent and forceful flushing of the catheter, (e) systemic heparinization consisting of an initial injection of aqueous heparin followed by periodic catheter irrigation with a heparin-saline solution, (f) the use of an anticoagulant coating agent on the surface of catheters and on guide wires should be considered, and (g) guide wires and catheters should be used for one procedure only and then discarded.

Arteriovenous Shunt Determination—The Complementary Roles of Radiology, Angiography and Nuclear Medicine. Frank A. Giargiana, Jr., M.D., A. Everette James, Jr., M.D., N. David Greyson, M.D., Buck A. Rhodes, Ph.D., Michael Siegel, M.D., Henry N. Wagner, Jr., M.D., and Robert I. White, Jr., M.D., The Johns Hopkins Hospital, Baltimore, Maryland.

The methocology of determining the presence of arteriovenous shunts using technetium 99m albumin microspheres was illustrated. The application of this technique was demonstrated, using scans, angiograms, and roentgenograms obtained during evaluation of various clinical entities, such as Paget's disease, fibrous dysplasia, peripheral vascular disease, postoperative sympathectomy, and peripheral arteriovenous malformations.

Experimental data demonstrating the absence of arteriovenous shunts in patients with peripheral vascular disease, both before and after contrast media injection, were presented. The application of this technique, utilizing the information gained by a combined approach of radioisotopic studies and angiography was stressed.

Radiology of the Pterygomaxillary Fossa. Leo V. Gould, M.B., E. Robert Heitzman, M.D., John P. Marangola, M.D., Joseph J. Moro, R.T., and Daniel D. Rabuzzi, M.D., Syracuse, New York.

There has been increasing interest in the surgery of the pterygomaxillary fossa during the recent decade. Accurate interpreta-

tion of roentgenograms of this area requires an understanding of the roentgenographic anatomy of the area.

This exhibit correlated illustrations of normal anatomic preparations and roentgenograms thereof with plain film roentgenograms and tomograms of normal subjects, selected examples of trauma, and other pathology in the pterygomaxillary fossa.

Late Sequelae of Renal Trauma in the Pediatric Age Group. Robert Hutchison, and M. Bernadette Nogrady, M.D., The Montreal Children's Hospital, Montreal, P.Q., Canada.

The clinical and radiologic documentation of late sequelae of renal trauma in 123 children was presented. Illustrative cases were used to demonstrate the various changes after trauma of the kidney.

On the basis of the study, the following conclusions were made: (1) prognosis cannot be made on the basis of immediate posttraumatic urogram; (2) to quantitate renal damage urography is mandatory; (3) the degree of permanent damage is likely to be established within a year of renal trauma; (4) the growth of the recognizably traumatized kidney is abnormal; (5) focal hypertrophy may simulate neoplasm; (6) hypertension is not necessarily found in the pediatric age group; and (7) conservative treatment seems reasonable.

Cerebrospinal Fluid in Hydrocephalus: Patterns of Flow and Absorption in Experimental Studies. A. Everette James, Jr., M.D., Bruce Konigsmark, M.D., Charles E. Jordan, M.D., George P. Saba, II, M.D., T. H. Milhorat, M.D., Ernst P. Strecker, M.D., and Giovanni Di Chiro, M.D., Johns Hopkins Hospital, Baltimore, Maryland; Children's Hospital Center, Washington, D.C.; and National Institutes of Health, Bethesda, Maryland.

Cerebrospinal fluid (CSF) imaging (cisternography) is a diagnostic study based on the premise that certain radiopharmaceuticals, after subarachnoid injection, reflect the bulk movement of cerebrospinal fluid. In patients with communicating hydrocephalus, following injection of the radiopharmaceutical in the lumbar area, the cisternographic images show abnormal entry of the radiopharmaceutical into the lateral ventricles. "Stasis" of the radiopharmaceutical in the lateral ventricle for longer than 24 hours has been correlated with marked neurologic improvement after a CSF diversionary shunt.

Experimental models for hydrocephalus have recently been developed in attempts to document and explain these abnormalities. The clinical observations, research investigations, and formulated theoretic explanations were presented in detail. The exhibit emphasized the research project.

Coronary Collateral Circulation. David C. Levin, M.D., Michael K. Kauff, M.D., and Harold A. Baltaxe, M.D., New York Hospital-Cornell Medical Center, New York, New York; and Montefiore Hospital and Medical Center, Bronx, New York.

In patients with coronary artery disease (CAD), the presence of collateral circulation is one of the key factors in preservation of myocardial function.

Coronary arteriograms of 200 patients with significant CAD have been analyzed for the purpose of studying this collateral circulation. All studies utilized both cine filming and rapid serial roentgenograms.

A series of well-defined, collateral pathways can be seen in obstructions of both the right and left coronary arteries. These pathways were illustrated diagrammatically and angiographically and their relative frequencies of occurrence indicated.

High Magnification X-Ray Sensitive Vidicon Photography. Eric N. C. Milne, M.B., and Werner W. Roeck, Eng. (Grad.), Radiological Research Laboratories, University of Toronto, Toronto, Ontario, Canada.

An x-ray sensitive Vidicon tube is used in place of film. The $\frac{1}{2} \times \frac{1}{2}$ inch input face is scanned by a 1029 line raster giving a potential resolution of 40 line prs./mm. (actual resolution 15 line prs./mm.). The output face is a 20 inch T.V. monitor giving a 40× electronic magnification.

Dynamic studies of minute renal, cardiac,

pulmonary and tumor vessels and other microstructures are easily carried out.

Using a special effects generator, physiologic information and images are simultaneously recorded on videotape.

The apparatus is a valuable research device, but also has obvious great clinical potential.

Peritoneography: Normal and Pathologic Anatomy; A New Radiographic Method for Evaluation of the Peritoneal Cavity and Its Contents. Morton A. Meyers, M.D., The New York Hospital-Cornell University Medical College, New York, New York.

Peritoneography is the double-contrast examination of the peritoneal cavity and its contents using a water-soluble positive contrast medium and gas. It is a simple, safe technique which can be done in any x-ray department. It permits the roentgenographic visualization and determination of external surfaces and contours of abdominal viscera, peritoneal reflections and recesses, and the thickness of the wall of portions of the alimentary tract.

Peritoneography has been shown capable of identifying both the presence and extent of intraabdominal disease which may not be recognized by either routine contrast studies of the alimentary tract or peritoneoscopy.

Nonmalignant Causes of Positive Bone Scans with Fluorine 18. Robert E., O'Mara, M.D., University of Arizona School of Medicine, Tucson, Arizona; Patrick J. Conte, M.D., Lourdes Memorial Hospital, Binghamton, New York; and John G. McAfee, M.D., S.U.N.Y., Upstate Medical Center, Syracuse, New York.

Many nonmalignant conditions may produce positive bone scans with any agent. A few are merely normal variations rather than a true benign process. Some are common, some more rare.

The Exhibitors illustrated the appearance of these on bone scans by discussing selected cases from a group of 23 such processes that have been encountered in their experience in scanning the osseous system with F^{18}. These were: (1) normal structures and variations. (2) soft tissue abnormalities; (3) osseous abnormalities; and (4) abnormal regional decrease in activity.

A few of the conditions which may produce an abnormal distribution of F^{18} are: calcific tendinitis; osteomyelitis; arthritis; and Paget's disease.

Cutaneous Manifestations of Gastrointestinal Disorders. William A. Renert, M.D., and William B. Seaman, M.D., Columbia-Presbyterian Medical Center, New York, New York.

The Exhibitors collected 22 cases of systemic diseases involving the gastrointestinal tract and the skin simultaneously.

A color transparency of the typical skin findings was shown with a roentgenogram of the associated lesions in the gastrointestinal tract. The skin lesion should alert the physician to a possible underlying gastrointestinal abnormality.

The following disorders were presented: acanthosis nigricans; acrodermatitis enteropathica; anaphylactoid purpura; dermatitis hepatiformis; Bowen's disease; Canada-Cronkhite disease; Dego's disease; dermatomyositis; epidermolysis bullosa; Gardner's syndrome; graft *vs.* host disease; herpes zoster; Kaposi sarcoma; lymphosarcoma; mastocytosis; melanoma; neurofibromatosis; Osler-Weber-Rendu disease; pemphigoid; Peutz-Jeghers syndrome; pyoderma gangrenosum; regional enteritis; and scleroderma.

Nonoperative Catheter Therapy of Massive Gastrointestinal Hemorrhage. Josef Rosch, M.D., Charles T. Dotter, M.D., and Marcia K. Bilbao, M.D., University of Oregon Medical School Portland, Oregon.

Nonoperative catheter control of acute gastrointestinal bleeding is a simple and, in uncomplicated cases, very effective method, often eliminating the need for emergency surgery.

Selective arterial infusions of epinephrine or pitressin used in 52 patients controlled bleeding in 38.

Selective embolization of a bleeding artery was successful in one treated patient with a severe hemocoagulation defect.

Gravitational Pneumonia. Gerard T. Scanlon, M.D., and Peter J. Kafura, M.D., Medical College of Wisconsin, Milwaukee, Wisconsin.

Gravitational pneumonia is a unique syndrome with highly characteristic clinical and roentgenologic findings. It is not to be confused with aspiration pneumonia, an altogether different condition.

In patients with a diminished level of consciousness, "bronchial emboli" of oral secretions pass into the most dependent bronchus—usually the axillary segment of the right upper lobe or the superior segment of the lower lobe. The effect is an intense pneumonia with early cavitation producing a typical roentgenographic appearance. Cultures show organisms characteristic of the oral cavity. As distinct from aspiration pneumonia, gravitational pneumonia apparently has not been described in the radiologic literature.

The Exhibitors presented examples of such cases.

Radiologic and Clinical Evaluation of Polycystic Disease in Infants and Children. Alfred L. Weber, M.D., and Richard C. Pfister, M.D., Department of Radiology, Massachusetts General Hospital, Boston, Massachusetts.

The different types of polycystic disease in infants and children were discussed. The radiographic findings were described in each type with particular reference to mode of examination such as intravenous pyelography, nephrotomography, and angiography. The pathologic findings were briefly reviewed and correlated with the radiographic patterns.

The clinical manifestations, including prognosis in the various forms, were analyzed. Selective cases were shown to illustrate the clinical and roentgenographic spectrum encountered in this disease. The intravenous urogram, nephrotomogram and renal angiogram are important for the diagnosis, classification, and prognosis of renal cystic disease.

The classification is as follows: (1) perinatal polycystic disease; (2) neonatal polycystic disease; (3) infantile polycystic disease; (4) adult polycystic disease; (5) juvenile polycystic disease; (6) medullary cystic disease; (7) multicystic dysplastic kidney; (8) simple cyst; and (9) urinary tract obstruction.

The Solitary Mass in the Lungs of Coal Miners. John Lecoq Williams, M.D., and George Anton Moller, M.D., Geisinger Medical Center, Danville, Pennsylvania.

Roentgenograms of 1,500 Pennsylvania coal miners who had worked underground more than 15 years showed one or more lung masses in 9.6 per cent of the anthracite miners and in 3.8 per cent of the bituminous miners.

Most of the masses are caused by conglomerate pneumoconiosis (progressive massive fibrosis). The lesions may closely simulate a carcinoma when only a single mass is present, especially if there is little or no roentgen evidence of pneumoconiosis elsewhere in the lungs.

The exhibit demonstrated that the correct diagnosis can be made and unnecessary biopsy avoided when the characteristic contour, density and calcifications or progressive massive fibrosis are present.

Rickets and Antiepileptic Therapy: Rochester Experience. Lionel Wesley Young, M.D., Gilbert B. Forbes, M.D., Agneta D. Borgstedt, M.D., and Michael F. Bryson, M.D., University of Rochester Medical Center, Rochester, New York.

The relationship between therapy with antiepileptic drugs, particularly diphenylhydantoin, and the development of rickets has recently been reported from Germany.

Three Rochester children who during the course of long-term antiepileptic therapy for debilitating neurologic illnesses developed rickets confirmed by clinical chemistry and roentgenology, were reported. Healing occurred following vitamin D therapy despite continued management with antiepileptic drugs.

The pathogenesis of the disturbed mineral metabolism in these patients is now being elucidated, due to advances in identification of the active metabolites of vita-

min D and an understanding of the pharmacologic action of the anticonvulsant drugs.

Since rickets and osteomalacia can result from antiepileptic therapy, special attention should be paid to the vitamin D intake of children who are receiving antiepileptic drugs.

OTHER EXCELLENT EXHIBITS WERE:

An Analog System for External Beam Treatment Planning and Dosimetry. R. B. Adams, M.S., K. Pillai, M.S., E. C. Gregg, Ph.D., and W. Voelker, Case Western Reserve University, Cleveland, Ohio.

The theory and use of the analog system for multibeam treatment planning were depicted.

The planning system consists of a library of film analogs of the treatment beams, a viewing system for arranging and observing the results of up to 4 beams on a patient profile, and a multiple densitometry system with digital readout to measure the dose in rads at any point of interest in the beams. The film analogs represent the x-ray beams in tissue equivalent material.

The analog system permits the real time optimization and measurement of the dose distribution in the patient profile within 20 minutes.

Pediatric Case of the Day. D. H. Altman, M.D., R. E. Litt, M.D., L. A. Greenberg, M.D., and M. Jordan, M.D., Variety Children's Hospital, Miami, Florida.

A different pediatric case was presented each day.

The history and pertinent roentgenograms permitted a single diagnosis.

The answer was revealed the following day with a discussion and demonstration in color of the pathology.

A reward was given to all correct diagnosticians.

Tumors of the Diaphragm. Larry Simon Anderson, M.D., and John Vincent Forrest, M.D., Mallinckrodt Institute of Radiology, St. Louis, Missouri.

The experience at the Washington University Medical Center with these lesions in the past 20 years was reviewed and typical roentgenographic appearances were demonstrated.

A variety of rare tumors may involve the diaphragm. Examples of cysts, benign tumors, endometriosis, primary and metastatic malignancy including direct invasion were presented.

Benign Proliferative Lesions of the Bladder. Peter H. Arger, M.D., and Robert H. Dann, M.D., Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania.

Benign proliferative bladder lesions, cystitis cystica and cystitis glandularis, are confused on cystoscopy and roentgenographic study with bladder tumors.

The trigone and bladder neck are the primary sites of involvement. Both give rise to metaplastic bladder epithelium lesions due to noxious stimuli.

The relationship of bladder epithelium metaplastic lesions to one another, conditions associated with these lesions, and the differential diagnosis were detailed.

The roentgenographic changes of, and the statistics on 21 biopsy proven cases were shown in this exhibit.

Case of the Day. Charles E. Bickham, Jr., M.D., Doctor's Hospital, Washington, D. C.

A challenging adult roentgenologic diagnostic problem was presented daily with awards for the correct diagnoses posted on the following days.

Veterinary Radiology. Larissa Bilaniuk, M.D., Darryl Biery, D.V.M., and Wallace T. Miller, M.D., University of Pennsylvania, Philadelphia, Pennsylvania.

Veterinary Radiology, like Radiology involving human subjects, has progressed greatly in the last several decades. Interesting analogies between animal diseases and human diseases were presented. Potential benefit to understanding human disease by investigating their animal counterparts is obvious.

Radiologic Description of Coal Workers' Pneumoconiosis. Leonard Bristol, M.D., Dale Trout, B.S., D.Sc., Ray Moore, M.D.,

and George Jacobson, M.D., Chairman, American College of Radiology, Chevy Chase, Maryland.

This exhibit recapitulated the UICC/Cincinnati Classification of the roentgenographic appearances of the pneumoconioses and discussed the film quality required for demonstrating parenchymal lesions in the lungs.

Lesions Causing Radiolucent Defects in the Renal Pelvis. Robert C. Brown, M.D., Maynard C. Jones, Jr., M.D., Robert A. Boldus, M.D., and Rubin H. Flocks, M.D., University of Iowa Hospitals, Iowa City, Iowa.

Filling defects in the renal pelvis have been a problem to radiologists and urologists since the inception of urography. The Exhibitors collected 17 examples that demonstrate some of these defects.

The radiolucencies in the renal pelvis may be due to intrinsic or extrinsic causes. The diagnosis of their etiologies is dependent on their roentgenographic manifestations as well as careful attention to the history, physical signs and various laboratory procedures.

A review of the salient findings in these conditions was presented.

Diagnostic Radiological Health Sciences Learning Laboratory. Reynold F. Brown, M.D., Kevin M. Kelly, M.D., and John C. Villforth, M.S., Bureau of Radiological Health, U. S. Public Health Service, DHEW, Rockville, Maryland.

This system provides educational material directed at all 3 phases of the radiologic examination: selection of the patient; conduct of the examination; and interpretation of the results.

The material pertinent to patient selection was presented for inclusion in lectures to medical students. A self contained cabinet x-ray machine is used in lecture laboratory exercises to present practical diagnostic physics principles to residents. The Learning file is an instructional system for "basic" education in the interpretive aspects of the radiologic examination suitable for medical students, residents and refresher training, and consists of 1,700 cases containing 3,500 full sized reproductions that simulate the original roentgenograms.

The values of the system were presented in the exhibit.

Polytomographic Evaluation of Thoracolumbar Neural Arch Fractures. Bernard M. Casey, M.D., S. Boyd Eaton, Jr., M.D., James J. Du Bois, M.D., Myron D. Tremaine, M.D., and Carl J. Mani, M.D., Letterman General Hospital, San Francisco, California.

Roentgen diagnosis of thoracolumbar spinal trauma is a matter of considerable clinical and medicolegal interest. Because the detection and assessment of such injuries from conventional roentgenograms are so difficult, the current classification of spinal fractures depends largely on clinical criteria. Twenty-eight patients with thoracolumbar spinal injuries were evaluated by conventional and polytomographic methods.

Representative examples of fractures involving each of the neural arch elements were shown. Accurate evaluation and classification of potential neural arch trauma are best accomplished by hypocycloidal tomography in the coronal plane.

Roentgenographic Profile of Infundibular Stenosis: A New Angiographic Sign, "Wedge Sign of Infundibulum." Eung Man Cha, M.D., Chicago Medical School at Mt. Sinai Hospital, Chicago, Illinois; and George H. Khoury, M.D., University of West Virginia Medical Center, Morgantown, West Virginia.

Three distinct angiographic patterns of infundibular stenosis in the lateral view were correlated in 15 patients with infundibular stenosis and associated ventricular septal defect (V.S.D.), using chest roentgenograms, hemodynamic data, and surgical findings. The 3 patterns were:

1. The wedge sign of infundibulum, a new sign describing a triangular opacity with its apex directed caudad in the infundibulum due to diffuse hypertrophy; V.S.D. was large.

2. Localized or discrete stenosis at the

site of the papillary muscle of the conus; V.S.D. was usually small.

3. Scattered filling defect in the right ventricle denoting anomalous muscle bundles.

Pitfalls of Catheter Route in Congenital Heart Disease. Eung Man Cha, M.D., Chicago Medical School at Mt. Sinai Hospital, Chicago, Illinois; and George H. Khoury, M.D., University of West Virginia Medical Center, Morgantown, West Virginia.

It is desirable to make a correct diagnosis of cardiac anomaly on anteroposterior and lateral spot roentgenograms of the chest with a cardiac catheter in place. The arterial or venous catheter courses in such a specific way that one is able to assume the diagnosis when the route of the cardiac catheter is stated.

The diagnosis can be made correctly in anomalies of the great vessels, and cyanotic and acyanotic heart disease. The size of the patent ductus arteriosus (PDA) can be predicted by evaluating the angle of the catheter between the aorta and PDA.

The purpose of this study was to demonstrate different courses of the cardiac catheter on spot roentgenograms in various conditions.

Eosinophilic Lung Disease (How to Slice P.I.E.). Lawrence A. Citro, M.D., Michael E. Gordon, M.D., and Wallace T. Miller, M.D., Hospital of University of Pennsylvania, Philadelphia, Pennsylvania.

An organized approach to the diagnosis of eosinophilic lung disease was presented with emphasis on classification.

Eosinophilic lung disease may be divided into the following categories: 1. simple pulmonary eosinophilia; 2. prolonged pulmonary eosinophilia; 3. tropical eosinophilia; 4. pulmonary eosinophilia with asthma; and 5. (a) polyarteritis nodosa, (b) Wegener's granulomatosis.

Dislocated Total Hip Prostheses: Early Roentgen Diagnosis. Richard H. Daffner, M.D., John A. Gehweiler, M.D., John Olson, M.D., James W. Wilson, M.D., Ph.D., and Terrence S. Carden, M.D., Duke University Medical Center, Durham, North Carolina.

The McKee-Farrar, Charnley-Müller and Ring total hip prostheses have one or more projections on the acetabular component which bears a constant geometric relationship to the metallic femoral head. Dislocation will disturb this normal geometric relationship, which allows early diagnosis in frontal roentgenograms. Fortunately, dislocation of total hip prostheses is rare.

Three signs, the Floating Mine Sign, the Saturn Ring Sign, and the Tin Woodsman Sign, respectively, were demonstrated for each prosthesis.

An Angiographic Study of the Meningorachidian Venous System. D. Dilenge, M.D., and B. Perey, M.D., Centre Hospitalier Universitaire, Sherbrooke, P.Q., Canada.

The meningorachidian venous plexus is a complex vascular network which communicates with the caval system at all levels of the trunk as well as with the intracranial circulation.

This plexus which contains few valves, if any, provides crucial compensatory mechanisms in obstruction of either vena cava. Also, it may be the main pathway of cerebral venous return in certain pathologic or even physiologic states.

Some of these features were illustrated in this exhibit.

Extensive angiographic findings from experiments on monkeys similarly gave certain typical hemodynamic patterns of this plexus.

Multisystem Radiographic Analysis of Complications in Thermally Burned Patients. Steven R. Dennis, M.D., Raoul O. Hagen, M.D., and Lee F. Rogers, M.D., Brooke General Hospital, Fort Sam Houston, Texas.

To familiarize the radiologist with the broad spectrum of acute and chronic complications that occur in thermally burned patients, the following topics were presented:

a. Respiratory complications: airborne pneumonia, hematogenous pneumonia, pul-

monary edema, inhalation injury and iatrogenic injuries.

b. Gastrointestinal complications: paralytic ileus, esophageal stricture, monilial esophagitis, Curling's ulcer, superior mesenteric artery syndrome, acalculous cholecystitis.

c. Musculoskeletal complications: periarticular calcifications, fundal osteomyelitis, septic arthritis, growth disturbance, and acromutilation of digits.

d. Miscellaneous complications: suppurative thrombophlebitis, monilial pyelonephritis, and ecthyma gangrenosum.

Dumbbell Neuroblastoma-Ganglioneuroma of the Spine. Charles J. Fagan, M.D., Leonard E. Swischuk, M.D., and Glenn A. Meyer, M.D., University of Texas Medical Branch, Galveston, Texas.

Seven cases of dumbbell neuroblastoma-ganglioneuroma of the spine were illustrated. It is not well known that this tumor can present in this fashion, but it is important that it be recognized, for emergency decompressive laminectony is frequently required.

The roentgenographic findings and their specificity in suspecting this diagnosis were outlined. These are: (1) paraspinal mass with or without calcification; (2) sclerosis; (3) rib erosion; (4) pedicular flattening or erosion; (5) posterior vertebral body scalloping; etc.

The Roentgenologic Appearance of Hiatal Hernia Repairs. David S. Feigin, M.D., A. Everette James, Jr., M.D., Frederick P. Stitik, M.D., Martin W. Donner, M.D., and David B. Skinner, M.D., The Johns Hopkins Hospital, Baltimore, Maryland.

The 4 major types of hiatal hernia repairs were described with regard to the surgical procedures. Typical examples of postoperative roentgenologic manifestations and their differential features were illustrated.

Following a diaphragmatic crural repair, no subdiaphragmatic esophagus will be seen. The fundus of the stomach will appear normal. Other common procedures create an intraabdominal esophagus. The Nissen fundoplication creates a "fundic pseudotumor" effect, the posterior gastropexy (Hill) leaves a smoothly curved lower esophagus and the Mark IV (Belsey) repair creates an acutely angulated terminal esophagus.

Recognition of these features is essential in analyzing the effectiveness of the repair and in evaluating surgical complications.

Abnormalities of Pulmonary Aeration in the Neonate. Alvin H. Felman, M.D., Barry V. Kirkpatrick, M.D., and Donald V. Eitzman, M.D., University of Florida, Gainesville, Florida.

Unusual pulmonary complications are being recognized with increasing frequency by the radiologist who deals with the neonate in respiratory distress. The roentgenologic changes in many cases are characterized by abnormalities of pulmonary aeration and are frequently associated with newer techniques of assisted ventilation.

This exhibit consisted of cases which illustrated a wide variety of usual and unusual patterns of abnormal pulmonary aeration. The early recognition of these changes and clues to the prediction of more catastrophic events were stressed. Correlation with clinical findings, particularly blood gas determinations, was also included.

Percutaneous Transhepatic Cholangiography. Murray G. Fischer, M.D., Abraham Geffen, M.D., and Sezen Ozoktay, M.D., Beth Israel Medical Center, New York, New York.

Percutaneous transhepatic cholangiography was performed in 36 patients, with successful visualization in 89 per cent.

The indications and contraindications for the procedure were detailed and the technique of the lateral approach used in this series was elaborated. The importance of the inability to enter the biliary ducts in nonobstructive jaundice was stressed. Emphasis was placed on postcholangiography drainage in helping to decrease the incidence of bile leakage as a complication in cases of obstructive jaundice.

Details of representative and unusual cases were presented.

Angiography in Pulmonary Tension Disorders of Infants and Children. Edmund A. Franken, Jr., M.D., Roger A. Hurwitz, M.D., and J. Stanley Battersby, M.D., Indiana University Medical Center, Indianapolis, Indiana.

Unilateral tension disorders of the lung in children can be classified as lesions of overexpansion and underexpansion. Roentgenography and fluoroscopy determine if over- or underexpansion is present, but for precise diagnosis further procedures are necessary.

The use of pulmonary angiography in infants and children with various tension disorders was presented. Emphasis was placed on the substitution of angiography for bronchography in the diagnosis of this category of diseases.

Pulmonary angiography is considered a safer and more accurate diagnostic procedure in this group of patients.

Radiologic Evaluation of Common Hip Surgery. Matthew Freedman, M.D., University of Rochester, Strong Memorial Hospital, Rochester, New York.

This exhibit surveyed the radiologic appearance of operations performed on the hip and the more common of the complications encountered.

Surgery on both the adult and pediatric hip was covered by means of illustrations and examples from actual cases.

Congenital Malformation of the Ears. Bob W. Gayler, M.D., and Janet L. Strife, M.D., Johns Hopkins Hospital, Baltimore, Maryland.

Roentgenograms of 100 patients with congenital deafness and/or external ear abnormalities seen at the Johns Hopkins Hospital from 1964–1972 were reviewed. All patients had pluridirectional tomograms to delineate their structural abnormalities. Various types of congenital malformations were demonstrated. Broadly they were divided into external, middle and inner ear abnormalities. Examples of each category were illustrated with line drawings and correlated clinical information.

Roentgenographic Evaluation of Femoral Head Prostheses. Thomas Stanley Harle, M.D., Lee Frank Rogers, M.D., James Elmer Butler, III, M.D., and Stanley C. Jones, M.D., The University of Texas Medical School at Houston, and Hermann Hospital, Houston, Texas.

The radiologist is frequently faced with the problem of evaluating the postoperative status of the hip following replacement of the femoral head. Unfortunately, little information is available as to what constitutes a desired result.

This exhibit illustrated with roentgenograms from over 40 cases the desired and undesired results postoperatively and the long term complications. The 2 most frequently used prostheses (Thompson and Moore) were shown and their differences in design stressed. The important relationships between the prosthesis and acetabulum and remaining femur were noted.

Roentgenographic Picture of Selected Surgical Procedures of the Hip in the Pediatric Age Group. Norma Fallis Harris, M.D., Gene C. Cunningham, M.D., and David Merten, M.D., Department of Radiology, Baylor College of Medicine, Houston, Texas.

Pre- and postoperative roentgenograms were presented which depicted selected orthopedic problems in children, involving surgical correction at the hip. The procedures discussed were the Salter, Colonna, Pemberton, Chiari, Sharrard, Southwick, Haggie Penning procedures and valgus osteotomy.

How is This Chest Roentgenogram Taken? Kenneth R. Kattan, M.D., and Jerome F. Wiot, M.D., University of Cincinnati, College of Medicine, Cincinnati, Ohio.

The question of how a chest roentgenogram was obtained, either in the anteroposterior or posteroanterior projection, is occasionally important in the adequate interpretation of the roentgenogram.

The Exhibitors found that when the appearance of C-6, C-7 and D-1 vertebral bodies, the superior and inferior vertebral plates and the joints of Luschka are well seen, the roentgenogram was obtained in

the anteroposterior projection. On the other hand, when the neural arch and the articular processes and laminae of C-6, C-7 and D-1 are well seen, the roentgenogram was made in the posteroanterior projection. The exhibit included an explanation of this phenomenon.

Trauma and "No Trauma" of the Cervical Spine. Kenneth R. Kattan, M.D., Department of Radiology, University of Cincinnati College of Medicine, Cincinnati, Ohio.

This exhibit showed some of the anatomic features of the cervical spine. Roentgenograms of anatomic specimens were illustrated to show the reason why some traumatic lesions are missed. Different clinical examples of obvious and subtle traumata of the cervical spine were shown. Stress was placed on the difficult cases and how one can arrive at the proper diagnosis (one example is how to diagnose anterior dislocation when a lateral roentgenogram is difficult to obtain). A few nontraumatic cases were shown for the sake of differential diagnosis.

The Angiographic Anatomy of Aortico-coronary Bypasses. Michael K. Kauff, M.D., David C. Levin, M.D., and Harold A. Baltaxe, M.D., New York Hospital-Cornell Medical Center, New York, New York.

This exhibit demonstrated the authors' technique of evaluating aorticocoronary bypass surgery angiographically. Examples of normal saphenous vein and internal mammary artery bypasses with bidirectional flow were shown. The significance of bidirectional *vs.* unidirectional flow was briefly discussed. Occluded bypasses were shown and the need for restudying the coronary artery stressed. Other abnormalities, such as intimal hyperplasia of veins and false aneurysm formation, were shown.

Renal Arteriography in Sickle Cell Nephropathy (A Preliminary Report Correlating the Angiographic and Urographic Changes in Sickle Cell Nephropathy). Mansour Khademi, M.D., and James Ralph Marquis, M.D., New Jersey College of Medicine and Dentistry, United Hospital Medical Center, Newark, New Jersey.

Eight cases of sickle cell disease (4 S-S, 1 S-A, 1 S-E, 1 S-C) who had abnormal excretory urograms were studied by renal arteriography.

There were no significant renal symptoms. The major urographic findings were distortion and blunting of the calyces (sickle cell caliectasis) and the kidneys were either normal or large.

The most common arteriographic finding was focal cortical hypertrophy. In 1 patient a pattern similar to brain convolution (pseudobrain pattern) was produced. Other arteriographic findings were scar formation, and irregularity and pruning of the arterial tree. Marked thinning of the cortex and relatively enlarged medullary zone were seen in a case of S-C and a case of S-S disease.

Angiographic Diagnosis of Chronic Inflammatory Pancreatic Diseases. Mansour Khademi, M.D., Eric Lazaro, M.D., and Robert R. Rickert, M.D., New Jersey College of Medicine and Dentistry at Markland Hospital, Newark, New Jersey.

Selective celiac and superior mesenteric arteriography was found to be the most revealing test in the diagnosis of chronic pancreatitis. Surgical exploration and tissue examination confirmed the arteriographic diagnosis of chronic pancreatitis, pseudocyst or abscess of pancreas in 35 cases. The diagnosis in another 43 cases was based on clinical, laboratory and arteriographic findings.

The most common arteriographic findings were: hypervascularity of the organ; irregularity and beading of the small arteries without encasement of the major arteries; and staining. The vascular changes were more generalized and involved only small arterial branches, in contradistinction to carcinoma. Focal or generalized capillary blush of the pancreas was the basic diagnostic finding of chronic pancreatitis. The splenic vein did not visualize in 12 per cent of the cases and the spleen drained *via* collateral venous channels. This finding is considered to be pathognomonic of pancreatitis of the body or tail in the absence of encasement of adjacent major arteries.

Direct Magnification Cerebral Arteriography. E. Leone Kier, M.D., William E. Allen, III, M.D., and Stephen L. G. Rothman, M.D., Yale University School of Medicine, New Haven, Connecticut.

This exhibit demonstrated the enhanced diagnostic quality of direct magnification angiography and subtraction techniques as well as the applications of stereo-angiography. When employed routinely, the finer vascular anatomy of the cranial circulation can be clearly demonstrated and, consequently, more accurate criteria for the topographic and pathologic evaluation of intracranial mass lesions can be established. In addition, these techniques have provided an improved modality for the teaching of normal and pathologic neuroradiologic anatomy.

Various features of meningeal, choroidal, midbrain perforators, tentorial, pontine and cerebellar vessels were demonstrated, and representative pathologic entities were presented.

Placental Migration Demonstrated by Ultrasonography. Donald L. King, M.D., Columbia-Presbyterian Medical Center, New York, New York.

Nine symptomatic patients in 2nd or 3rd trimester pregnancy were found by ultrasonography to have varying degrees of marginal placenta previa. On follow-up examinations prior to delivery the placenta previa was no longer found to be present. Although the placental position has been regarded as fixed after implantation, the evidence in these cases demonstrated that with uterine growth the placenta can migrate with respect to a fixed point, the cervix.

This evidence indicates that the diagnosis of placenta previa early in pregnancy should not be considered an absolute indication for cesarean section, and that placental ultrasonography should be repeated at term to determine if a change in placental location has occurred.

Battered Child Syndrome: Patterns of Injury and Significance of Uncommon Fractures. Marvin S. Kogutt, M.D., Leonard E. Swischuk, M.D., and Charles J. Fagan, M.D., University of Texas Medical Branch, Galveston, Texas.

The incidence and distribution of skeletal trauma in 100 battered children were reviewed and it was discovered that many infants did not present with the classic picture.

Less common, but nonetheless diagnostically significant, fractures of the spine, sternum, distal end of the clavicle, scapula, and ribs were also assessed, and it was determined that many of these fractures were as important as the epiphyseal-metaphyseal type.

The Panograph in Facial Trauma. David A. Krause, M.D., and Gary Erickson, D.D.S., University of Minnesota Hospitals, Minneapolis, Minnesota.

The panograph (circular facial tomograph) is a superior device for examining the mandible and maxilla, especially for facial trauma. Its ease of use, the clarity of the anatomic display, and the reproducibility in technique allow accurate assessment of injury and follow-up of healing. The detection of small fractures of the condyles is easier.

The normal anatomy was shown. Cases of pathology with corresponding standard roentgenograms for comparison were also shown.

New Roentgenologic Findings in Myotonia Dystrophica: An Analysis of 18 Patients. K. Francis Lee, M.D., Shu-Ren Lin, M.D., and Philip J. Hodes, M.D., Thomas Jefferson University Hospital, Philadelphia, Pennsylvania.

Eighteen cases of myotonic dystrophy in 5 families with special reference to new skull findings were presented. The reported roentgenologic findings include: diffuse thickening of the calvarium; hyperostosis frontalis; large frontal sinus; small sella turcica; elongated mandible; in addition to the findings of the gastrointestinal tract, as well as the respiratory and cardiovascular systems, due to involuntary muscular involvement.

Previously undescribed roentgenologic

findings are: decreased basal and nasal angles, and hypotelorism which were observed in most of the patients studied in the Exhibitors' series.

Pseudo-Meningiomatous Hyperostosis. K. Francis Lee, M.D., Shu-Ren Lin, M.D., and Norman J. Schatz, M.D., Thomas Jefferson University Hospital, Philadelphia, Pennsylvania.

Two cases of chromophobe adenomas, 1 case each of craniopharyngioma, epidermoidoma, thrombosed intracavernous aneurysms, sphenoid sinus carcinoma, chondroblastoma, and 2 cases of osteoblastic sellar metastases which produced juxtasellar hyperostosis constituted the material of this exhibit.

Other examples of entities which may produce juxtasellar hyperostosis were also presented. Juxtasellar hyperostosis has commonly been associated with visual disturbance.

The Arterial Relations of the Diaphragma Sellae—Preliminary Report. K. Francis Lee, M.D., Wesley Parke, Ph.D., and Shu-Ren Lin, M.D., Thomas Jefferson University Hospital, Philadelphia, Pennsylvania.

By means of arterial injections of mixtures of either micropaque and gelatin or neoprene latex and India ink in adult and fetal human cadavers, this study attempted to demonstrate the vascular relations of the diaphragma sellae.

The injected specimens showed that the marginal areas of the diaphragma could be divided into quadrants with respect to the sources of the fine arterial filaments that could be visualized within its tissue. With the exception of some inconstant contributions from the superior hypophyseal arteries, the 4 major arterial supplies were derived from the intracavernous sections of the carotid arteries and reached the diaphragma from its inferior aspect. The most readily demonstrable of these were fine terminals of the inferior hypophyseal arteries that served the posterior quadrant of the diaphragma located behind the stalk.

Radiologic Approach to Intraabdominal Masses in Children. James W. Lecky, M.D., Michael T. Gyepes, M.D., Paul Lee, M.D., and Ralph J. Uri, M.D., Department of Radiology, UCLA School of Medicine, Los Angeles, California.

The radiologist is frequently called upon to assess a palpable intraabdominal mass in the newborn or older child. Adequate localization and diagnostic evaluation are extremely important. Radiologic evaluation of renal, adrenal, hepatic and lumbosacral retroperitoneal structures was presented and discussed.

The indications and usefulness of special procedures such as antegrade pyelography and angiography were illustrated.

Diseases of Hands and Feet in Xeroradiography. John W. Li, M.D., and John N. Wolfe, M.D., Hutzel Hospital, Affiliated Hospital of Wayne State University, School of Medicine, Detroit, Michigan.

This exhibit contained xeroradiograms of the hands and feet with a variety of diseases. The pathology displayed included the different arthritides, manifestations of hyperparathyroidism, scleroderma, acro-osteolysis and other miscellaneous diseases. The purpose of the display was to demonstrate the advantage of obtaining excellent detail of the bone structures and soft tissues together in one xeroradiogram, which is not possible with the conventional roentgenographic film.

Details of radiation exposure were also discussed.

Gas Producing Perinephric Abscess. Leon Love, M.D., and Dennis Baker, M.D., Stritch School of Medicine, Loyola University of Chicago, Maywood, Illinois.

Gas producing perinephric abscess is a rare form of infection in the retroperitoneal space. Nine cases were demonstrated by scout roentgenography, pyelography and occasionally nephrotomography and arteriography. Six of the 9 cases occurred in diabetics. The roentgenographic and anatomic findings were shown. A differential diagnosis of extraluminal gas in the upper quadrants (perforated retroperitoneal hollow viscus, peritoneal and lesser sac abscess, emphysematous gastritis, pancreatic

abscess and others) was discussed.

Experimental Work in the Pathogenesis of Chronic Pyelonephritic Scarring. T. M. J. Malling, M.B.C.H.B., M.C.R.A., C. J. Hodson, F.R.C.P., F.F.R., P. J. McManamon, M.B.B.C.H., D.M.R.D., F.I.C.P., G. L. Rolleston, M.B.C.H.B., F.F.R., F.C.R.A., Departments of Radiology, Memorial University of Newfoundland, St. Johns, Newfoundland, and Christ's Church Hospital, Christ's Church, New Zealand.

Nearly all cases of chronic atrophic pyelonephritis are associated with psychoureteric reflux at some time in the patient's history. There is evidence to suggest that one of the links between psychoureteric reflux and the pyelonephritic scar is intrarenal reflux of urine from the bladder.

The Exhibitors demonstrated the phenomenon of intrarenal reflux on micturating cystourethrography in infants and experimentally in swine. It has been possible to show focal scarring in relation to the previously demonstrated areas of intrarenal reflux in the pig.

Routine Tomography in the Nonvisualized Gallbladder (A Method for Improved Diagnosis). Milton Margulies, M.D., and George T. Wohl, M.D., Lankenau Hospital, Philadelphia, Pennsylvania.

Failure to opacify the gallbladder following oral administration of contrast medium is an indication of gallbladder disease, providing that the common bile duct can be adequately visualized. Routine tomography in patients with nonvisualized gallbladder will generally demonstrate an opacified common duct and occasionally reveal the obstructing calculus. Rarely, a poorly visualized gallbladder will show good visualization that was not appreciated beforehand. The accuracy of diagnosis obviates the need for intravenous cholangiography. Results with illustrative cases were demonstrated.

Diseases of the Jaw. Richard J. Mark, M.D., Charles G. Baker, D.M.D., and Jack M. Tishler, M.D., University of Manitoba and Winnipeg General Hospital, Winnipeg, Manitoba, Canada.

The purpose of this exhibit was to demonstrate various manifestations of systemic and local disease in the teeth and jaw. Various modalities for roentgenographic examination of the teeth and jaw were also demonstrated. Examples were shown of manifestations in cleidocranial dysostosis, acromegaly, Paget's disease, and adenocystic carcinoma.

Radiology of Extraperitoneal Effusions—An Anatomic Approach. Morton A. Meyers, M.D., Joseph P. Whalen, M.D., Kenneth Peelle, M.D., and Alfred S. Berne, M.D., The New York Hospital-Cornell University Medical College, New York, New York.

The extraperitoneal space is not a "vast hinterland with shadowy fascial boundaries" but is clearly defined by anatomic planes into 3 compartments: the perirenal space; and the anterior and posterior pararenal spaces. Characteristic roentgenologic features distinguish the specific compartmentalization of extraperitoneal fluid collections and lead to identification of their source. The correlation of the distribution of fluid to the extraperitoneal fascial planes was based on anatomic sections derived from frozen cadavers, postmortem injection studies and selected clinical cases in varied disease states.

The American Registry of Radiologic Technologists—50th Anniversary. Mark M. Mishkin, M.D., Patricia O. Mueller, R.T., and Rowland C. McGowan, B.S., R.T., American Registry of Radiologic Technologists, Minneapolis, Minnesota.

The American Registry of Radiologic Technology was founded in 1922 and has been the examining and certifying body for organized technology since that time. Certification by the ARRT is recognized nationally and internationally as the standard of excellence. The R.T.-Radiologist team depends upon the professional excellence of both members. The history of the Registry, its current operations, and its role in a rapidly changing health care environment was presented.

Selective Management of Stabwounds of the Abdomen. Joseph M. Moynahan, M.D.,

George Haddad, M.D., Walter F. Pizzi, M.D., and Edgar P. Fleishmann, Knickerbocker Hospital, New York, New York.

The demonstration of the value of contrast material injection of stab wounds of the abdomen in determining penetration or nonpenetration of the peritoneum was the purpose of this exhibit.

The management consists of repeated examinations and the administration of intravenous infusions and antibiotics. The most important clinical finding was spasm or tenderness of the abdomen. Laparotomy was performed in 80 patients. The usual hospital course was 3 to 4 days.

The Lungs and Drug Abuse. Louis Dominic Napoli, M.D., Olcay Selime Cigtay, M.D., Homer Lee Twigg, M.D., Sol Katz, M.D., and Harold Weiss, M.D., Departments of Radiology and Medicine, Georgetown University Hospital and Providence Hospital, Washington, D. C.

An estimated 150,000–200,000 persons in the United States are addicted or habituated to opiates and other drugs. A countless number of others use these drugs intermittently.

The social and psychologic aspects of drug abuse are receiving considerable publicity at the present time. However, at the same time, the medical diseases associated with drug abuse have received relatively little attention.

The intent of the Exhibitors was to emphasize the medical complications, giving special reference to those changes involving the lungs. These pulmonary complications associated with drug abuse include: increased incidence of respiratory infections; pulmonary edema; septic pulmonary emboli; and foreign body reaction with angiothrombotic pulmonary hypertension.

Two previously unreported complications were presented and discussed: (1) multiple large talc granulomas in a heroin user; and (2) repeated episodes of pneumothorax associated with deep inhalation of marijuana.

The Enigma of Renal Parenchymal Damage in Children. M. Bernadette Nogrady, M.D., Montreal Children's Hospital, Department of Radiology, Montreal, P.Q., Canada.

Hypoplasia, dysplasia, sequelae of renal vascular damage, trauma, sepsis and urinary tract infection, or a combination of these, may be difficult or impossible to differentiate, unless roentgenologic and clinical documentation is available. A number of illustrative cases were displayed to emphasize the importance of urographic examination and the problems of interpretation.

"Addition"—A Method of Photographic Contrast Enhancement in Cerebral Angiography. John R. Olson, M.D., John A. Goree, M.D., and John P. Jimenez, M.D., Department of Radiology, Duke University Medical Center, Durham, North Carolina.

Addition contrast enhancement is a simply performed adjunct to photographic subtraction in cerebral angiography. In particular, the intermediate or "cerebrogram" phase may be improved to visualize characteristics of certain lesions which are not readily discernible on routine subtraction films.

The method consists of repeated copies of paired subtraction images to obtain an "added" image of the vascular elements. Individual vessels are adequately demonstrated by subtraction in most instances, but the poorly defined "cerebrogram" is more easily evaluated with the addition technique, and the problem of the "angiographically avascular" lesion often can be resolved.

This was amply demonstrated in the exhibit.

Some Aspects of the Roentgenologic Assessment of Speech. Heidi Brigitte Patriquin, M.D., Montreal Children's Hospital, Montreal, P.Q., Canada.

The roentgenologic investigation of disorders of articulation includes: (a) examination of the facial bones, especially the palate, mandible and teeth; and (b) examination of the soft palate and pharynx.

These structures are coated with thick colloidal barium sulphate and speech is examined in the mento-occipital and lateral

projections, using a combination of video and audiotape recording. With the aid of spot roentgenograms, this exhibit showed the structure and function of the facial bones and pharynx: (a) normally; (b) after adenoidectomy; (c) with cleft palate; and (d) following pharyngoplasty.

Diagnosis of Renal Masses. Howard M. Pollack, M.D., Barry B. Goldberg, M.D., and Jose Morales, M.D., Department of Radiology, Episcopal Hospital, Philadelphia, Pennsylvania.

This exhibit highlighted the virtues and pitfalls of each of the various modalities used in defining the nature of a renal mass; including nephrotomography, arteriography, radionuclide imaging, ultrasound and cyst puncture. It featured a dichotomous flow diagram (decision-making tree) indicating the proper order of studies in any given clinical situation. Unnecessary studies are eliminated and appropriate treatment is expedited by this technique.

Cerebrospinal Fluid Absorption. D. Gordon Potts, M.D., Daniel Gomez, Ph.D., and Vishnu Deonarine, R.T., Cornell University Medical College, New York, New York.

This exhibit discussed the following topics:

(1) Perfusion of the pathways with artificial cerebrospinal fluid, water-soluble contrast media, radiopaque substances and microscopically recognizable materials.

(2) Measurements of the gradient across the arachnoid villi in various positions.

(3) Light and electron microscopic appearances of villi and granulations with different pressure gradients across the villi and granulations.

(4) Blood vessels related to the arachnoid villi and granulations.

(5) Clinical significance of these findings.

The Spectrum of Soft Tissue Calcifications: Differential Diagnosis in Musculoskeletal Disease. Barry D. Pressman, M.D., and Frieda Feldman, M.D., Columbia-Presbyterian Medical Center, New York, New York.

Soft tissue calcifications are a valuable guide in the evaluation of musculoskeletal disease and in the direction of clinical investigations into more fruitful channels. Particular patterns were discussed and categorized, and the diagnostic value of certain combinations and distributions considered.

Column of Bertin: Diagnosis by Nephrotomography. Barry D. Pressman, M.D., William M. Green, M.D., Bruce L. McClennan, M.D., and William J. Casarella, M.D., Columbia-Presbyterian Medical Center, New York, New York.

Column of Bertin is a renal pseudotumor of no clinical significance. The universal opinion expressed in the literature is that arteriography is necessary to make this diagnosis.

However, it is the contention of the Exhibitors that in the presence of several classical findings of intravenous urography and, expecially, nephrotomography this diagnosis can be made without arteriography. This was demonstrated in the exhibit.

Roentgenologic Manifestations of Asbestosis. Howard W. Raymond, M.D., Department of Radiology, University of Cincinnati, College of Medicine, Cincinnati, Ohio; and Richard M. Spiegel, M.D., U. S. Public Health Service, National Institute for Occupational Safety and Health, Division of Field Studies and Clinical Investigations, Cincinnati, Ohio.

Examples of the various roentgenologic manifestations of asbestosis and its complications were presented with roentgenograms and clinical histories. Photographs of gross specimens and photomicrographs offered roentgenologic-pathologic correlations.

Early roentgenographic diagnosis using the UICC/Cincinnati Classification of small densities was discussed through representative chest roentgenograms of asbestos workers. The material presented was from the files of the National Institute for Occupational Safety and Health, the Department of Radiology, University of Cincinnati and the Environmental Science

Laboratory, Mt. Sinai School of Medicine (courtesy of Dr. Irving Selikoff).

Disseminated Intravascular Coagulation: Potential Roentgenologic Significance. Robert R. Renner, M.D., Douglas A. Nelson, M.D., Charles J. Aucreman, M.D., and Robert L. Lorenzo, M.D., Upstate Medical Center, Syracuse, New York.

There is a growing list of disease states where disseminated intravascular clotting (DIC) is either of primary etiologic significance or represents a serious complication of a previous illness. Clinically, DIC may create a hemorrhagic diathesis, ischemic tissue damage particularly in the kidneys, red cell damage and hemolysis, and may potentiate shock.

When present, the roentgenographic findings are often nonspecific, but DIC can result in roentgenographic findings such as hemorrhage or hematoma formation, thromboses and emboli, decreased or absent renal function, pulmonary edema, cardiac enlargement and pleural or pericardial effusion.

The exhibit discussed the basic pathology and pathophysiology of DIC and showed examples of roentgenograms demonstrating changes in these patients.

Transposition of the Great Arteries. Henrietta Kotlus Rosenberg, M.D., and Santiago Paredes, M.D., Department of Radiology, Boston City Hospital, Boston, Massachusetts.

Transposition of the great arteries accounts for the majority of deaths in infants less than 1 year of age with cyanotic congenital heart disease. The clinical findings as well as the anatomic basis including the embryologic development of the normal heart were described.

The roentgenologic features of the various types of transposition (complete, corrected, and partial) and the current methods of therapy (balloon septostomy, Blalock and Hanlon, and Mustard procedures) were presented. The text was accompanied by many roentgenograms and colorful illustrative diagrams.

Biliary-Duodenal Dynamics in Man: Cineroentgenographic and Manometric Studies Following Administration of Drugs and Hormones. Julian O. Salik, M.D., Charles I. Siegel, M.D., and Albert I. Mendeloff, M.D., Sinai Hospital, Baltimore, Maryland.

Characteristic responses of the function of the common duct and duodenum to various drugs and hormones were obtained, using cineroentgenography, 70 mm. films, as well as intraluminal manometry, performed concurrently.

These findings (films and manometric studies) were presented in the exhibit.

Roentgenologic and Radioisotopic Diagnosis of Perihepatic Abscess. Roger C. Sanders, M.D., Sanford D. Minkin, M.D., and A. Everette James, Jr., M.D., Johns Hopkins Hospital, Baltimore, Maryland.

The detection and evaluation of abscesses in and around the liver require a multidisciplined approach. The clinical symptoms are often masked by antibiotic therapy and the primary responsibility for diagnosis often rests with proper use of radioisotopic and roentgenographic procedures.

Characteristic scans and roentgenographic patterns of subphrenic, subhepatic, intrahepatic, and lesser sac abscesses were presented. An approach stressing the proper sequential selection of diagnostic studies was described.

Normal Chest . . . or Normal Chest????? Austin Robert Sandrock, M.D., University Hospitals, Iowa City, Iowa.

Selected lesions were presented which may be overlooked in a single posteroanterior chest roentgenogram if the heart is not adequately penetrated. Some of the lesions are common; others are rare or unusual. The purpose of the exhibit was to refresh the memory and widen the differential diagnosis.

Roentgen Evaluation of the Paranasal Sinuses in Children. Charles E. Shopfner, M.D., and Jorge Omar Rossi, M.D., University of Alabama in Birmingham, Birmingham, Alabama.

Sinus roentgenograms were evaluated in children: with suspected sinusitis; upper respiratory infection without sinusitis; and

without either suspected sinusitis or upper respiratory infection.

Mucosal thickening, opacity and fluid occurred equally in children younger than 6 years of all the 3 groups. Children older than 6 years with upper respiratory infection had roentgen pathology more often than those with suspected sinusitis. Only 6 of 120 children had acute sinusitis, but the roentgen findings were nonspecific and did not affect treatment.

If sinus roentgenograms are to be utilized in general pediatric practice, the omnipresence and relationship of the roentgen pathology to upper respiratory infection must be recognized.

Uterine Phlebography: Methods and Diagnostic Findings. Philip W. Silverberg, M.D., Gilbert S. Melnick, M.D., Eugene J. Slowinski, M.D., and Norman Reiss, M.D., St. Michael's Medical Center, Newark, New Jersey.

The exhibit demonstrated the transmyometrial technique of uterine phlebography, supplemented by selective left ovarian vein phlebography when necessary.

Normal anatomy and variations, varicosities of the broad ligament, the "ovarian vein syndrome" with right hydronephrosis, and pelvic thrombosis with thrombophlebitis were shown.

The illustrations consisted of approximately 30 roentgenograms, 2 Kodachrome enlargements and several anatomic drawings.

Roentgenology of the Ileocecal Valve. Bruce D. Smith, M.D., and Welland F. Short, M.D., University of Pittsburgh, Pittsburgh, Pennsylvania.

The variable appearance of the normal ileocecal valve may present a perplexing diagnostic problem. Failure to appreciate normal variations of this structure and the ways in which it is affected by disease may lead to misinterpretation.

The roentgenographic appearance of normal and abnormal ileocecal valves, the distinguishing features, and the techniques which may help in differentiation were presented as a guide to accurate interpretation.

Liver Calcification, a new Entity? Clement A. Smith, M.D., and Charles B. Cuono, M.D., West Virginia University Medical Center, Morgantown, West Virginia.

This exhibit was based on 105 cases of unusual liver calcification. Previous description of these lesions has not been found. The character and site of the lesions tend to be characteristic. The cause and nature of the disease is not known and the histologic study up to present is not informative.

The Pediatric Skull: Variations and Artifacts. Leonard E. Swischuk, M.D., and Charles J. Fagan, M.D., University of Texas Medical Branch, Galveston, Texas.

The skull of the infant and older child is an endless source of interest because of its numerous normal variations. To be sure, there is probably as much difficulty in interpreting these findings as in the pathologic change.

It is mandatory that the roentgenologist have a firm foundation in the recognition of such findings; this exhibit outlined most of the common, and some of the uncommon, normal variations and artifacts.

Mammary Duct Injection. Barbara Threatt, M.D., Elizabeth Schmidt, M.D., and Henry Appelman, M.D., University of Michigan Medical Center, Ann Arbor, Michigan.

Mammary duct injection is a safe, simple, nonpainful procedure for the evaluation of spontaneous nipple discharge. Visualization and localization of the duct with discharge is of great value to the surgeon, since most significant nipple discharges (serous or hemorrhagic) present without an associated mass.

The technique of mammary duct injection was shown, along with the normal anatomy and some normal variations. Benign and malignant duct disease was demonstrated and correlated with mammographic and pathologic case material.

Population Exposure to X-Rays: 1964 and 1970 Studies. John C. Villforth, M.S.,

Samuel Sperling, M.S., William S. Cole, M.D., Joseph N. Gitlin, M.S., and Paul L. Roney, M.D., Bureau of Radiological Health, FDA, U.S. PHS, DHEW, Rockville, Maryland.

This exhibit was based on a PHS study designed to provide information on the x-ray experience of the U.S. population. It is essentially a repetition of the 1964 study to compare: the frequency and rate of persons examined and treated by x-rays; the number of various types of examinations and procedures; and the number of films per radiographic, fluoroscopic, or dental examination, to list a few examples of the parameters under study. Estimates of the genetically significant dose were also compared with the 1964 data.

Comparisons with the 1964 data revealed significant reductions in radiation exposure of the U.S. population from medical and dental roentgenography.

Cineroentgenographic Evaluation of Prosthetic Cardiac Valves. Alan F. White, M.D., and Robert E. Dinsmore, M.D., Massachusetts General Hospital, Boston, Massachusetts.

A study was made of 41 patients with prosthetic cardiac valves who were clinically suspected of having a paravalvular leak.

Cineroentgenography was carried out in each case to determine the degree of tilt along the long axis of the prosthetic valve. Aortic root angiography was performed in all patients with aortic valve prostheses and left ventricular angiography, and in all those with prosthetic mitral valves to confirm or exclude the presence of a paravalvular leak.

Nonleaking mitral prostheses were found to have a wider range of tilt in the normal than nonleaking aortic prostheses, because of a difference in the supporting structures in the aortic annulus and mitral annulus.

A valve tilt greater than 6° for a prosthetic aortic valve, and a tilt greater than 12° for a prosthetic mitral valve were determined to be strong evidence of separation of the prosthesis from the valve bed with a paravalvular leak.

Radiographic Manifestations of Meckel's Diverticulum. Alan F. White, M.D., Kook Sang Oh, M.D., Alfred L. Weber, M.D., and A. Everette James, Jr., M.D., Massachusetts General Hospital, Boston Massachusetts and Johns Hopkins Medical Institutions, Baltimore, Maryland.

Meckel's diverticulum is a common condition and 25 per cent of the patients will develop symptoms from its complications. Most of these patients have surgery without radiographic examination. The diagnosis can definitely be made and often suggested by radiographic examination.

The radiographic manifestations of Meckel's diverticulum are: (1) gas within the diverticulum; (2) abscess formation or fluid (blood) in the abdomen from perforation; (3) small bowel obstruction; (4) demonstration of the diverticulum; (5) intussusception; (6) persistent separation of bowel loops about an inflamed Meckel's diverticulum; (7) persistent fecal umbilical fistula; (8) uptake of Tc^{99m} by gastric mucosa lining a Meckel's diverticulum; and (9) angiographic demonstration of bleeding from a Meckel's diverticulum in the small bowel.

An example of each of these manifestations was presented together with a discussion of the embryology, pathology, and clinical presentation.

Pharmacoangiography of the Splanchnic Venous System. Warren C. Widrich, M.D., David L. Nordahl, M.D., and Alan H. Robbins, M.D., Veterans Administration Hospital, Boston, Massachusetts.

The use of pharmacologic techniques, especially the 10 minute postepinephrine celiac arteriogram and the use of papaverine in the superior mesenteric artery, have decidedly improved visualization of the splenic, superior mesenteric, and portal veins in the Exhibitors' experience of over 200 splanchnic angiographies.

Pharmacoangiograms and discussion of lesions enhanced by these techniques were presented.

Vasodilator Response to Contrast Agents in Parallel Vascular Beds. Gerald L. Wolf, Ph.D., M.D., Ward A. Chambers, M.D., Joe R. Stock, B.S., and William J. Wilson, M.D., University of Nebraska Medical Center, Omaha, Nebraska.

The physiology of vascular stenosis and the pharmocologic response to contrast agents are important aspects of angiography. Very large stenoses are necessary in many vascular beds before resting flow is diminished. However, there are significant and proportional losses of vasodilator reserve with even small stenoses. As the degree of stenosis increases, the vascular bed becomes more dependent upon perfusion pressure to maintain a nutrient flow.

• A model was presented describing the potentiation of the deleterious pharmacologic effects of contrast agents (vasodilatation, hypotension, and histotoxicity) when the stenotic bed must compete with parallel vascular beds possessing greater dilator reserve.

Chronic Temporomandibular Joint Syndrome. H. Y. Yune, M.D., J. Robert Hall, DDS, Charles R. Hutton, DDS, and Eugene C. Klatte, M.D., Indiana University Medical Center, Indianapolis, Indiana.

The roentgenograms of 74 patients with chronic temporomandibular pain were reviewed. Initially in an earlier group of 43 patients, conventional and open mouth roentgenograms were obtained. The roentgenographic technique in the last 31 patients was altered to include a submentovertex view, lateral zonography, and serial multidirectional laminography of both temporomandibular joints.

Utilizing only conventional roentgenographic studies, 14 of 43 patients had demonstrable pathology. With complete roentgenographic studies, 25 of 31 patients were noted to have anatomic changes.

JAMES F. MARTIN, M.D.
Chairman
Committee on Scientific Exhibits

THE TECHNICAL EXHIBITS

The Technical Exhibits of the Seventy-third Annual Meeting of the American Roentgen Ray Society, held October 3–6, 1972, were of great artistic display and, because of the many splendid advances achieved during the past year, they contributed immeasurably to the over-all success of the Annual Meeting. They were located in the ultramodern Convention Hall of the Washington Hilton Hotel, which also housed the Scientific Exhibits. This was a very fortunate arrangement, since it afforded the members and the many guests of the Society ample opportunity to simultaneously study both the technical and scientific aspects of the latest and most timely developments in radiology and allied fields.

The Society is greatly indebted to Dr. James C. Cook, Manager, and Dr. George A. Kling, Associate Manager of the Annual Meeting for assembling and organizing these magnificent Technical Exhibits, and expresses its sincere thanks to the following participating firms: Access Corporation, Cincinnati, Ohio; Ames Color File Corporation, Somerville, Massachusetts; Atomic Energy of Canada Limited, Ottawa, Ontario; Automatic Seriograph, Division of Litton Industries, Des Plaines, Illinois; Barnes-Hind Pharmaceuticals, Inc., Sunnyvale, California; Bar-Ray Products, Inc., Brooklyn, New York; Becton-Dickinson, Division of Beckton, Dickinson and Company, Rutherford, New Jersey; CGR Medical Corporation, Baltimore, Maryland; Cook, Inc., Bloomington, Indiana; Cordis Corporation, Miami, Florida; Digital Equipment Corporation, Maynard, Massachusetts; Dunlee Corporation, Bellwood, Illinois; Du Pont Company, Wilmington, Delaware; Eastman Kodak Company, Rochester, New York; Eimac Division of Varian, Salt Lake City, Utah; Electro Catheter Corporation, Rahway, New Jersey; Elec-Trol International Inc., Lauderdale-by-the-Sea, Florida; Elema-Schonander, Inc., Elk Grove Village, Illinois; Encyclopaedia Britannica, Inc., Chicago, Illinois; Eureka X-Ray Tube Corporation, Chicago, Illinois; E-Z EM Company, Inc., Westbury, New York; Field Emission Corporation, McMinnville, Oregon; Flow Pharmaceuticals, Inc., Palo Alto, California; GAF Corporation, New York, New York; General Electric Company, Milwaukee, Wisconsin; Gordon Consultants, Inc., New York, New York; Grune & Stratton, Inc., New York, New York; Hoechst Pharmaceuticals, Inc., Somerville, New Jersey; Ilford Inc., Paramus, New Jersey; Katum Corporation, Boulder, Colorado; Keuffel & Esser X-Ray, Inc., Teterboro, New Jersey; Liebel-Flarsheim Company, Cincinnati, Ohio; J. B. Lippincott Company, Philadelphia, Pennsylvania; LogEtronics Inc., Springfield, Virginia; Low X-Ray Corporation, Valhalla, New York; The Machlett Laboratories, Inc., Stamford, Connecticut; Mallinckrodt Chemical Works, St. Louis, Missouri; MDT Instrument Company, South Salt Lake City, Utah; Medical Plastics Laboratory, Inc., Gatesville, Texas; Medi-Tech, Inc., Watertown, Massachusetts; Medrad Inc., Allison Park, Pennsylvania; Memco Inc., Riverside, New Jersey; Micro X-Ray Recorder, Inc., Chicago, Illinois; Neutron Products, Inc., Dickerson, Maryland; N & H Instruments, Inc., Carrollton, Texas; Nuclear Associates, Inc., Westbury, New York; Pako Corporation, Minneapolis, Minnesota; Philips Medical Systems, Inc., Shelton, Connecticut; Picker Corporation, Cleveland, Ohio; Profexray, Divison of Litton Medical Products, Inc., Des Plaines, Illinois; Radiographic Specialties, Oceanside, New York; Radx Corporation, Houston, Texas; S & S X-Ray Products, Inc., Brooklyn, New York; W. B. Saunders Company, Philadelphia, Pennsyl-

vania; Frank Scholz X-Ray Corporation, Needham Heights, Massachusetts; Siemens Corporation, Iselin, New Jersey; Spectrotherm Corporation, Mountain View, California; Spectrum X-Ray Corporation, Westville, New Jersey; E. R. Squibb & Sons, Inc., Princeton, New Jersey; Texas Instruments Inc., Houston, Texas; Charles C Thomas, Publisher, Springfield, Illinois; Unified Micromed Systems, Reading, Pennsylvania; United States Radium Corporation, Brooklyn, New York; The Williams & Wilkins Company, Baltimore, Maryland; Winthrop Laboratories, New York, New York; Wolf X-Ray Corporation, Jamaica, New York; Xerox—Xeroradiography, Pasadena, California; Year Book Medical Publishers, Chicago, Illinois.

NEWS ITEMS

NEW OFFICERS OF THE AMERICAN ROENTGEN RAY SOCIETY

At the Seventy-third Annual Meeting of the American Roentgen Ray Society, held at the Washington Hilton Hotel, Washington, D. C., October 3–6, 1972, the following new officers were elected: *President:* J. Scott Dunbar, M.D., Vancouver, B.C., Canada; *President-Elect:* William B. Seaman, M.D., 622 W. 168th Street, New York, New York 10032; *1st Vice-President:* Robert G. Fraser, M.D., Montreal, P.Q., Canada; *2nd Vice-President:* Walter M. Whitehouse, M.D., Ann Arbor, Michigan; *Secretary:* Ted F. Leigh, M.D., Emory University Clinic, Atlanta, Georgia 30322; and *Treasurer:* Colin B. Holman, M.D., Mayo Clinic, Rochester, Minnesota 55901.

The Chairman of the Executive Council is Ralph M. Scott, M.D., 500 S. Floyd, Louisville, Kentucky 40208.

The Seventy-fourth Annual Meeting of the Society will be held at the Queen Elizabeth Hotel, Montreal, P.Q., Canada, September 25–28, 1973.

SOCIETY FOR PEDIATRIC RADIOLOGY

At the Fifteenth Annual Meeting of the Society for Pediatric Radiology held at the Washington Hilton Hotel, Washington, D. C., October 1–2, 1972, the following officers were elected: *President:* Donald H. Altman, M.D., Miami, Florida; *President-Elect:* Hooshang Taybi, M.D., Children's Hospital Medical Center of Northern California, 51st & Grove Streets, Oakland, California 94609; *Secretary-Treasurer:* John L. Gwinn, M.D., 4650 Sunset Boulevard, Los Angeles, California 90027.

The Sixteenth Annual Meeting of the Society will be held at the Queen Elizabeth Hotel, Montreal, P.Q., Canada, September 23–24, 1973.

ROCKY MOUNTAIN RADIOLOGICAL SOCIETY

At the last Annual Meeting of the Rocky Mountain Radiological Society, the following new officers for 1973 were elected: *President:* Verne G. LaTourette, Jr., M.D., Denver, Colorado; *President-Elect:* W. Kemp Absher, M.D., Pueblo, Colorado; *1st Vice-President:* William E. Jobe, M.D., Denver, Colorado; *2nd Vice-President:* Jerome D. Sutherland, M.D., Denver, Colorado; *Secretary-Treasurer:* Lorenz R. Wurtzebach, M.D., Denver, Colorado 80220; and *Historian:* H. Milton Berg, M.D., Bismarck, North Dakota.

The next Annual Meeting of the Society will be held in Denver at the Brown Palace Hotel on August 2, 3, and 4, 1973.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS

The Association of University Radiologists (AUR) wishes again to announce an Annual Memorial Medal Award that will be given to the radiology resident writing the best original paper. The paper can represent the work of one or more investigators, but the resident submitting the paper must qualify as the senior author.

The winner of the competition will be invited to present his work at the Annual Meeting of the AUR and will receive a monetary award in addition to the medal.

Manuscripts to be considered for the 1973 competition must be received by March 1, 1973. Inquiries should be addressed and papers submitted to: Melvyn H. Schreiber, M.D., Department of Radiology, University of Texas Medical Branch, Galveston, Texas 77550.

The next AUR Annual Meeting will be in Vancouver, B.C., Canada, May 9–12, 1973.

INSTITUTE FOR PEDIATRIC RADIOLOGY

The Institute for Pediatric Radiology and Chas. E. Shopfner, M.D. will present 2 spring meetings on the Current Status of Pediatric Radiology series.

"Radiology of the Pediatric Chest" will

be presented March 10–11, 1973 at the Royal Sonesta Hotel in New Orleans, La.; "Radiology of the Pediatric Gastrointestinal Tract" will be presented May 5–6, 1973 at the Prom Sheraton Motor Hotel in Kansas City, Mo.

For information please write Institute for Pediatric Radiology, 4148 North Cleveland Avenue, Kansas City, Missouri 64117.

COURSE IN PEDIATRIC GENITOURINARY RADIOGRAPHY

The Department of Radiology of the University of Utah School of Medicine will sponsor a course on "Pediatric Genitourinary Radiography," April 14 through 17, 1973, at the Las Vegas Hilton Hotel in Las Vegas.

Small groups of approximately 12 participants will meet daily in morning and afternoon 2 hour seminars, rotating among the various faculty members. Other than short introductory sessions, no formal lectures will be given, with each faculty member directly supervising detailed case analysis by the participants, allowing personal faculty-participant interaction.

For further information, please write to Patrick D. Lester, M.D., University of Utah Medical Center, Salt Lake City, Utah 84112.

SYMPOSIUM ON PEDIATRIC RADIOLOGY

UNIVERSITY OF KENTUCKY,
LEXINGTON, KENTUCKY

A three day symposium to be held May 2–4, 1973, will deal with many practical problems in the diagnosis of abdominal, chest, and skeletal disease in childhood. A distinguished Guest and University of Kentucky Faculty will join in presenting the conference, organized to meet the needs of practicing pediatricians and radiologists.

The Guest Faculty includes: David H. Baker, M.D., New York, New York; Lawrence A. Davis, M.D., Louisville, Kentucky; John Dorst, M.D., Baltimore, Maryland; Herman Grossman, M.D., Durham, North Carolina; John Gwinn, M.D., Los Angeles, California; Herbert Kaufmann, M.D., Philadelphia, Pennsylvania; M. B. Ozonoff, M.D., Newington, Connecticut; Edward Singleton, M.D., Houston, Texas; and John Tampas, M.D., Burlington, Vermont.

Please address inquiries to Frank R. Lemon, M.D., Continuing Education, College of Medicine, University of Kentucky, Lexington, Kentucky 40506.

ADVANCES IN PEDIATRIC RADIOLOGY

The Indiana University School of Medicine, Indianapolis, Indiana, will present, May 16–18, 1973, a Pediatric Radiology Postgraduate Course dealing with the recent developments in the field, including neonatology, angiography, neuroradiology, bone dysplasias, gastrointestinal and genitourinary radiology.

All sessions will be held in Emerson Auditorium of the Indiana University School of Medicine. Advance registration is requested.

In addition to members of the Local Faculty the Guest Faculty includes: David H. Baker, M.D., Columbia-Presbyterian Medical Center, New York; J. Scott Dunbar, M.D., Faculty of Medicine, University of British Columbia; Herman Grossman, M.D., Duke University Medical Center; Derek Harwood-Nash, M.D., Ch.B., FRCP(C), Hospital for Sick Children, Toronto, Canada; William McSweeney, M.D., University of Cincinnati School of Medicine; William Northway, M.D., Stanford University School of Medicine; Andrew K. Poznanski, M.D., University of Michigan Medical Center; Frederic Silverman, M.D., University of Cincinnati School of Medicine; and Hooshang Taybi, M.D., University of California School of Medicine.

For further information please write to: Mr. Steven D. Berkshire, Program Coordinator, Division of Postgraduate Medical Education, Indiana University School of Medicine, 1100 West Michigan Street, Indianapolis, Indiana 46202.

NUCLEAR CARDIOLOGY

The Divisions of Nuclear Medicine and Cardiology of the Johns Hopkins Hospital announce a symposium titled "Nuclear Cardiology."

This symposium will consider the use of nuclear medicine techniques in clinical cardiology. Both the practical aspects and the areas of future development will be emphasized by a Faculty of Invited Guests.

The symposium will last for 2½ days and occur on April 12, 13, and 14, 1973.

For further information please write to: A. Everette James, Jr., Sc.M., M.D., Department of Radiology, Johns Hopkins Hospital, Baltimore, Maryland 21205.

POSTGRADUATE COURSE IN NEURORADIOLOGY

Columbia-Presbyterian Medical Center

The Department of Radiology, College of Physicians and Surgeons, Columbia University, announces a Postgraduate Course in Neuroradiology to be held at the Columbia-Presbyterian Medical Center, April 30–May 4, 1973, under the direction of Ernest H. Wood, M.D.

The course is a comprehensive review of diagnostic neuroradiology and is designed primarily for radiologists, neurologists and neurologic surgeons. Emphasis will be placed on basic information used frequently in neuroradiologic diagnosis, especially angiography.

One day will be devoted to pediatric neuroradiology.

Topics of the course will also include plain film roentgenographic diagnosis, pneumography, myelography, radiology of the orbits, and neuroradiologic approaches to the solution of specific clinical problems.

Inquiries should be addressed to: Melvin D. Yahr, M.D., Associate Dean, Office of Graduate Medicine, College of Physicians & Surgeons, 630 West 168 Street, New York, N. Y. 10032.

BOOK REVIEW

RADIOLOGY OF THE SKULL AND BRAIN: THE SKULL. Volume I. In 2 books. Edited by Thomas H. Newton, M.D., Professor of Radiology, Neurology and Neurosurgery; Chief, Section of Neuroradiology, University of California School of Medicine, San Francisco Medical Center, San Francisco, Calif.; and D. Gordon Potts, M.D., Professor of Radiology, Cornell Medical College; Attending Radiologist, The New York Hospital, New York, N. Y. Cloth. Pp. 874, plus an index, with 2,314 illustrations. Price, $78.50. C. V. Mosby Company, 3207 Washington Boulevard, St. Louis, Mo. 63103, 1971.

This 2 book set represents the first volume of a 3 volume opus which, when completed, will represent a veritable encyclopedia of Neuroradiology. The rapid growth of this subspecialty necessitated the production of a work which would gather together the tremendous mass of accumulated information and distill it into a unified, comprehensive, and readable presentation. In this first volume, the distinguished editors and their 55 expert contributors have succeeded most admirably. Its 873 pages contain the most exhaustive and comprehensive treatment to date of the radiographic characteristics of the normal skull and of the many disease processes and syndromes which affect the skull. Most important for a radiologic publication, the 41 chapters are profusely illustrated. The illustrations are large, the quality of reproduction is generally good, and the legends are clear.

The first book and a small portion of the second book are devoted to the normal skull and to a consideration of alterations in size, shape, thickness, and texture. There are detailed discussions of skull maturation, cranial sutures and fontanelles, and basal foramina. The individual seeking a differential diagnosis for a particular roentgen finding will certainly be satisfied in these pages.

The remainder of the second book utilizes a disease oriented approach and includes sections on congenital anomalies, metabolic diseases, tumors, trauma, and infection. The chapters on craniofacial anomalies, Lückenschädel, and skull trauma were particularly impressive to this reader.

This dual format, with the first 570 pages devoted to radiographic findings and their differential diagnosis and the last 300 pages structured according to disease processes, leads to a moderate degree of overlap and repetition. However, these are not necessarily bad characteristics since real learning must involve repetition to achieve understanding. Furthermore, since one opens a volume such as this to look up particular details or to try this or that subject, there is little danger of finding the coverage of any topic to be redundant. Consistent with the rest of the volume, the index appears to be complete and well organized, enabling the reader to quickly locate his subject of interest.

An introductory chapter on the history of Neuroradiology was most interesting to this reviewer, but seemed to be somewhat slanted toward the European contributions to the development of this field with less than adequate credit for the more recent North American renaissance in Neuroradiology. Some of the best chapters were written by outstanding, well recognized, and experienced American and Canadian authors who have made important investigative contributions in a variety of subjects and whose chapters on those subjects in this volume can best be described as definitive.

The editors state that this volume and the two to follow (Volume 2 on angiography and Volume 3 on air studies, isotope studies, and other diagnostic techniques) are primarily intended for physicians interested in the roentgenologic diagnosis of neurologic problems. This magnificent first volume on the skull is an outstanding reference work and can be highly recommended not only to neuroradiologists, but also to students and experienced clinicians in diagnostic radiology, neurology, and neurosurgery.

STEPHEN A. KIEFFER, M.D.

BOOKS RECEIVED

MAMMOGRAPHY. Second edition. By Robert L. Egan, M.D., Professor of Radiology and Chief, Mammography Section, Department of Radiology, Emory University School of Medicine, Atlanta, Ga. Cloth. Pp. 498, with many illustrations. Price, $35.50. Charles C Thomas, Publisher, 301–327 East Lawrence Avenue, Springfield, Ill. 62717, 1972.

Vectorcardiography in Congenital Heart Disease: a Method for Estimating Severity. By R. Curtis Ellison, M.D., Associate in Cardiology, Children's Hospital Medical Center; Assistant Professor of Pediatrics at the Children's Hospital, Harvard Medical School, Boston; and Norma J. Restieaux, M.B., Ch.B., F.R.A.C.P., M.R.C.P. (London), Senior Lecturer in Medicine and Cardiologist, University of Otago Medical School, New Zealand; Formerly, Fellow in Cardiology, Children's Hospital Medical Center, Boston. Cloth. Pp. 209, with 138 illustrations. Price, $13.50. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1972.

Early Care of the Injured Patient. By the Committee on Trauma; American College of Surgeons. Cloth. Pp. 441, with 200 illustrations. Price, $9.00. W. B. Saunders Company, West Washington Square, Philadelphia, Pa. 19105, 1972.

Medical Specialty Terminology: X-ray and Nuclear Medicine. Volume 2. By Clara Gene Young, Retired Technical Editor and Writer (Medical), U. S. Civil Service; former Instructor, Medical Terminology, Good Samaritan Hospital, Phoenix, Ariz.; and Joseph J. Likos, M.D., F.C.A.P., Chief, Laboratory Service and Nuclear Medicine Veterans Administration Hospital, Phoenix, Ariz. Cloth. Pp. 284, with 124 illustrations. Price, $15.75. C. V. Mosby Company, 11830 Industrial Drive, St. Louis, Mo. 63141, 1972.

The Analysis of the Venous System. By John Ludbrook, M.D., Professor of Surgery, University of Adelaide, South Australia. Standardization of Cardio-Angiological Methods: 1. Paper. Pp. 94, with 24 illustrations. Price, Fr. 23.-/ DM 21.-. Hans Huber Publishers, Marktgasse 9, Länggassstrasse 76, Box CH 3000 Berne 9, Switzerland, 1972.

The Role of Chromosomes in Cancer Biology. By Peo C. Koller. Volume 38 of Recent Results in Cancer Research. Cloth. Pp. 122, with 42 figures. Price, $15.30. Springer-Verlag New York, Inc., 175 Fifth Avenue, New York, N. Y. 10010, 1972.

Fédération des Sociétés de Gynécologie et d'Obstétrique de Langue Française, 24th Congrès. First report: La Prévention des Immunisations Foeto-maternelles. Second report: Incontinence Urinaire d'Effort. Round table: De la Contamination du Nouveau-Né Pendant et Après l'Accouchement. Paper. Pp. 136, with some figures. Supplement 1 to Journal de Gynécologie, Obstétrique et Biologie de la Reproduction. Masson et Cie, Paris, France, 1972.

Radiology in Renal Disease. Introduction by Professor R. E. Steiner. British Medical Bulletin, September, 1972, Volume 28, Number 3. Paper. Pp. 73. The British Council, 97 & 99 Park Street, London W1Y 4HQ, England, 1972.

SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. Ted F. Leigh, Emory University Clinic, Atlanta, Ga. 30322. Annual Meeting: Queen Elizabeth Hotel, Montreal, P. Q., Canada, September 25–28, 1973.

AMERICAN RADIUM SOCIETY
Secretary, Dr. Felix N. Rutledge, M. D. Anderson Hospital and Tumor Institute, Houston, Texas 77025. Annual Meeting: Broadmoor Hotel, Colorado Springs, Colorado, April 22–26, 1973.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. Hillier L. Baker, Jr., Mayo Clinic, Rochester, Minn. 55901. Annual Meeting: Palmer House, Chicago, Ill., November 26–December 1, 1972.

AMERICAN COLLEGE OF RADIOLOGY
Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago, Ill. 60606. Annual meeting: San Francisco, Calif., April 2–7, 1973.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. Antolin Raventos, Davis, Calif. Annual Meeting: New York, N. Y., June 24–28, 1973.

AMERICAN BOARD OF RADIOLOGY
Secretary, Dr. C. Allen Good. Correspondence should be directed to Kahler East, Rochester, Minn. 55901.

Oral examinations will be held in the following cities during the next 2 years: Atlanta, Ga., Dec. 4–8, 1972, Sheraton-Biltmore Hotel; Chicago, Ill., June 4–8, 1973, Marriott Motor Hotel; and Dallas, Tex., Dec. 3–7, 1973, Statler Hilton Hotel.

Written examinations are scheduled in 13 large centers.

Applications must be received in the Board Office before Sept. 30 of the year preceding the one in which the candidate wishes to be examined.

Deadline for filing applications for any examination in 1973 was September 30, 1972.

AMERICAN BOARD OF NUCLEAR MEDICINE, INC. A Conjoint Board of the American Boards of Internal Medicine, Pathology and Radiology and sponsored by the Society of Nuclear Medicine.

Chairman, Dr. Merrill A. Bender, Suite 2402, 211 E. 43rd St., New York, N. Y. 10017.

Examination for certification in the Specialty of Nuclear Medicine will be given April 7, 1973. The deadline for filing application forms is January 1, 1973.

AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE
Secretary, D. John Wright, Ph.D., Department of Radiology, Temple Univ. Health Sciences Center, Philadelphia, Pa. 19140. Annual Meeting in Chicago, Ill., Sheraton-Blackstone Hotel, Nov. 30 and Dec. 1, 1972.

AMERICAN SOCIETY OF THERAPEUTIC RADIOLOGISTS
Secretary, Dr. Herman D. Suit, Radiation Medicine, Massachusetts General Hospital, Boston, Mass. 02114. Annual Meeting.

AMERICAN INSTITUTE OF ULTRASOUND IN MEDICINE
Secretary, John M. Reid, Ph.D., 556 18th Ave., Seattle, Wash. 98122. Annual Meeting.

AMERICAN SOCIETY OF NEURORADIOLOGY
Secretary, Dr. David O. Davis, Department of Radiology, 901 Twenty-third St., N. W., Washington, D. C. 20037. Annual Meeting, Boston, Mass., April 26–28, 1973.

THIRTEENTH INTERNATIONAL CONGRESS OF RADIOLOGY
President-Elect, Dr. Juan Gómez López. *Secretary General*, Dr. José Bonmatí, Lagasca, 27 Madrid 1, España. Meeting: Madrid, Spain, Oct. 15–20, 1973.

INTERNATIONAL SOCIETY OF RADIOLOGY
Hon. Secretary-Treasurer, Prof. Eric Samuel, Radiologist-in-Charge, The Royal Infirmary, Edinburgh EH3 9Y-W, Scotland.

ELEVENTH INTER-AMERICAN CONGRESS OF RADIOLOGY
Counselor for the United States, Dr. Juan A. del Regato, Penrose Cancer Hospital, 2215 North Cascade Ave., Colorado Springs, Colorado 80907. Meeting: Bogotá, Colombia in 1975.

INTER-AMERICAN COLLEGE OF RADIOLOGY
President, Dr. Miguel Dao y Dao, Apartado Postal 14213, Candelaria, Caracas, Venezuela, S.A.
Secretary General, Dr. Rubén Merenfeld, Instituto Diagnóstico, Apartado Postal 14213, Candelaria, Caracas, Venezuela, S.A. Meeting: Bogotá, Colombia in 1975.

THIRD CONGRESS OF THE EUROPEAN ASSOCIATION OF RADIOLOGY
Meeting in June, 1975 in Edinburgh, Scotland.

SECOND ASIAN AND OCEANIAN CONGRESS OF RADIOLOGY
Honorary Secretary, Dr. J. J. Martin, Box 805 F., G.P.O., Melbourne, 3001, Australia.
Meeting in 1975.

ALABAMA CHAPTER OF ACR
Secretary-Treasurer, Dr. Lowry R. Young, Jr., 905 Madison St., Huntsville, Ala. 35801. Meets time and place of Alabama State Medical Association.

ALASKA RADIOLOGICAL SOCIETY
Secretary, Dr. Bruce C. Wright, Providence Hospital, Anchorage, Alaska. Meets third Wednesday each month.

AMERICAN THERMOGRAPHIC SOCIETY
Secretary-Treasurer, Dr. Marc. S. Lapayowker, Department of Radiology, Temple Univ. Hosp., 3401 N. St., Philadelphia, Pa. 19140. Meets at New York-Hilton Hotel, June 23 and 24, 1973.

ARIZONA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Wesley S. Fee, 2421 E. 6th St., Tucson, Ariz. 85719. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

ARK-LA-TEX RADIOLOGICAL SOCIETY
Secretary, Dr. Erich K. Lang, Confederate Memorial Medical Center, LSU School of Medicine, Shreveport, La. 71101.

ARKANSAS CHAPTER OF ACR
Secretary-Treasurer, Dr. David H. Newbern, 4301 W. Markham, Little Rock, Ark. 72205. Meets twice annually, the Spring Meeting being in conjunction with and at the place of the State Medical Association.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS
Secretary-Treasurer, Dr. Melvyn H. Schreiber, 2909 Dominique Dr., Galveston, Tex. Annual Meeting, Vancouver, B. C., Canada, May 9–12, 1973.

ATLANTA RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Ernst Ruder, Dept. of Radiol., Crawford Long Memorial Hosp., Atlanta, Ga. 30308. Meets on four Thursday evenings during the academic year at a time announced in early September of each year, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

BAVARIAN-AMERICAN RADIOLOGIC SOCIETY
Secretary, Lucien F. Burke, Maj. MC, Radiol. Service, US Army Gen. Hosp., Frankfurt, APO, New York 09757.

BLOCKLEY RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. R. John Gould, 41 Lombardy Rd., Drexel Hill, Pa. 19026.

BLUEGRASS RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Eugene Slusher, Lexington

*** Secretaries of societies are requested to send timely information promptly to the Editor.**

Clinic, 1221 S. Broadway, Lexington, Ky. 40504. The Society meets once each month during the school year.

Bronx Radiological Society, New York State, Chapter ACR
Secretary-Treasurer, Dr. Leon J. Corbin, 1369 Rosedale Ave., Bronx, N. Y. 10472. Meets 4 times a year.

Brooklyn Radiological Society
Secretary-Treasurer, Dr. Sarah Lieberson, 1298 St. Marks Ave., Brooklyn, N. Y. 11213. Meets first Thursday of each month, October through June.

Buffalo Radiological Society
Secretary, Dr. Ru-Kan Lin, 191 North St., Buffalo, N. Y. 14201. Meets second Monday evening each month, October to May inclusive, at University Club.

California Radiation Therapy Association
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets semiannually in San Francisco and Los Angeles.

California Radiological Society, California Chapter of ACR
Secretary-Treasurer, Dr. John L. Gwinn, 4650 Sunset Blvd., Los Angeles, Calif. 90027.

Catawba Valley Radiological Society
Secretary, Dr. Emmett R. White, P.O. Box 10, Rutherford College, N. C. 28671. Meets every Thursday, Dept. of Radiology, Valdese General Hosp., Valdese, N. C., at 12:30 P.M.

Central New York Radiological Society
Secretary-Treasurer, Dr. John Sanborn, Radiology Department, Crouse-Irving Memorial Hosp., Irving Ave., Syracuse, N. Y. 13210. Meets first Monday each month October through May.

Central Ohio Radiological Society
Secretary-Treasurer, Dr. Terry Meyer, 175 S. Merkle Rd., Columbus, Ohio 43209. Meets second Thursday in October, November, January, and March 15, and May 19 at Fort Hayes Hotel, Columbus, Ohio.

Chicago Radiological Society, Division of the Illinois Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Raymond L. Del Fava, 355 Ridge Ave., Evanston, Ill. 60202. Meets third Thursday of each month, October to April, except December, at the Bismarck Hotel, Chicago, Ill.

Cleveland Radiological Society
Secretary-Treasurer, Dr. Daniel E. Wertman, 11311 Shaker Blvd., Cleveland, Ohio 44104. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and April.

Colorado Radiological Society, Chapter of ACR
Secretary, Dr. Jerome D. Sutherland, 3705 E. Colfax Ave., Denver, Colo. 80206. Meets third Friday of each month at Denver Athletic Club from September through May.

Connecticut Valley Radiologic Society
Secretary, Dr. Carl W. Scheer, 335 Cook Ave., Meriden, Conn. 06450. Meets in April and October.

Dallas-Fort Worth Radiological Society
Secretary-Treasurer, Dr. Norman L. Prealey, 222 Medical Tower, 1550 W. Rosedale, Fort Worth, Tex. 76104. Meets the 3rd Monday of every month at 6:30 P.M., at the Cibola Inn, Arlington, Tex.

Delaware Chapter of ACR
Secretary, Dr. Seymour R. Kaplan, Kent Gen. Hosp., Dover, Del. 19901.

East Bay Radiological Society
Secretary-Treasurer, Dr. Peter E. Kane, Children's Hosp. Med. Ctr., Oakland, Calif. 94609. Meets first Thursday each month, Oct. through May, at University Club, Oakland, Calif.

East Tennessee Radiological Society
Secretary-Treasurer, Dr. George H. Wood, 205 Medical Arts Bldg., Knoxville, Tenn. 37902. Meets in January and September.

Florida Radiological Society, Chapter of ACR
Secretary, Dr. Ralph C. Aye, Tampa General Hospital, Tampa, Fla. 33606. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

Florida West Coast Radiological Society
Secretary-Treasurer, Dr. J. C. Hewitt, 1 Davis Blvd., Tampa, Fla. 33606. Meets in January, March, May, July, September and November.

Georgia Radiological Society, Chapter of ACR
Secretary, Dr. Richard S. Colvin, Emory University Clinic, Atlanta, Ga. 30322. Meets in spring and fall at Annual State Society Meeting.

Greater Cincinnati Radiological Society
Secretary-Treasurer, Dr. Owen L. Brown, 2421 Auburn Ave., Cincinnati, Ohio 45219.

Greater Louisville Radiological Society
Secretary-Treasurer, Dr. L. D. LeNeave, 315 E. Broadway, Louisville, Ky. 40202. Meets monthly.

Greater Miami Radiological Society
Secretary-Treasurer, Dr. Stanley I. Worton, 1400 N.W. 10th Avenue, Miami, Florida 33136. Meets monthly, third Wednesday at 8:00 P.M. at various member hospitals, Miami, Fla.

Greater St. Louis Society of Radiologists
Secretary-Treasurer, John J. Lang, 507 Fairways Circle, St. Louis, Mo. 63141.

Hawaii Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ghim L. Yeoh, 1481 King St., Ste. 202, Honolulu, Hawaii 96814. Meets third Monday of each month at 7:30 P.M.

Health Physics Society
Secretary, John H. Pingel, Argonne National Laboratory, 9700 S. Cass Ave., Argonne, Ill. 60439. Annual Meeting: Deauville Hotel, Miami Beach, Fla., June 17–21, 1973.

Houston Radiological Society
Secretary, Dr. Neill B. Longley, 6436 Fannin, Suite 601, Houston, Tex. 77025. Meets fourth Monday of each month, except June, July, August and December, at 6:00 P.M., at 103 Jesse H. Jones Library Building, Texas Medical Center, Houston, Tex. 77025.

Idaho State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Hugh P. Smith, Jr., 130 E. Bannock, Boise, Id. 83702. Meets in the spring and fall.

Illinois Radiological Society, Inc., Chapter of ACR
Secretary, Dr. B. Jay Hill, Mercy Hospital, Stevenson at King Drive, Chicago, Ill. 60616. Meets in the spring and fall.

Indiana Roentgen Society, Inc., Chapter of ACR
Secretary, Dr. L. Ray Stewart, 852 S. Alvord Blvd., Evansville, Ind. 47714.

Iowa Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John Huston Jr., 1948 First Ave. N.E., Cedar Rapids, Iowa 52402. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

Kansas Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ralph H. Baehr, 310 Medical Arts Bldg., Topeka, Kan. 66604. Meets in spring with State Medical Society and in winter on call.

Kentucky Chapter of ACR
Secretary-Treasurer, Dr. Joseph G. Whelan, Jr., 1313 St. Antony Place, Louisville, Ky. 40204. Meets in April and September.

Kings County Radiological Society
Secretary, Dr. Sidney Hendler, 1880 Ocean Ave., Brooklyn 30, N.Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

Knoxville Radiological Society
Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

Long Island Radiological Society
Secretary, Dr. Jerome Zwanger, 1200 Stewart Ave., Garden City, N. Y. 11533. Meets monthly.

LOS ANGELES RADIOLOGICAL SOCIETY
Secretary, Dr. Gabriel H. Wilson, UCLA Center for Medical Sciences, Los Angeles, Calif. 90024. Meets second Wednesday of month in September, November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif. Midwinter Radiological Conference, Century Plaza Hotel, Los Angeles, Calif. 90067, Feb. 2–4, 1973.

LOUISIANA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Robert T. Cook, Southern Baptist Hosp., 2700 Napoleon Ave., New Orleans, La. 70115.

LOUISIANA-TEXAS GULF COAST RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Edward A. Sheldon, 109 Doctors Bldg., Beaumont, Tex. 77701.

MAINE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Richard W. Taylor, Radiology Department, St. Mary's General Hospital, Lewiston, Maine 04240. Meets in June, September, December and April.

MARYLAND RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. George Burke, 4000 North Charles St., Baltimore, Md. 21218.

MASSACHUSETTS RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Norman L. Sadowsky, The Faulkner Hosp., 1153 Centre St., Jamaica Plain, Mass. 02130.

MEMPHIS ROENTGEN SOCIETY
Secretary-Treasurer, Dr. Jerry C. Phillips, The University of Tennessee College of Medicine, Department of Radiology, Walter C. Chandler Bldg., 865 Jefferson Ave., Memphis, Tenn. 38103. Meets first Monday Oct. through May at the University Club.

MIAMI VALLEY RADIOLOGICAL SOCIETY
Secretary, Dr. Darwood B. Hance, Reid Memorial Hospital, Richmond, Ind. Meets third Thursday of fall, winter and spring months at 7:30 P.M. at Miami Valley Hospital, Dayton, Ohio.

MICHIGAN RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. George A. Kling, Harper Hospital, 3825 Brush St., Detroit, Mich. 48201. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

MID-HUDSON RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Herbert S. Berlin, Hopewell Junction, N. Y. Meets 7:00 P.M., first Wednesday of each month, September to May.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary-Treasurer, Dr. Thomas C. Lipscomb, 1004 North Tenth St., Milwaukee, Wis. 53233. Meets monthly on fourth Monday, October through May, at University Club.

MINNESOTA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Harlan W. Hawkinson, 6 Buffalo Rd., St. Paul, Minn. 55113. Meets twice annually, fall and winter.

MISSISSIPPI STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Nancy W. Burrow, 1327 Peachtree St., Jackson, Miss. 39202. Meets third Thursday of each month at the Heidelberg Hotel, Jackson, at 6:00 P.M.

MISSOURI RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Ronald G. Evens, Mallinckrodt Institute of Radiology, 510 S. Kingshighway, St. Louis, Mo. 63110.

MONTANA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Dexter W. Johnson, Columbus Hosp., Great Falls, Mont. 59401.

NEBRASKA CHAPTER OF ACR
Secretary-Treasurer, Dr. John T. McGreer, III, 924 Sharp Bldg., Lincoln, Nebr. 68508. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

NEVADA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. William G. Arbonies, 887 Marsh, Reno, Nev. 89520.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. Stefan C. Schatzki, 1180 Beacon St. Brookline Mass. 02146. Meets the third Friday of each month, October through April, excluding December, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 4:30 P.M.

NEW HAMPSHIRE ROENTGEN RAY SOCIETY, CHAPTER OF ACR
Secretary-Treasurer, Dr. Edward P. Kane, Claremont Gen. Hosp., Claremont, N. H. 03743. Meets four to six times yearly.

NEW MEXICO SOCIETY OF RADIOLOGISTS, CHAPTER OF ACR
Secretary, Dr. Carole R. Simmons, Dept. of Radiol., Presbyterian Hospital Center, Albuquerque, N. M. 87106. Four meetings annually, three held in Albuquerque, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.

NEW ORLEANS RADIOLOGICAL SOCIETY, INC.
Secretary-Treasurer, Dr. Jimmy L. Mains, P.O. Box 446, Gretna, La. 70053. Meets bimonthly at local restaurants selected by the President.

NEW YORK ROENTGEN SOCIETY
Secretary-Treasurer, Dr. Howard R. Gould, 153 West 11th Street, New York, N. Y. 10011. Meets monthly on the 3rd Monday at the New York Academy of Medicine at 4:30 P.M. Annual Spring Conference, Waldorf-Astoria Hotel, New York, N. Y., Apr. 26–28, 1973. Further information may be obtained from Dr. Albert A. Dunn, Roosevelt Hosp., New York, N. Y. 10019.

NEW YORK STATE CHAPTER OF ACR
Secretary-Treasurer, Dr. John J. Magovern, 74 N. Village Ave., Rockville Centre, N. Y. 11570.

NORTH CAROLINA CHAPTER OF ACR
Secretary-Treasurer, Dr. James F. Martin, 300 S. Hawthorne Road, Winston-Salem, N. C. 27103.

NORTH DAKOTA RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Marshall Landa, 1702 13th St., So., Fargo, N. D. 58102. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.

NORTH FLORIDA RADIOLOGICAL SOCIETY
Secretary, Dr. David F. Bew, University Hospital of Jacksonville, 655 W. Eighth Street, P.O. Box 2751, Jacksonville, Florida 32203. Meets quarterly in March, June, September and December.

NORTHEASTERN OKLAHOMA RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Theodore J. Brickner, Jr., 5919 S. Gary Place, Tulsa, Okla. 74110.

NORTHEASTERN NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Robert C. Friedman, 325 Presidential Way, Guilderland, N. Y. 12085. Meets in Albany area on third Wednesday of October, November, March, April, and May.

NORTHERN CALIFORNIA RADIATION THERAPY ASSOCIATION
Secretary-Treasurer, Dr. James R. Eltringham, Stanford Medical Center, Stanford, Calif. 94304. Meets quarterly.

NORTHERN CALIFORNIA RADIOLOGICAL SOCIETY
Secretary, Dr. J. Richard McProuty, 6620 Coyle Ave., Carmichael, Calif. 95608. Meets fourth Monday of Nov., Jan., and March at the Mansion Inn, Sacramento, Calif.

NORTHWESTERN NEW YORK RADIOLOGICAL SOCIETY
Secretary, Dr. Barbara E. Chick, Glens Falls Hospital, Glens Falls, N. Y. 12801.

NORTHWESTERN OHIO RADIOLOGICAL SOCIETY
Secretary, Dr. Vito J. Zupa, Mercy Hospital, Department of Radiology, Toledo, Ohio.

OHIO STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. Thomas F. Ulrich, Barberton Citizens Hospital, Barberton, Ohio 44203.

OKLAHOMA STATE RADIOLOGICAL SOCIETY, CHAPTER OF ACR
Secretary, Dr. George H. Ladd, 4616 Denison, Muskogee, Okla. 74401. Meets in January, May and October.

Orange County Radiological Society
Secretary, Dr. Norman Arnett, 400 North Pamona, Fullerton, Calif. 92632. Meets on fourth Tuesday of the month, excluding June, July, August, and December, at the Orange County Medical Association Bldg., Orange, Calif.

Oregon State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Ronald W. Philips, 2305 Washington, Milwaukie, Ore. 97222. Meets on second Wednesday of month, October through April, at the University Club, Portland, Ore.

Orleans Parish Radiological Society
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans, La. 70113. Meets second Tuesday of each month.

Pacific Northwest Radiological Society
Secretary-Treasurer, Dr. Robert S. Miller, 13753 S.W. Farmington Rd., Beaverton, Oregon 97005. Annual meeting: Benson Hotel, Portland, Ore., May 11–13, 1973.

Pennsylvania Radiological Society, Chapter of ACR
Secretary, Dr. Theodore A. Tristan, Harrisburg Polyclinic Hosp., Harrisburg, Pa. 17105.

Philadelphia Roentgen Ray Society
Secretary, Dr. Howard M. Pollack, Department of Radiology, Episcopal Hospital, Philadelphia, Pa. 19125. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.

Pittsburgh Roentgen Society
Secretary, Dr. Stephen C. Bruno, Shadyside Hospital, 5230 Centre Ave., Pittsburgh, Pa. 15232. Meets second Wednesday of month, October through June, at Park Schenley Restaurant.

Radiation Research Society
Executive Secretary, Richard J. Burk, Jr., 4211 39th St., N.W., Washington, D. C. 20016. Annual Meeting: St. Louis, Mo., April 29–May 3, 1973. International Congress of Radiation Research, Seattle, Wash., July 14–20, 1974.

Radiological Society of Connecticut, Inc., Chapter of ACR
Secretary-Treasurer, Dr. Carl W. Scheer, 335 Cook Ave., Meriden, Conn. 06450. Meetings are held quarterly.

Radiological Society of Greater Cincinnati
Secretary-Treasurer, Dr. Alvin Nathan, c/o Academy of Medicine, 320 Broadway, Cincinnati, Ohio 45202. Meets first Monday of each month at Cincinnati Academy of Medicine.

Radiological Society of Greater Kansas City
President-Secretary, Dr. Arch W. Templeton, Univ. of Kansas Med. Ctr., Kansas City, Kansas 66103. Meets 5 times a year on given dates.

Radiological Society of Louisiana, Chapter of ACR
Secretary-Treasurer, Dr. Robert T. Cook, 1820 Octavia St., New Orleans, La. 70115. Meets semiannually during Louisiana State Medical Society meeting and 6 months later.

Radiological Society of New Jersey, Chapter of ACR
Secretary, Dr. Sidney Ketyer, St. Elizabeth Hosp., 225 Williamson St., Elizabeth, N. J. 07207. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.

Radiological Society of Rhode Island, Chapter of ACR
Secretary-Treasurer, Dr. James B. Leach, Jr., 115 Cass Ave., Woonsocket, R. I. 02895.

Radiological Society of Southern California
Secretary-Treasurer, Dr. Marshall Rowen, Dept. Radiol., St. Joseph Hosp., 1100 Stewart Dr., Orange, Calif. 92668. Meets three times a year, usually October, February and May.

Radiological Society of the State of New York
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester, N. Y. 14618.

Redwood Empire Radiological Society
Secretary, Dr. Lee F. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.

Richmond County Radiological Society
Secretary, Dr. Donald P. King, 218 Gun Club Rd., Richmond, Va. 23221. Meets first Thursday of each month at various hospitals.

Rochester Roentgen Ray Society, Rochester, N. Y.
Secretary-Treasurer, Dr. Kenneth E. Robinson, 1441 East Avenue, Rochester, New York 14610. Quarterly meetings on the call of the President, at the Rochester Academy of Medicine.

Rocky Mountain Radiological Society
Secretary-Treasurer, Dr. Lorenz R. Wurtzebach, 4200 E. Ninth Ave., Denver, Colo. 80220. Annual meeting: Brown Palace Hotel, Denver, Colo., Aug. 2–4, 1973.

San Antonio-Civilian Military Radiological Society
Secretary, Dr. Robert M. Maurer, Santa Rosa Medical Center, San Antonio, Tex. 78207. Meets third Wednesday of each month at Fort Sam Houston Officers' Club at 6:30 P.M.

San Diego Radiological Society
President-Secretary, Dr. James M. Lee, Scripps Clinic and Research Found., 476 Prospect St., La Jolla, Calif. 92037. Meets first Wednesday of each month at the Town & Country Hotel.

San Francisco Radiological Society
Secretary-Treasurer, Dr. James G. Moore, Mary's Help Hospital, 1900 Sullivan Ave., Daly City, Calif. 94015.

Section on Radiology, California Medical Association
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.

Section on Radiology, Medical Society of the District of Columbia
Secretary-Treasurer, Dr. Louis Wener, Cafritz Memorial Hosp., 1310 Southern Ave., S.E., Washington, D. C. 20032. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.

Section on Radiology, Southern Medical Association
Secretary, Dr. Mario A. Calonje, 1514 Jefferson Highway, New Orleans, La. 70121. Annual Meeting.

Section on Radiology, Texas Medical Association
Secretary, Dr. George F. Crawford, St. Elizabeth Hospital, Beaumont, Tex. Meets annually with the Texas Medical Association.

Shreveport Radiological Club
Secretary, Dr. W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.

Society for Pediatric Radiology
Secretary, Dr. John L. Gwinn, Children's Hospital, 4614 Sunset Blvd., Los Angeles, Calif. 90027. Annual meeting: Queen Elizabeth Hotel, Montreal, P. Q., Canada, September 23–24, 1973.

Society of Gastrointestinal Radiologists
Secretary-Treasurer, Dr. Wm. B. Seaman, The Presbyterian Hospital, Columbia-Presbyterian Medical Center, 622 W. 168th St., New York, N. Y. 10032.

Society of Nuclear Medicine
Secretary, Dr. E. James Potchen, Edward Mallinckrodt Institute of Radiology, 660 S. Euclid Ave., St. Louis, Mo. 63110. *Administrative Officer*, Mrs. Margaret Glos, 211 E. 43rd St., New York, N. Y. 10017. Annual meeting: Americana Hotel, Miami Beach, Fla., June 12–15, 1973.

South Bay Radiological Society
Secretary, Dr. A. D. Benjamin, 596 Carroll St., Sunnyvale, Calif. 94086. Meets the 2nd Monday of each month at the Santa Clara County Medical Association Bldg., 700 Empey Way, San Jose, Calif.

South Carolina Radiological Society, Chapter of ACR
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.

South Coast Radiological Society
Secretary-Treasurer, Dr. James T. McClintock, P.O. Box 837, Santa Barbara, Calif. 93102. Meets fourth Monday of the month, Oct. through May.

South Dakota Radiological Society, Chapter of ACR
Secretary, Dr. M. F. Petereit, 2416 S. Phillips, Sioux Falls, S. D. 57105. Meets in spring with State Medical Society and in fall.

Southern California Radiation Therapy Society
Secretary-Treasurer, Dr. John Byfield, 1000 West Carson St., Torrance, Calif. 90502. Meets quarterly.

Southern Radiological Conference
Secretary-Treasurer, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 7544, Mobile, Ala. 36607. Seventeenth Annual Meeting: Grand Hotel, Point Clear, Ala., Jan. 26–28, 1973.

Southwestern Radiological Society
Secretary, John M. McGuire, 304 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.

Tennessee Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Carl T. Stubblefield, Bedford County Hospital, Columbia, Tenn. 38401. Meets annually at the time and place of the Tennessee State Medical Association meeting.

Texas State Radiological Society, Chapter of ACR
Secretary, Dr. Herman C. Sehested, 815 Medical Tower, Room 100, 1550 W. Rosedale St., Fort Worth, Tex. 76104. Annual meeting.

The Fleischner Society
President, Dr. Robert G. Fraser, 1555 Summerhill Ave., Montreal 109, P.Q., Canada. Annual meeting.

Tri-State Radiological Society
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.

Upper Peninsula Radiological Society
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.

Utah State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John F. Stucki, Department of Radiology, McKay-Dee Hosp., 3939 Harrison Blvd., Ogden, Utah 84403. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.

Vermont Radiological Society, Chapter of ACR
Secretary, Dr. J. Lorimer Holm, R.R. #3, Barre, Vt. 05641.

Virginia Chapter of ACR
Secretary-Treasurer, Dr. James S. Redmond, Suite 7, Medical Center, Lynchburg, Va. 24501. Next Meeting: Nov. 6, 1972 at the Conference Ctr., Williamsburg, Va.

Washington, D. C., Chapter of ACR
Secretary-Treasurer, Dr. Albert M. Zelna, 9110 Seven Locks Rd., Bethesda, Md. 20014.

Washington State Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. John N. Roehr, 1011 Medical Dental Bldg., Seattle, Wash. 98101.

West Virginia Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Andrew W. Goodwin, II, 200 Med. Arts Bldg., Charleston, W. Va. 25301. Meets concurrently with annual meeting of West Virginia State Medical Society; other meetings arranged by program committee.

Westchester County Radiological Society
Secretary, Dr. Max C. King, 314 Woodland Hills Rd., White Plains, N. Y. 10603. Meets on second Tuesday of October, December, February and May.

Wisconsin Radiological Society, Chapter of ACR
Secretary-Treasurer, Dr. Marvin L. Hinke, 630 S. Central Ave., Marshfield, Wisc. 54449. Meets twice a year, May and September.

Wisconsin Society of Therapeutic Radiologists
Secretary, Dr. Robert W. Ecland, Gundersen Clinic, LaCrosse, Wisc. 54601. Meets quarterly.

Wyoming Radiological Society, Chapter of ACR
Secretary, Dr. Ross J. Collie, Box 96, Lander, Wyo. 82520. Meets in fall with State Medical Society and in spring on call of President.

Mexico, Puerto Rico and Central America

Asociación Costarricense de Radiología
Secretary, Dr. Jorge Vargas Segura, Apartado 5367, San José, Costa Rica.

Asociación de Radiólogos de Centro America y Panamá. Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá
Secretary-General, Dr. Francisco Mirambell, Apartado 3352, San José, Costa Rica, Central America.
Meets annually in a rotating manner in the six countries.

Asociación Puertorriqueña de Radiología
Secretary, Dr. R. E. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.

Sociedad de Radiología de El Salvador
Secretary, Dr. Carlos Mejia, 5a Av. Nte. No. 434, San Salvador, Rep. El Salvador.

Sociedad de Radiología de Guatemala
Secretary, Dr. Carlos E. Escobar, 9ª. Calle A 0-05, Zona 1, Guatemala.

Sociedad Mexicana de Radiología, A.C.
Coahuila No. 35, México 7, D.F., México.
Secretary-General, Dr. Jorge Cano Coquí. Meets first Monday of each month.

Sociedad Radiológica Panameña
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.

Sociedad Radiológica de Puerto Rico
Secretary, Dr. Heriberto Pagán Sáez, Box 9387, Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.

British Commonwealth of Nations

Association of Radiologists of the Province of Quebec
Moyne, 121 Boul. Taschereau, Greenfield Park, P.Q., Canada. Meets four times a year.

British Institute of Radiology
Honorary Secretary, Dr. T. Kerr, 32 Welbeck St., London, W1M/7PG, England. Meets monthly from October until May.

Canadian Association of Physicists, Division of Medical and Biological Physics
Honorary Secretary-Treasurer, Dr. R. G. Baker, Ontario Cancer Foundation, Ottawa Civic Clinic, 1053 Carling Ave., Ottawa 3, Ont., Canada.

Canadian Association of Radiologists
Honorary Secretary, Dr. Champlain Charest, 1555 Summerhill Ave., Montreal 109, Que., Canada. Annual meeting: Montreal, Que., Canada, March 11–17, 1973.

Edmonton and District Radiological Society
Secretary, Dr. L. A. Koller, Suite 360, Professional Bldg., 10830 Jasper Ave., Edmonton 15, Alberta, Canada. Meets third Thursday of each month October to May, except December, at various Edmonton Hospitals.

Faculty of Radiologists
Honorary Secretary, Robert Morrison, 47 Lincoln's Inn Fields, London, W.C.2, England. The scientific meetings held in January, February and March 1973 will be combined sessions with the British Institute of Radiology and the Section of Radiology of The Royal Society of Medicine.

Faculty of Radiologists, Royal College of Surgeons in Ireland
Hon. Secretary, Dr. D. F. Canwell, 123 St. Stephens Green, Dublin 2. Ireland.

Montreal Radiological Study Club
Secretary, Dr. W. Paul Butt, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets third Friday each month at 8:15 P.M. at the Royal Society of Medicine, 1 Wimpole St., London, W. 1 M&AE, England.

Société Canadienne-Française de Radiologie
Secretary General, Dr. Hubert Sasseville, Verdun Hospital, Montréal, P.Q., Canada. Meets every third Tuesday from October to April.

The Hospital Physicists' Association
Honorary Secretary, J. B. Massey, B.Sc., Christie Hosp. and Holt Radium Institute, Physics Department, Withington, Manchester M20 9BX, England.

Toronto Radiological Society
Secretary, Dr. George Wortzman, Toronto General Hosp., Toronto 12, Ont., Canada. Meets second Monday of each month, September through May.

Royal College of Radiologists of Australasia
Honorary Secretary, Dr. T. P. Loneragan, 147 Macquarie St., Sydney, N.S.W. 2000, Australia.

South America

Colégio Brasileiro de Radiologia
Secretary-General, Dr. Miguel Mario Céntola, Caixa Postal 5984, São Paulo, Brazil.

Federación Argentina de Sociedades de Radiología
Branches of the Federation are: Sociedad de Radiólogos de la Provincia de Córdoba; Sociedad Argentina de Radiología; Asociación Argentina de Radiología; Sociedad de Radiología, Radioterapéutica y Medicina Nuclear de Rosario; Sociedad de Radiología y Medicina Nuclear del Centro y Sudeste de la Provincia de Buenos Aires (Mar del Plata); Sociedad Salteña de Radiología y Medicina Nuclear; Sociedad de Radiología de Tucumán; Sociedad de Radiología del Nordeste Argentino; Sociedad de Radiología de La Plata; and Sociedad de Radiología de San Juan.
Secretary-General-Treasurer, Dr. Roberto Habichayn, Av. Colón 637, Córdoba, Argentina.

Sociedad Argentina de Radiología
Secretary-General, Dr. Armando B. de Onaindia, Santa Fé 1171, Buenos Aires, Argentina. Meets first Wednesday evening, April through December.

Sociedad Boliviana de Radiología
Secretary, Dr. Javier Prada Méndez, Casilla 1182, La Paz, Bolivia. Meets monthly. General assembly once every two years.

Sociedade Brasileira de Radiologia
Secretary, Dr. Armando Rocha Amoédo, Caixa Postal 1532, Rio de Janeiro, Brazil. General Assembly meets every two years in December.

Sociedade Brasileira de Radioterapia
Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.

Sociedad Chilena de Radiología
Secretary, Dr. Claudio Cortés, Casilla, 13426 Santiago, Chile. Meets first Friday of each month at Av. Santa María 1810 at 7:00 P.M.

Sociedad Colombiana de Radiología
Secretary-General, Dr. Rodrigo Caro Molina, Cali, Colombia. Meets last Thursday of each month.

Sociedad Ecuatoriana de Radiología
Secretary, Dr. Carlos Palau Jimenez, Casilla 5469, Guayaquil, Ecuador.

Sociedad Paraguaya de Radiología
Secretary, Dr. Hugo Volpe Rios, 15 de Agosto 322, Asunción, Paraguay.

Sociedad Peruana de Radiología
Secretary-General, Dr. Miguel Chiappori Cambana, Av. General Santa Cruz No. 315, Miraflores, Lima, Perú, Casilla Correo, 2306, Lima, Perú. Meets monthly except during January, February, and March.

Sociedad de Radiología del Atlantico
Secretary, Dr. Raul Fernandez, Calle 40 #41-110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiología.

Sociedad de Radiología del Nordeste Argentino
Secretary-General, Dr. Francisco J. Velar, Catamarca 561, Corrientes, Argentina.

Sociedad de Radiología de La Plata
Secretary, Dr. Hiram D. Haurigot, Calle 50 No. 374, La Plata, Argentina.

Sociedad de Radiología, Cancerología y Física Médica del Uruguay
Secretary-General, Dr. Manuel Gonzáles Maseda, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.

Sociedade de Radiología de Pernambuco
Secretary, Dr. Manoel Medeiros Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.

Sociedad de Roentgenología y Medicina Nuclear de la Provincia de Córdoba
Secretary-General, Dr. Lucas C. Di Rienzo, Ave. Grl. Paz. 151, Córdoba, Argentina.

Sociedad de Radiología, Radioterapéutica y Medicina Nuclear, de Rosario
Secretary-General, Dr. Federico Frágola, Santa Fe 1798, Rosario, Argentina.

Sociedad Salteña de Radiología y Medicina Nuclear
Secretary, Dr. Julio Luis Baldi, Av. Sarmiento 536, Salta, Argentina.

Sociedad Venezolana de Radiología y Medicina Nuclear
Secretary-General, Dr. Salvador Itriago Borgas, Apartado Postal 9213, Candelaria, Caracas, Venezuela. Meets monthly, third Friday at Colegio Médico del Distrito Federal, Caracas.

Continental Europe

Österreichische Röntgen-Gesellschaft
Secretary, Dr. Ernst Kotcher, Wiener Allgemeine Poliklinik Mariannengasse 10, A-1090 Wien, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik. The 1973 Annual Meeting is a joint meeting with the Deutsche Röntgengesellschaft and will be held in Vienna, April 12–14, 1973.

Société Royale Belge de Radiologie
General Secretary, Dr. Joseph Baeyens, Alost, Belgium. Meets in February, March, May, June, September, October, November and December.

Société Européenne de Radiologie Pédiatrique
Secretary, Dr. P. Hicken, The Children's Hosp., Ladywood Middleway, Birmingham B16 8ET, Great Britain. Tenth Annual Meeting, Birmingham, April 11–13, 1973.
Permanent Secretary, Clément Fauré, Hôpital des Enfants-Malades, 149, rue de Sèvres, 75 Paris 15e, France.

Société Française de Radiologie Médicale, Médecine Nucléaire et Electrologie, and its branches: Société du Sud-Ouest, du Littoral méditerranéen, du Centre et Lyonnais, du Nord, de l'Ouest, de l'Est, et d'Alger et du Massif Central. Parent Society meets third Monday of each month, except during July, August and September, rue de Seine 12, Paris, France.
Secretary-General, Dr. Ch. Proux, 9 rue Daru Paris 8e, France.

Société Française de Neuroradiologie
Secretary-General, Professeur agr. R. Djindjian 16, rue de l'Université 75, Paris 7e, France. Annual meeting.

Československá Radiologická Společnost
Secretary, Associate Professor Jaromír Kolář, M.D., Sc.D., Radiological Clinic, Praha 2, U nemocnice 2, Czechoslovakia. Meets monthly except during July, August and September. Annual general meeting.

Deutsche Röntgengesellschaft
President, Professor Dr. med. Walter Frommhold, Director of Medizinisches Strahleninstitut der Universität Tübingen, Röntgenweg 11, 7400 Tübingen, Germany.

The 1973 Annual Meeting is a joint meeting with the Österreichische Röntgengesellschaft and will be held in Vienna, April 12–14, 1973.

Società Italiana di Radiologia Medica e Medicina Nucleare
Administrative Secretary, Prof. E. Conte, Ospedale Mauriziano, 10128 Torino, Italy. Meets annually.

Societas Radiologica Danica
Secretary, Dr. Bent Langefeldt, Tetommervej 20a, DK-8240 Risskov, Denmark.

Nederlandse Vereniging voor Radiologie
Secretary, Dr. J. D. Mulder, Dept. of Rad., Akademisch Ziekenhuis, Leiden, Netherlands.

Scandinavian Radiological Society
Secretary-General, Professor C. E. Unnérus, M.D., Hagalung-Tapiola, Havsvindsvägen 5 C., Finland.

Sociedad Española de Radiología y Electrología Médicas y de Medicina Nuclear
Secretary, Dr. José Bonmatí, Villanueva, 11, Madrid 1, España. Meets every second Friday of each month, Oct. to June, inclusive, in Madrid.

Sociedade Portuguesa de Radiologia e Medicina Nuclear
Secretary-General, Dr. Luís Aires de Sousa, Av. Elias Garcia, 123- 1, Esg.-Lisboa i-Portugal.

Schweizerische Vereiningung für Radiologie, Nuklearmedizin und Strahleneiologie (Association Suisse de Radiologie, Médecine Nucléaire et de Radiobiologie)
Secretary, Dr. med. Gustav Schoch, Bethesdaspital, Gellerstrasse 144, 4000 Basel, Switzerland.

Asia

Indian Radiological Association
Secretary, Dr. L. R. Parthasarathy, 44 Constable Road, Madras—23, India.

Indonesian Radiological Society
Secretary, Dr. Gani Iljas Sasmitaatmadja, Radiology Dep., Faculty of Medicine, University of Indonesia, Salemba 6, Djakarta, Indonesia.

Iranian Radiological Society
Secretary, Dr. Issa Yaghmai, P.O. Box No. 14-1151, Teheran, Iran. The Society meets on the second Saturday of each month. Third National Iranian Congress of Radiology: Isfahan, Iran, May 5–10, 1973. Deadline for acceptance of papers Feb. 20, 1973.

Israel Radiological Society
President, Dr. I. Izkovitch, 2 Wingate Ave., Beit Harofe, Haifa, Israel.

Japanese American Radiological Society
Secretary, Thomas J. Imray, Maj. MC, Chief, Radiol. Dep., US Army Hosp., Camp Zama, Japan, APO San Francisco, Calif. 96343. Meets 3rd Thursday, monthly at various locations in Tokyo.

Philippine College of Radiology
Secretary-Treasurer, Dr. Edmundo V. Villacorta, Box 1284, Commercial Center, Makati, Rizal D-708, Philippines. Meets every 3 weeks on Fridays. Annual convention: Jan. 1973, Manila.

Radiological Society of Thailand
Secretary, Prof. Rojana Suvansuthi, M.D., Ph.D., Depart. of Rad., Sirizaj Hospital, Faculty of Medicine, Mahidol University, Bangkok 7, Thailand.

Africa

Association of Radiologists of West Africa
Honorary Secretary, Dr. T. M. Kolawole, University College Hospital, University of Ibadan, Ibadan, Nigeria.

Radiological Society of South Africa
Secretary, Dr. B. Rosenberg, P.O. Box 3200, Johannesburg, South Africa.

South African International Radiological Congress
Director, Dr. Paul Sneider, P.O. Box 4878, Johannesburg, South Africa.

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ABSTRACTS OF RADIOLOGIC LITERATURE

E. FREDERICK LANG, M.D., Editor

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ROENTGEN DIAGNOSIS

Head

Kloss, K. Wachsende Schädelfrakturen. (Growing fractures of the skull.) *Wien. klin. Wchnschr.*, Apr., 1972, *84*, 229–231. (Address: Neurochirurgische Abteilung der Chirurgischen Universitätsklinik, A-6020, Innsbruck, Austria.)

Linear skull fractures in the newborn and the small infant are often not identified roentgenographically because of the thinness of the cranial tables. Nevertheless, sharp edges of the fragments tend to cut easily into the attached dura mater and even into the underlying brain tissue. When the elastic fragments return to a perfect apposition, nothing unusual may be suspected. In due course of time, however, interposition of the lacerated dura and brain takes place. Finally, the pressure from the cerebrospinal fluid causes an increasing gap of the fragments so that a typical meningocele develops.

Roentgenologically, a characteristic skull defect manifests itself already 4 weeks after the injury. In the differential diagnosis, a cephalohematoma and a true encephalocele have to be excluded. These conditions, however, present as midline lesions, whereas the fractures are predominantly located laterally.

Clinically, cerebral concussion may be observed in the beginning to be followed by a pulsating prolapse. Aphasia, hemiparesis, and finally, traumatic epilepsy develop in 10 to 15 per cent of the cases.

The presented material comprised 5 pertinent cases. The patients' ages ranged from 3 to 21 months. Large fluctuating masses, measuring up to 3 by 12 cm., were found. A traumatic history was elicited in all instances, but a fracture line could not be identified at first. Instead, a characteristic bone defect developed in the temporoparietal area together with an overlying cystic mass.

In 4 patients, an operation was performed. The procedure included repair of the laceration with a dural flap. In addition, bone chips were used for filling-in the skull defect. A second operation became necessary in 1 patient 3 years later because originally the defect was believed to be too small for using bone chips. In the fifth patient, the parents refused an operation. As a result, the child suffered posttraumatic epilepsy and died 2 years after the injury.—*Ernest Kraft, M.D.*

Psenner, L. Die fibröse Knochendysplasie im Bereiche der Nasennebenhöhlen. (Fibrous dysplasia of the bone in the region of the paranasal sinuses.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Apr., 1972, *116*, 456–469. (Address: Röntgendiagnostisches Institut der Universität Wien, A-1095 Wien, Alserstrasse 4, Austria.)

Fibrous dysplasia of the facial bones can usually be diagnosed from the plain roentgenograms, although tomography may occasionally be very helpful. An osteoma of the maxilla or facial bone and a meningioma of the sphenoid bone can cause a problem in differential diagnosis, but the age and clinical course usually clarify the condition. Fibrous dysplasia is a disease of childhood and early age groups, but may manifest itself only after the third or fourth decades. Multiplicity of lesions is in favor of a diagnosis of fibrous dysplasia.

In this condition, the bone is thickened and the lesion has a sharp margin, although the contour is irregular or wavy. The marrow is replaced by a thick fibrous connective tissue, which forms masses of sclerotic bone. Characteristic of older lesions are the so-called homogeneous "calcareous pearls" (sometimes only seen on tomography). Cystic rarefaction, while fairly common elsewhere, is rarely noted in the lesions of the skull and facial bones.

In fibrous dysplasia, the apparent cloudiness of the maxillary antrum (or frontal sinus) is due to the increased thickness of the osseous wall. Confusion with the cloudiness caused by sinusitis is less likely: if there is no history of infection; if the findings were present for a long time without change; if the adjacent bone is densely and diffusely sclerotic; and if eccentric encroachment on the air space is noted (when there is no residual air space, differentiation from sinusitis may be impossible without an adequate history).—*J. Zausner, M.D.*

Russell, D. B., and Miller, Jack D. R. Orbital venography. *Radiology*, May, 1972, *103*, 267–273. (From: Department of Radiology, University of Alberta Hospital, Edmonton, Alberta, Canada.)

The authors review the anatomy of the orbital veins, the technique of orbital venography and their experience with 21 cases involving orbital venography.

The procedure is performed by puncture of a frontal vein of the forehead. An elastic band about the head above the needle occludes drainage into the scalp. The patient compresses the infraorbital ridges and side of nose to occlude facial drainage. Approximately 10 ml. of meglumine iothalamate 60 per cent is injected and 3 roentgenograms at 1 per second in anteroposterior, angled anteroposterior and lateral projections are obtained. Basal projection may be used to visualize the cavernous sinus.

The findings of orbital venography are grouped into 5 categories:

Endocrine exophthalmos. This finding is related to different degrees of increased intraorbital pressure. The superior orbital vein is either not visualized, or the flow is decreased. The occurrence is usually bilateral.

Mass. The relative mobility of the superior oph-

thalmic vein allows for early displacement by a mass. The orbital venogram may localize and estimate its size.

Inflammation. Attenuation and displacement of the superior orbital vein may occur with orbital cellulitis.

Venous malformation. Orbital varices and congenital venous malformation make up this group.

Cavernous sinus. This study aids in assessing the pathology in this area and in the preoperative measurement of the sellar width.

The indications have been primarily to demonstrate venous angiomas of the orbit. In addition, valuable information regarding orbital mass lesions, such as the quadrant of the eye affected and size of the lesion, may be obtained. Suspected cavernous sinus disease may also be studied by this method.—*J. L. Montgomery, M.D.*

JORDAN, CHARLES E., JAMES, A. EVERETTE, JR., and HODGES, FRED J., III. Comparison of the cerebral angiogram and the brain radionuclide image in brain abscess. *Radiology*, Aug., 1972, *104*, 327–331. (From: Department of Radiology, The Johns Hopkins Medical Institutions, Baltimore, Md.)

In spite of modern antibiotic therapy, brain abscess continues to be a serious problem. High mortality rates and neurologic deficits are associated. A correct diagnosis should be made as early as possible. The sensitivity, accuracy, and complementary nature of cerebral angiography and radionuclide brain imaging are presented.

Since January 1966, 20 patients with brain abscess had either one or both of these procedures performed at Johns Hopkins Hospital. The diagnosis was confirmed at surgery or autopsy.

Cerebral angiography was done percutaneously: right brachial or left carotid arteries, with renografin 60 and biplane film changer. Radionuclide brain imaging was performed after administration of 10–15 mc of Tc^{99m} intravenously, 1 mg. of atropine sulfate intramuscularly, and 200 mg. of potassium perchlorate orally. Images were obtained in the anterior, posterior, vertex, and both lateral planes.

No real age or sex predilection was recorded. The clinical presentations were varied, and so were the etiologic pathogens. Twenty-nine abscess cavities were found in the 20 patients, but only 1 was localized below the tentorium.

One or more radionuclide brain images were obtained in 17 of the 20 patients prior to surgery or autopsy. The study was always abnormal after the onset of neurologic symptoms. Multiple lesions were demonstrated, when present. Six patients had studies postoperatively, and the images correlated well with the postoperative course of the disease.

Nineteen of the 20 patients had cerebral angiography. Initially, 14 demonstrated focal abnormalities, 4 were negative, and 1 was unsuccessful. Of the 14 abnormalities, 12 had evidence of a mass (6 showing pathologic stains, 5 stains being ring-like), but the other 2 exhibited only localized flow abnormalities. Two studies suggested multiple masses. Repeat of 2 of the initially negative studies demonstrated abnormalities 3 weeks later.

The radionuclide brain image is positive early in the clinical course of brain abscess. Within this series of 20 cases, 3 positives were demonstrated without a concurrent positive angiogram. The less noxious radionuclide study is also useful in the postoperative period. Multiple lesions were more readily identified on radionuclide studies; however, the radionuclide brain image is histologically nonspecific.

The angiogram is more specific. Three specific abnormalities were noted: regional vascularity, particularly a ring-like stain; the rapid development of a mass; and a mass associated with a *small* arterial branch occlusion. Other findings of an avascular mass or early venous drainage are rather nonspecific.

Clearly, these studies should be used in a complementary manner to facilitate the establishment of the diagnosis of brain abscess.—*F. C. Petty, M.D.*

NECK AND CHEST

MA, HECTOR T. G. The radiologic signs of recurrent laryngeal nerve paralysis. *J. Canad. A. Radiologists*, June, 1972, *23*, 125–132. (Address: 24–26 Pottinger Street, 1/F, Hong Kong.)

Eleven cases of recurrent laryngeal nerve paralysis are reported. All cases are unrelated to any intrinsic malignant lesion of the larynx.

The larynx is studied with tomography and positive contrast laryngography. Tomographic exposures are performed with the patient inspiring deeply, phonating E, reversed phonating E, Valsalva maneuver, modified Valsalva maneuver, and panting.

Spot roentgenography and cineroentgenography are used for contrast laryngography.

The radiologic signs of recurrent laryngeal nerve paralysis are demonstrated by signs associated with cord atrophy, signs associated with failure of abduction of the cords, and signs associated with immobility of the pyriform fossa.

During tomography when one side is paralyzed, the true cord undergoes atrophy and the shape of the cord is changed from a lobular to a triangular appearance. Because of the atony of the surrounding musculature, the laryngeal ventricle on the paralyzed side dilates. The curvature or right angle which is maintained by the lateral wall of the upper trachea and the inferior surface of the true cord disappears and is replaced by a straight line in the subglottic area or the so-called paralytic curve. The cross sectional area of the cord is decreased.

The failure of abduction is seen on tomography as the normal side shows a double blurred image of the laryngeal structures and the paralyzed side shows good definition of a single image. The immobility is also demonstrated on the paralyzed side during laryngography.

In a normal patient, during inspiration, abduction of the laryngeal cords is associated with upward displacement of the pyriform fossa which also becomes smaller in volume. When one side is paralyzed, the pyriform fossa of the paralyzed side remains in a constant position and there is no change in the volume.

The most valuable radiologic sign of laryngeal nerve paralysis is failure of abduction; this sign was present in all 11 cases. Cord atrophy was demonstrated in 9 cases, and pyriform sinus immobility was present in 7 of the 11 cases.

The cine laryngogram was the most valuable in demonstrating recurrent laryngeal nerve paralysis.—*Ronald J. Ross, M.D.*

Forrest, John V. Radiographic findings in *pneumocystis carinii* pneumonia. *Radiology*, June, 1972, *103*, 539–544. (From: Mallinckrodt Institute of Radiology, Washington University School of Medicine, St. Louis, Mo.)

Pneumocystis carinii pneumonia is a protozoan infection primarily of the alveolar interspaces. This disease has been reported in the United States, recurring almost exclusively in patients with congenital or acquired immunologic defects, but is being found with increasing frequency as a common cause of diffuse progressive pneumonia in patients with treated reticuloendothelial neoplasms. The typical roentgenographic appearance is described as a diffuse, bilateral, progressively coalescing pneumonia which often spares the peripheral lung fields. The absence of this usual roentgenographic appearance has been thought to make the diagnosis of this disease unlikely.

The author summarizes the findings in 14 cases of pneumocystis pneumonia seen at the Washington University Medical Center and describes the spectrum of roentgenographic findings, many of which have not been previously reported.

Six cases are described in detail and the unusual roentgenographic findings in these cases are discussed. One case presented initially with 2 areas of localized lobar pneumonic processes and did not show evidence of diffuse involvement of the lungs until 12 days after admission. A significant pleural effusion has been felt to be strong evidence against a diagnosis of pneumocystis pneumonia, but in 2 cases significant effusions developed during the course of this disease. In 2 of the cases no infiltration was evident in areas of the lung which had previously received radiation therapy for intrapulmonary Hodgkin's disease. This was thought to be due to the post-irradiation changes in the lung which created an environment apparently unacceptable to the pneumocystis organism.

The roentgenographic differential diagnosis includes other opportunistic pneumonias including cytomegalic, virus and fungi, pulmonary edema, pulmonary hemorrhage, pulmonary alveolar proteinosis, aspiration pneumonia, and diffuse bronchopneumonia.

It is felt that since pneumocystis carinii pneumonia can be effectively treated by pentamidine therapy in many cases, early diagnosis is imperative and that unusual roentgenographic findings such as lobar distribution, pleural effusion, or sparing of areas of damaged lung should not cause this diagnosis to be disregarded.—*Donald N. Dysart, M.D.*

Gahl, K., Greiser, E., and Stender, H. St. Röntgenologische Befunde bei primär vaskulärer pulmonaler Hypertonie. (Roentgenologic findings in primary vascular pulmonary hypertension.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, May, 1972, *116*, 589–599. (Address: Prof. Dr. H. Stender, Institut für Klinische Radiologie, Medizinische Hochschule, 3 Hannover, Podbielskistrasse 380, Germany.)

Primary vascular pulmonary hypertension is characterized by pathologic changes of the peripheral branches of the pulmonary artery. A progressive narrowing of these vessels leads to an increased resistance of the blood flow with subsequent enlargement of the right heart and the hilar vessels. For proper evaluation of the chest roentgenograms in these cases the question arises how rapidly the changes become manifest and whether the roentgen findings can be correlated with the time and quantity factors.

For this purpose 28 proved cases were compared with other groups in which cardiac catheterization was also undertaken for the exclusion of pulmonary hypertension specially following medication with anoretics. Increased pulmonary pressure with enlargement of the right ventricle and the pulmonary artery was found in all proved cases.

A second group comprised 10 women with suspected pulmonary hypertension following ingestion of anoretics. The roentgen findings remained unremarkable.

In a third group of 5 patients, the pulmonary artery pressure increased slightly following exercise and anoretic medication.

The fourth group included 5 patients with normal findings under the same conditions.

A fifth group comprised 6 patients with normal catheter and roentgen findings for the purpose of comparison.

On the conventional roentgenograms various cardiac measurements and pulmonary structures were evaluated. In 12 patients, selective angiography was

also performed. As a result all cardiac parameters were increased in the 28 patients with positive findings. There also was right heart enlargement with bulge of the right heart border and of the pulmonary artery segment. The hilar densities were increased in size and there was a paucity of vascular densities, with increased radiolucency of the pulmonary fields. In 1 case only did the narrowing of vessels extend to include the hilar structures.

Recurrent episodes of embolic occlusion of the peripheral arteries could only be ascertained in 2 of the 28 patients. In all the other patients an endarteritis existed. The end-diastolic right ventricular pressure was also elevated in 24 patients.

Pulmonary hypertension is found to develop quite rapidly. There is, however, no direct correlation of the time factor and the quantitative cardiopulmonary changes. Nevertheless, the parameters permit a diagnostic conclusion of pulmonary hypertension from the roentgenograms alone. The condition, however, has to be differentiated from a left-to-right shunt and from congestion secondary to mitral valve lesions. Pulmonary emphysema may simulate pulmonary hypertension, but it presents with a much smaller heart as opposed to cardiomegaly encountered in this group.—*Ernest Kraft, M.D.*

MARANO, PASQUALE, ALQUATI, PALMIRO, and ROMANELLI, FRANCESCO. Aspetti radiologici del circolo polmonare in pazienti sottoposti a dialisi periodica. (Radiologic aspects of the pulmonary circulation in periodic dialysis patients.) *Radiol. med.*, Apr., 1971, *57*, 257–265. (Address: Policlinico A. Gemelli, Via Pineta Sacchetti 526, I-00168 Roma, Italy.)

The authors discuss the variation in the pulmonary vascular pattern in patients subjected periodically to dialysis, as demonstrated in chest roentgenograms taken with the patient in the standing or recumbent position.

It is noted that in the roentgen studies performed in the erect position there is a thinning out of the vascular bed in the upper segments of the lungs, apparently due to vascular spasm, while the pulmonary vasculature appears dilated in the lower fields. These findings are reversed in the roentgenograms of the chest taken with the patient supine.

It is felt that the vascular spasm is produced by a reflex vasospastic mechanism occurring only when a certain level of venous pressure is reached.

Once the pulmonary alterations secondary to interstitial edema have taken place, these variations in the appearance of the vascular bed are not affected by the position in which the chest roentgenograms are obtained.—*A. F. Govoni, M.D.*

BANKL, H. Die Transposition der Herzostien: Ein Versuch ihrer Erklärung. (Transposition of heart ostia: an attempted explanation.) *Wien. klin. Wchnschr.*, May, 1972, *84*, 324–330. (Address: Pathologisch-Anatomisches Institut der Universität Wien, Spitalgasse 4, A-1090 Wien, Austria.)

In transposition of the cardiac ostia the aorta usually originates in the right ventricle and the pulmonary artery in the left ventricle. There are, however, many variations and exceptions to the typical cases.

The anatomic criteria for a complete simple transposition of the great vessels are based on the following observations: (1) presence of an abnormal subaortic muscular conus; (2) absence of a direct fibrous continuity of the mitral and the aortic ostia; (3) presence of a pulmonic-mitral continuity in the simple typical transposition; (4) appearance of a combined subaortic and subpulmonic conus in atypical simple transposition; (5) shifted position of the arterial ostia in anteroposterior direction so that the aorta is located anteriorly on the right side and the pulmonary artery posteriorly on the left side; (6) formation of a subaortic conus with absence of subpulmonic musculature leads to a characteristic transposition with displacement of the outflow and of the arterial ostia; (7) formation of a combined subostial conus when both arteries arise from the right ventricle; (8) complete absence of a subostial conus when both arteries arise from the left ventricle.

On the basis of 93 complete transpositions and of 7 so-called corrected transpositions an explanation of the pathogenesis of the congenital heart lesion is attempted. A *typical completely crossed* transposition exists only when the ring of the aortic ostium has no direct continuity with the aortic sail of the mitral valve, but the pulmonic ostium becomes continuous instead. In the *atypical variety* a continuity is lacking.

In the *anatomically corrected* transposition, the aorta receives venous blood from the right atrium. The left ventricle in this instance can be either on the left or on the right side. In the *functionally corrected* transposition, the aorta originates from the right ventricle but gets arterial blood from the left atrium.

The attempted classification centers on the development of the muscular conus in 3 stages: (1) a regular loop-curving of the heart results in a normal location of the ventricles; (2) a subaortic conus is formed while the subpulmonic musculature is absent and the arterial ostia are displaced; (3) subsequent septation represents a secondary phenomenon. When both arteries originate from the left ventricle, a subostial conus remains absent. The lack of the conus musculature facilitates a direct connection of the pulmonic and aortic valves with the mitral valve.

It is postulated that primarily a myocardial damage exists and that the abnormal position of the ostia and septation represent the result and not the cause of the developmental abnormality.—*Ernest Kraft, M.D.*

Cha, Eung Man, and Khoury, George H. Persistent left superior vena cava: radiologic and clinical significance. *Radiology*, May, 1972, *103*, 375–381. (Address: E. M. Cha, M.D., Department of Radiology, Chicago Medical School at Mount Sinai Hospital, Chicago, Ill. 60608.)

Persistent left superior vena cava is a rare venous anomaly which has received little attention in the radiologic literature. Although the incidence of this vessel is approximately 0.3 per cent in patients with normal hearts, the incidence approaches 4.4 per cent in patients with congenital heart disease.

The authors present a review of their findings in 12 patients with persistent left superior vena cava, discovered in a series of 275 angiocardiograms.

Normally, the embryologic anterior and posterior cardinal veins, of each side, unite at each side of the sinus venosus forming the right and left ducts of Cuvier. The right duct becomes a portion of the right superior vena cava, whereas the left duct partially obliterates to become the coronary sinus. When this left portion persists as a left superior vena cava, it usually directs the left jugular and left subclavian venous drainage into the coronary sinus which empties into the right atrium. This root has no physiologic significance, but cyanosis results when the vein drains into the left atrium or the left pulmonary vein. The left superior vena cava usually persists in conjunction with a right superior vena cava (10 out of 12 patients), but maya exist alone (2 patients).

A presumptive diagnosis of persistent left superior vena cava can be based on: (a) the clinical finding of abnormal left jugular pulsation; or (b) radiographic evidence of an abnormal shadow on the plain chest roentgenogram or during fluoroscopy. This vessel may present as a widening of the aortic shadow or vascular pedicle, a paramediastinal bulge below the aortic arch, a strip of lessened density along the left upper cardiac border, or a paramediastinal crescent. Presence of a thymus in this area will obscure these signs. The paramediastinal crescent sign is a crescentic vascular shadow coursing from the left upper border of the aortic arch toward the middle third of the left clavicle. In some instances, it presents as a straight vertical strip. The plain film roentgenographic crescent or strip sign was positive in 7 patients and suggestive in another 4.

A persistent anomalous left vertical pulmonary vein should not be confused with a persistent left superior vena cava. This anomalous pulmonary vein receives part or all of the left pulmonary drainage and drains superiorly into the left innominate vein and has no cardiac connection. One such case is included, for comparison, with the plain roentgenograms demonstrating an oblique density extending upward and medially from the main pulmonary area to the aortic arch.

The authors conclude that the persistent left superior vena cava can be suspected clinically and roentgenographically and can be confirmed by cardiac catheterization and/or angiocardiography. In their series, catheterization through the left arm vein does not necessarily increase the detection of this anomaly and possesses the disadvantages of difficulty in maneuvering the catheter and a higher incidence of arrhythmia.—*Edward B. Best, M.D.*

Abdomen

Faust, H., Hartweg, H., and Eugenidis, N. Zur Differentialdiagnose neoplastischer Prozesse im Duodenum. (The differential diagnosis of neoplastic processes in the duodenum.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, April, 1972, *116*, 499–508. (Address: Oberarzt Dr. H. Faust, Universitätsinstitut für Röntgendiagnostik und Strahlentherapie, Bürgerspital Basel, CH 4000 Basel, Switzerland.)

The incidence of malignant tumors of the duodenum is 0.033 per cent based on 350 autopsies. Of all gastrointestinal malignancies, 0.3–1.7 per cent are found in the duodenum, whereas 17–45 per cent of all small bowel neoplasms are duodenal in origin. In order of frequency, the most common duodenal neoplasms are adenocarcinoma, leiomyosarcoma, lymphosarcoma, reticulum cell sarcoma and carcinoid (the latter are rare in the duodenum). Carcinomas of the duodenum are usually classified into suprapapillary, peripapillary and infrapapillary. The suprapapillary lesions may cause gastric outlet obstruction, but the vomitus will contain bile; peripapillary lesions produce constant or recurrent jaundice; infrapapillary tumors may be associated with acholic stools in the absence of jaundice and the vomitus usually contains old food particles. Primary carcinoma of the duodenal bulb is extremely rare. Metastases to the duodenum are common in melanoma and from breast, lung, chorioepithelioma and other genital neoplasms as well as by direct extension from involved retroperitoneal lymph nodes, pancreas, gallbladder, colon and kidney.

About 0.002 per cent of all benign tumors are found in the duodenum. They consist of adenomas and adenomatous polyps, leiomyomas, fibromas, neurinomas, lipomas, hemangiomas and mixed tumors. According to Bockus, approximately 7 per cent become malignant. Leiomyomas tend to ulcerate and bleed much more commonly than adenomas. Benign tumors of the bulb have to be differentiated from Brunner's adenomas; in the rest of the duodenum, neurofibromatosis and Peutz Jeghers disease have to be considered.

The roentgenologic findings in duodenal neoplasms consist of a decreased elasticity of the wall or rigidity, eccentric or circular narrowing of the lumen, loss

of a normal mucosal relief pattern, a constant filling defect with a broad-based attachment (unlike the slender usually longer stalk of a polyp) and almost always a single lesion. Leiomyomas greater than 5 cm. in diameter should be suspected of being neoplastic. Eccentric contour defects are more apt to be metastatic than primary lesions. Changes in the normal course of the duodenal sweep, when not an anomalous variant, are probably due to tumors of the pancreas, gallbladder and bile ducts, kidneys, colon or retroperitoneal lymph nodes.

Benign tumors present with sharp, round or polycyclic contours; they often have a thin stalk; the mucosa is intact; the wall is pliable. An aberrant pancreas often has the characteristic dimple. Fibrotic changes of inflammatory conditions, such as Crohn's disease or other granulomatous conditions, produce a narrowed lumen, some rigidity of the wall, some destruction of the mucosa, and ulceration.—*J. Zausner, M.D.*

GROCELLÀ, ALDO, BRUNO, SALVATORE, NANNELLI, UMBERTO, and VITA, GIUSEPPE. Colangiografia perfusionale potenziata con glucosio. (Glucose-potentiated perfusion cholangiography.) *Radiol. med.*, June, 1971, *57*, 417–428. (Address: Ospedale O. Basilewsky, Via Lorenzo il Magnifico 104, I-50129 Firenze, Italy.)

The value of potentiated intravenous cholangiography has been debated in the past few years by several authors searching for a procedure which could offer a satisfactory roentgen evaluation of the extrabiliary system and, at the same time, diminish the incidence of secondary reactions to the injected contrast medium.

The authors have examined 600 patients by potentiated intravenous cholangiography.

The technique used was as follows: 20 cc. of Biligrafin, an iodipamide, and more recently Bilivistan, an ioglycamide, were dissolved in 200 cc. of a glucose solution; the time of infusion was 60 minutes.

The results obtained were considered very satisfactory and the tolerance to the contrast medium was judged to be far superior to the one previously noted in the standard intravenous cholangiography.

Reproductions of cholangiograms of several patients illustrate the article.—*A. F. Govoni, M.D.*

SUZUKI, TAKASHI, IMAMURA, MASAYUKI, KAWABE, KATSUMI and HONJO, ICHIO. Selective demonstration of the variant hepatic artery. *Surg., Gynec. & Obst.*, Aug., 1972, *135*, 209–215. (From: Department of Surgery, Kyoto University Medical School, Kyoto, Japan.)

The hepatic artery which originates from the superior mesenteric artery is the most frequently encountered variation. The authors met this variant in 16 per cent of 200 patients. Since the variant hepatic artery usually arises at an acute angle, superselective catheterization is often difficult.

The authors report in detail a simple, practical technique for the selective demonstration of the variant hepatic artery by the combined use of a coaxial, double catheter system and vasoconstrictive agents.

Following placement of the larger, outer catheter in the orifice of the superior mesenteric artery, a smaller catheter is advanced through the larger catheter beyond the origin of the variant hepatic artery. Noradrenaline is then administered through this catheter resulting in constriction of the intestinal branches alone. Subsequent contrast medium injection through the outer catheter then allows unimpeded visualization of the hepatic arteries with enhanced visualization of the smaller branches.

This method may also be used to improve visualization of the arterial supply to the head of the pancreas by injecting the vasoconstrictor distal to the origin of the inferior pancreaticoduodenal artery. Also, in those patients with celiac stenosis where the hepatic arterial supply is derived from the pancreaticoduodenal artery, selective perfusion of the liver is improved following vasoconstriction of the intestinal branches.

This method is useful not only for diagnosis, but also for single shot chemoperfusion in treatment of carcinoma of the liver. The anticancer drug can be delivered to the liver alone.

The double catheter technique was successfully employed in 15 of 16 patients with this anomaly found on diagnostic angiography. Several patients also underwent chemoperfusion following the arteriography.

Diagrams illustrate the method and roentgenographic reproductions demonstrate the value of the technique in a variety of conditions.—*Edmund E. Lewis, M.D.*

MARGOLIES, MICHAEL N., RING, ERNEST J., WALTMAN, ARTHUR C., KERR, WALTER S., JR., and BAUM, STANLEY. Arteriography in the management of hemorrhage from pelvic fractures. *New England J. Med.*, Aug., 1972, *287*, 317–321. (Address: M. N. Margolies, M.D., 275 Charles Street, Boston, Mass. 02114.)

Massive extraperitoneal hemorrhage remains the leading cause of death after pelvic fracture. Localization of the source of hemorrhage is not often made clinically and surgical exploration is exceedingly difficult. Transfemoral venography occasionally identifies lacerations of the common iliac veins, but arterial bleeding from pelvic fractures may be more common than has previously been supposed.

Three patients with pelvic fractures and massive, rapid retroperitoneal blood loss were examined by transfemoral aortography. In all of them bleeding

originated from the obturator artery branches adjacent to the pubic rami fractures. Posterior pituitrin infusion slowed, but did not arrest the hemorrhage. In 2 patients, hemorrhage was controlled by selective catheterization of the hypogastric artery, followed by injection of autologous clotted blood into the anterior division. In a third patient selective catheterization was not possible.

Control of hemorrhage by selective catheterization of a branch of the hypogastric artery is attractive because it may avoid exploration of the hematoma with its attendant risks. The experience reported here emphasizes the need for early diagnostic aortography when there is excessive retroperitoneal blood loss in patients with pelvic fractures.

When the described method of hemostasis fails, arteriographic localization of the bleeding site should improve selection of patients for surgical exploration or hypogastric artery ligation.—*Edmund E. Lewis, M.D.*

GENITOURINARY SYSTEM

ALFIDI, RALPH J., TARAR, RIAZ, FOSMOE, ROBERT J., FERRARIA, C., BOLTUCH, ROBERT L., and GIFFORD, RAY W., JR. Renal-splanchnic steal and hypertension. *Radiology*, March, 1972, *102*, 545–549. (From: Department of Hospital Radiology, The Cleveland Clinic Foundation, Cleveland, Ohio.)

Several hemodynamic detours resulting in steal mechanisms are well known. The authors propose the inclusion of renal-splanchnic steal in the list of steal mechanisms.

Three cases of occlusion of the celiac axis with collateral steal vessels emanating from the right inferior adrenal artery and/or phrenic artery in 3 patients with hypertension are reported. One case has been previously reported by these authors in 1967. These vessels anastomosed with various adrenal collaterals that eventually drained into part of the distribution of the celiac axis.

A number of similarities are noted in each of the cases. All patients were women, all showed large collateral vessels emanating from the right kidney and none from the left. All collaterals emptied into a part of the entire distribution of an occluded or severely stenosed celiac axis, and 3 of the 4 had at one time or another taken progestational agents.

The authors postulate that the steal mechanism is the cause of hypertension. This is based on the facts that experimentally and clinically, a renal arteriovenous fistula is capable of producing hypertension, and, in theory, renal-celiac steal is analogous to an arteriovenous fistula of the kidney. In both, there is diversion of renal arterial blood into a low pressure system such as the renal vein or branches of the celiac axis with an orificial occlusion or stenosis.

It is suggested by the authors that surgical attempts to correct this problem should be directed toward re-establishment of a normal splanchnic blood supply, rather than ligation of collateral vessels. This would establish normal hemodynamics and eliminate possible future demands for the formation of collateral vessels.—*F. R. Jenkins, Jr., M.D.*

DANIEL, WILLIAM W., JR., HARTMAN, GLEN W., WITTEN, DAVID M., FARROW, GEORGE M., and KELALIS, PANAYOTIS P. Calcified renal masses: a review of ten years' experience at the Mayo Clinic. *Radiology*, June, 1972, *103*, 503–508. (From: Mayo Clinic and Mayo Foundation, Rochester, Minn.)

The article is based upon the review of 2,709 renal masses seen during a 10 year period at the Mayo Clinic. Of these masses, there was calcification in 111 or 4.1 per cent. The incidence of calcification of 560 renal cell carcinomas was 10.3 per cent. This percentage is somewhat less than usually reported in the literature with figures ranging from 14 to 22 per cent. Benign simple cyst was the most commonly encountered mass (1,682) and the calcification incidence was approximately 1 to 2 per cent, if half of the indeterminate masses were considered simple cysts.

The calcifications were divided into 3 types: (a) peripheral; (b) nonperipheral; and (c) combinations. The authors attempted to determine: (1) whether calcification was an indicator of a benign or a malignant lesion; and (2) was the pattern of calcification helpful as regards the determination of benignancy *vs.* malignancy?

The conclusions reached are: (1) a mottled or punctate nonperipheral calcification was highly indicative of a malignant lesion; (2) the location of the calcification is more meaningful than the pattern of calcification in differentiation of cyst and tumor; and (3) purely peripheral "egg-shell" type of calcification has a greater chance of being a benign cyst, although the risk of malignancy is 20 per cent.

It is interesting to note that in an avascular calcified mass, the authors question the value of angiography.—*J. P. Eberts, M.D.*

ROBINS, JON M., and BOOKSTEIN, JOSEPH J. Regressing aneurysms in periarteritis nodosa: a report of 3 cases. *Radiology*, July, 1972, *104*, 39–42. (From: Department of Radiology, University of Michigan Medical Center, Ann Arbor, Mich.)

Multiple aneurysms of small renal arteries have been considered a highly reliable angiographic sign of periarteritis nodosa. The fact that these aneurysms may regress has not been generally appreciated. The authors have recently studied 3 patients with an angiographic diagnosis of periarteritis nodosa, in whom a second examination demonstrated disappearance of the aneurysms.

The incidence of renal aneurysms in autopsy specimens is low, occurring in 8.5 per cent of patients with a proved histologic diagnosis of periarteritis nodosa. The incidence of angiographically demonstrable aneurysms is unknown, as no large series of cases has been studied to date. In the authors' study, however, renal aneurysms have been found in 3 of the 4 cases with histologically confirmed periarteritis nodosa and 9 of 11 cases with a diagnosis of necrotizing angiitis.

The failure to demonstrate aneurysms in a previously involved organ may represent "healing" on a histologic basis, but should not be construed as evidence of disease quiescence or remission. Healed aneurysms have been seen with clinically quiescent histologically active disease, and histologically inactive disease with progressive clinical complications. The presence of intraparenchymal renal aneurysm shown by angiography is not specific for periarteritis nodosa. There are 5 basic classifications within the major classification of necrotizing angiitis. These are: hypersensitivity angiitis; allergic granulomatous angiitis; rheumatic arteritis; periarteritis nodosa; and temporal arteritis.

The authors feel that probably the angiographic demonstration of multiple aneurysms of small intraparenchymal arteries is not specific for periarteritis nodosa, but may be seen in a spectrum of necrotizing angiitides.—*E. J. Schoolar, M.D.*

SKELETAL SYSTEM

PROST, A., RAMBAUD, J. C., MIRAVET, L., BORDIER, P., HIOCO, D., PAOLAGGI, J., CAMUS, J. P., LIEVRE, J. A., DE SÈZE, S., and BERNIER, J. J. Les ostéomalacies révélatrices de la maladie coeliaque de l'adulte: à propos de sept observations. (Celiac disease of the adult revealed by osteomalacia; a report of 7 cases.) *Presse méd.*, May, 1972, *1*, 1329–1336. (From: Service de Médecine générale V et Rhumatologie, Hôpital Saint-Jacques, C.H.U., 85, rue Saint Jacques, F 44, Nantes, France.)

Out of a total of 20 cases of adult celiac disease (ACD) diagnosed by the authors, 7 patients presented an osteomalacic picture which led to the discovery of ACD. This gives an incidence of 35 per cent. However, the frequency of ACD among the causes of osteomalacia appears to be underestimated, at least in France according to the authors, because of an incomplete intestinal investigation.

Digestive disorders are often minimal in these cases; steatorrhea is mild or absent. Moreover, the clinical and radiologic improvement of the osteomalacic disease that follows administration of vitamin D could explain why the enteropathy might be overlooked.

For this reason, if there is no obvious lack of vitamin D in the diet, any case of osteomalacia should be thoroughly investigated for ACD, including a peroral biopsy.

In 6 of the reported cases anemia was present. In every case malabsorption of glucose was demonstrated, and malabsorption of D-xylose in 2 cases, where the test was performed.

The positive mucosal biopsy and the efficiency of a gluten-free diet are the basic criteria in the diagnosis of ACD.

The authors discuss the malabsorption of vitamin D and the problem of its vitamin-resistance in osteomalacia seconcary to ACD.

In concluding they also consider the parathyroid reaction to the lowered blood-level of calcium and magnesium.—*H. P Lévesque, M.D.*

RING, STUART M., BERANBAUM, ELLIOTT R., MADAYAG, MANUEL A., and NICOLOSI, CHARLES R. Angiography of aneurysmal bone cyst. *Bull. Hosp. Joint Dis.*, Apr., 1972, *33*, 1–7. (From: Department of Radiology, New York University Medical Center, New York, New York.)

A 15 year old male with pain and swelling of his left leg for 2 months was examined. Conventional roentgenograms showed a primary tumor of the shaft of the fibula, expansile, septated, and eccentric with a break in the thinned cortex near its lower end.

Angiography was carried out. The peroneal and anterior tibial arteries were displaced about the mass which had intense vascularity with staining. Arteriovenous shunting occurred rapidly, in 5 seconds, and there was intense vascular staining in the soft tissue extension of the mass through the region of cortical rupture, below. The central mass of the tumor presented faint spherical lucencies.

Because of the intense vascularity, early and rapid arterial venous shunting, and soft tissue extension of the lesion—features usually associated with sarcomas—local resection of the fibula was carried out following frozen section. Final histologic sections showed the typical appearance of aneurysmal bone cyst without evidence of malignancy, and no recurrence developed in the following year.

The present case shows considerable variation in the appearance of the ateriogram from that reported in other arteriographic examinations in aneurysmal bone cyst.—*E. Frederick Lang, M.D.*

CHAPLIN, D. M., and HARRISON, M. H. M. Pseudomalignant osseous tumour of soft tissue. *J. Bone & Joint Surg.*, May, 1972, *54 B*, 334–340. (From: Orthopaedic Department, The General Hospital, Birmingham, England.)

Nonmalignant tumors of soft tissue which exhibit histologic features resembling sarcoma in their cen-

tral parts, but contain mature bone at the periphery, have been called pseudomalignant osseous tumors of soft tissue. The authors' 2 cases constitute the twentieth and twenty first reported cases. Most of the tumors were in the extremities or gluteal region, and the size was remarkably constant, rarely exceeding 6 cm. in any diameter. Most of the cases were in women, usually young adults.

All of the reported lesions were noted to be well circumscribed from the surrounding tissues with no associated large blood vessels. Mature trabecular bone forms at the periphery of the tumor with spicules of osteoid tissue radiating toward the center, and ossification occurs only after an interval of at least 2 weeks. It may not be seen early roentgenographically. The central connective tissue mass is of varying density with fibroblasts generally arranged in bundles, with cellular pleomorphism, giant cells, and occasionally mitotic figures. The limitation in size may be associated with its poor blood supply.

Preceding trauma was encountered in only a few of the reported patients. Most of the tumors occurred in muscle and most were well circumscribed. In several the clinical features suggested preexisting infection.

All could be differentiated from myositis ossificans because of the good demarcation and lack of muscle fibers in the lesion. There was no associated hematoma. None of the 21 cases showed any indication of atypical cells and there was no histologic indication of neoplasm.

In one of the authors' cases, which was subjected to biopsy, the lesion was not removed and serial roentgenographic studies showed regression of the lesion which became densely ossified and consolidated with the adjacent femur.—*E. Frederick Lang, M.D.*

FIUMICELLI, ALIGI. Contributo allo studio delle acroosteolisi neurogene essenziali. (A contribution to the study of essential neurogenic acro-osteolysis.) *Radiol. med.*, Feb., 1971, *57*, 131–147. (Address: Istituto di Radiologia dell'Università, Policlinico, I-06100 Perugia, Italy.)

In the large chapter of the acropathies, in the past decades the rather homogeneous group of the neurogenic essential acro-osteolysis has attracted the interest of several authors.

The disease has a long course, protracted for years, and is characterized by the absence of any related inflammatory process.

The skeletal manifestations noted in the bones of the feet are typical: early epiphyseal fragmentation; centripetal reabsorption of the diaphysis; and involvement of the adjacent articulation. The process of acro-osteolysis may extend up to the lower third of the tibia and fibula. Angiographic studies show no gross alterations of the vascular bed.

The etiology and pathogenesis of the disease are still unknown, although it is felt that a primary neurogenic factor might produce a prolonged arteriolar constriction resulting in a damage to the most peripheral arterioles and then the appearance of the skeletal process of osteolysis.—*A. F. Govoni, M.D.*

HUVOS, A. G., HIGINBOTHAM, N. L., and MILLER, T. R. Bone sarcomas arising in fibrous dysplasia. *J. Bone & Joint Surg.*, July, 1972, *54-A*, 1047–1056. (From: Department of Pathology and the Bone Service, Department of Surgery, Memorial Hospital for Cancer and Allied Diseases, New York, N. Y.)

Fibrous dysplasia is one of the more common lesions of the skeleton and affects the ribs, femur, tibia, maxilla and the calvarium in descending order. Theories as to the origin of fibrous dysplasia include congenital anomalous malfunction of bone forming mesenchyme and arrest of bone maturation at the woven bone stage.

Twelve cases of fibrous dysplasia associated with primary bone sarcoma are reported. There is no sex predominance and the age range is 50 to 54 years. Six of the patients had monostotic involvement and 6 polyostotic involvement.

Radiation therapy was used in the treatment of fibrous dysplasia in only 1 of these patients. The histologic types of sarcomas included 8 osteosarcomas, 2 chondrosarcomas, and 2 spindle-cell sarcomas. A 5 year actuarial survival rate for the 12 patients from the time of diagnosis of the sarcoma was 52 per cent.

It is concluded that primary bone sarcomas occur more frequently in patients whose skeletal system is affected by other benign conditions such as fibrous dysplasia.—*Ronald J. Ross, M.D.*

LUKERT, BARBARA P., BOLINGER, ROBERT E., and MIIK, JOSEPH C. The effect of fluoride on ^{45}Ca kinetics in Paget's disease. *J. Clin. Endocrinol. & Metabol.*, Sept., 1972, *35*, 387–391. (From: University of Kansas, Kansas City, Kansas.)

Paget's disease of bone is characterized by increased rates of bone turnover, with increased bone resorption initially, and a compensatory increase in osteoblastic activity.

This report concerns 6 patients with Paget's disease who were studied on a metabolic ward, and after a 3 week control period with evaluation of bone metabolism, treatment with sodium fluoride was begun. Radioisotope studies were made during the control period, and 6 months to 2 years later bone biopsies were obtained after treatment.

After the administration of fluoride there was marked decrease in all parameters of bone metabo-

lism. Calcium balance improved in all but 1 of the patients treated. There were no significant changes in blood urea nitrogen, blood cell counts, serum calcium or phosphorus. There was a rise in serum alkaline phosphatase levels in all of the patients.

It seems likely that fluoride produces a state of osteomalacia in which the osteoid material fails to mineralize in a normal fashion. It is certain that the patients experience rather dramatic relief from pain. No roentgenographic changes were observed and it is not possible to make any decisions as to the beneficial effects of fluoride on Paget's disease.—*E. Frederick Lang, M.D.*

BLOOD AND LYMPH SYSTEM

WOLF, G., DIMOPOULOS, J., CANIGIANI, G., and CZECH, W. Lymphographiekontrolle und Re-Lymphographie, zwei Untersuchungsarten, die zur Erweiterung der Lymphknotendiagnostik beitragen. (Lymphography control and re-lymphography; two procedures contributing to the lymph node diagnostics.) *Wien. klin. Wchnschr.*, Apr., 1972, *84*, 269–272. (Address: Dr. G. Wolf, Röntgendiagnostisches Institut der Universität Wien, Alserstrasse 4, A-1090, Wien, Austria.)

This study depends on the difference in response between normal and abnormal lymph nodes to the injection of contrast material or radioactive therapy. On the initial lymphogram, selected lymph nodes are classified as normal or abnormal, and are carefully identified so that they may be compared with follow-up roentgenograms at monthly intervals or re-lymphography after the original contrast material is no longer visible. Metastatic lymph nodes tend to be round rather than oval because of the uniform expansive growth of tumors.

The purpose of this investigation is threefold: (1) to check on unclear or questionable findings; (2) to search for new metastases; and (3) to document the efficacy of therapeutic measures. Intralymphatic injection of contrast material produces enlargement of the lymph nodes not only by the mechanical presence of the substance, but also by the chemically induced inflammation. These changes are reversible so that in 10 days, a normal lymph node characteristically returns to its original size. Pathologic lymph nodes will not shrink as rapidly as normal lymph nodes or they may grow after first showing some tendency to decrease in size. In other words, pathologic lymph nodes will show a different curve on monthly check-ups. Occasionally a "normal" lymph node may show evidence of metastasis during the survey.

One cannot predict when the contrast material will disappear from the lymph nodes, since it has been observed as long as 2 years after an injection. This method is useful only as long as the contrast material is identifiable. After that, re-lymphography can be performed and a response of the lymph nodes similar to the one following the prior injection may be anticipated, provided that these lymph nodes have not been otherwise affected.—*J. Zausner, M.D.*

RADIATION THERAPY

GOUIN, B., CCUTURIER, D., HARDOUIN, J.-P., and DEBRAY, C. Les médiastinites néoplasiques: complications tardives des cancer du sein; à propos de trois observations. (Metastatic mediastinitis: late complication of mammary cancer; report of 3 cases.) *Presse méd.*, June, 1972, *1*, 1563–1568. (Address: C. Debray, Hôpital Bichat, 170, Bd. Ney, F 75, Paris, France.)

Among the various metastatic localizations of carcinoma of the breast, mediastinal involvement manifested by a solitary dysphagia at first appears unusual. The authors report 3 cases and review 42 detailed cases published in the world literature, which provides that this complication is not exceptional.

The authors' first case was operated on for a mammary cancer in 1963, after radiotherapy. The patient remained asymptomatic during the following 4 years. In mid 1967 she experienced progressive dysphagia with loss of weight. Subsequently, she developed a left basal pleurisy which cleared spontaneously after 3 weeks. An esophagogram revealed a tapering stenosis of the lower third of the esophagus, filiform in its distal centimeters, although the mucosal folds seemed preserved. The stomach was normal, without hiatal hernia or esophageal reflux. Endoscopy confirmed the stenosis and revealed a red mucosa. The biopsy specimen was reported as a simple hyperplasia. Cytology brush technique led to negative results. At laparoscopy a peritoneal carcinomatosis was observed. Esophageal dilatation did not bring any improvement.

The second patient was treated early in 1966 for a carcinoma of the right breast by radiotherapy only. At the end of 1969 she noticed a progressive dysphagia accompained by loss of weight. Pleural fluid was present at the right lung base. The esophagogram evidenced an 8 cm. long narrowing of the mid esophagus. The esophagoscope could not be passed through the stenotic segment; proximally, the mucosa was found to be normal. Pleural fluid analysis did not reveal cancer, but the pleural biopsy was positive. Esophageal dilatation was of no benefit.

The third patient was surgically treated in 1961 for a right mammary cancer. Late in 1965 she experienced a rapidly intensifying dysphagia. The esophagogram revealed a tight stenosis localized to the middle third and extending over 2 cm. length. The narrowing appeared regular, eccentric and relatively pliable. At esophagoscopy, an edematous and non-tumoral mucosa was seen. The esophageal biopsy

was negative for carcinoma; the cytology brush also gave negative results. At surgery a nonresectable paraesophageal metastasis was diagnosed and the patient was submitted to cobalt 60 teletherapy, coupled with high dosage of estrogens, with subsequent improvement.

Analysis of 42 similar cases collected from the medical literature, reveals that esophageal involvement occurred after a long symptom-free interval (an average of 7 years) following treatment of the causative mammary carcinoma. The metastatic localization to the esophagus was manifested by dysphagia, and caused a stenosis which in most cases involved the middle third and measured an average of 6 cm. in length. Roentgenologically, the narrowing was found to be mostly axial, rarely eccentric, with regular contours; and the mucosal folds were preserved in 7 cases. At endoscopy the mucosa appeared normal in 7 cases, congested in 4, and in 12 cases an impression of extrinsic compression was noted. In only 2 cases did the mucosa appear definitely neoplastic. These findings explain the difficulties encountered in establishing the diagnosis and the fact that endoscopic biopsy shows negative results.

Some roentgenologic signs are helpful in distinguishing this lesion from primary carcinoma and peptic esophagitis, but the extension of the disease to a distinct organ such as the pleura usually leads to the correct preoperative diagnosis.

As to treatment, instrumental dilatation should be avoided. Radiotherapy associated with chemotherapy seems the logical solution for the improvement of the patient's comfort, but the length of survival has not yet been determined.—*H. P. Lévesque, M.D.*

CHERNOMORDIKOVA, M. F., DEEMARSKY, L. Y., KHOLDIN, S. A., and SELEZNEV, I. K. Limits and possibilities of transsternal phlebography for detection of metastases in the internal mammary lymph nodes. *Radiology*, Feb., 1972, *102*, 291–297. (From: N. N. Petrov Research Institute of Oncology, Leningrad, U.S.S.R.)

Preoperative determination of metastases to the internal mammary lymph nodes in patients with cancer of the breast would be most helpful in establishing the best course of therapy and prognosis. Routine roentgenography, tomography with or without pneumomediastinography, and isotopic localization studies have all resulted in only limited success. Transsternal phlebography, a simple technique that consists of injecting a contrast agent into the sternum and roentgenographing the outlined internal mammary veins, was evaluated because the authors were fortunate to have sugical confirmation. One hundred and sixty-five cases of mammary cancer had extended axillary sternal mastectomy. In each case the result of transsternal phlebography was compared with the operative specimen of the internal mammary lymph nodes. Therefore, excellent correlation was afforded.

The roentgen diagnosis of lesions of the internal mammary collector system is based on the assumption that enlargement of its lymph nodes (metastasis) will cause deformity of adjacent venous trunks. The main roentgen signs of enlargement of the internal mammary lymph nodes are: (1) absence of contrast medium in the internal mammary vein or amputation of the vein (obstruction) at the level of the first, second or third intercostal space; (2) local narrowing of the vein trunk or impression on the vein wall; and (3) deviation of the venous trunk without narrowing of its lumen.

The anatomic variations of the internal mammary veins and the size, number and location of the lymph nodes relative to transsternal phlebographic interpretation are discussed.

The results were encouraging in that the roentgen diagnosis in 151 of 165 cases was correct (91.5 per cent). In only 4 cases (2.4 per cent) was the diagnosis a false negative and in only 10 cases (6.1 per cent) was there a false positive finding. The incorrect interpretation occurred in the presence of lymph node hyperplasia.

It is stressed that the size of a lymph node is not as important as its location (i.e., phlebograms show no abnormality in the presence of an enlarged lymph node in the third, fourth or fifth intercostal space because these lymph nodes are situated laterally to the vessels). In addition, enlargement of a lymph node should be considered as evidence of neoplastic spread even in the case of hyperplasia, since it is the opinion of some that hyperplasia is the result of tumor invasion which is not revealed by morphologic examination.—*Paul M. Kroening, M.D.*

FASSIN, Y., and JEANMART, L. La ponction des kystes du sein: apport de la pneumomastographie. (Puncture of the breast cysts: the contribution of pneumomastography.) *J. belge de radiol.*, Jan.-Feb., 1972, *55*, 39–52. (Address: L. Jeanmart, avenue Winston Churchill, 236, 1180 Bruxelles, Belgium.)

Gaseous cystography of the breast is a simple examination without adverse aftereffects. The image of the lesion which it provides is precise and easy to interpret. This technique furnishes the best means of establishing the differential diagnosis between benign and malignant cystic lesions.

The cyst is punctured by the fine needle of a syringe, and the liquid is withdrawn as completely as possible, and replaced by an equal volume of air, less 10 per cent. This examination, combined with the cytologic analysis of the aspirated fluid, increases the accuracy of the diagnosis and in most cases obviates a surgical exploration.

Between 1965 and 1970 the authors performed pneumomastography on 112 patients. Surgical excision was done in 25 of these, based on cytologic or radiologic indications.

An intracystic proliferation was diagnosed by this roentgenographic method in 5 cases, which represents a frequency of 5.4 per cent as compared with a frequency of 2 per cent reported by Foote and Stewart in 1945. The papillous proliferation proved malignant in 2 of these 6 cases.

Statistically, 60 per cent of the authors' patients did not breast-feed, and in 58 per cent of the cases the tumor was detected by the patient herself and was painless; 8 per cent of the cases were asymptomatic.—*H. P. Lévesque, M.D.*

MACKIE, RONA M., CARMICHAEL, DAVID C., and COCHRAN, ALISTAIR J. Assessment of prognosis in patients with malignant melanoma. *Lancet*, Sept., 1972, *2*, 455–456. (From: University Department of Dermatology and Pathology, Western Infirmary, Glasgow, Scotland.)

Attempts have been made to develop a rational basis for predicting survival in patients with malignant melanoma when first seen. The authors have modified their original prognostic score after observing its use.

Eighty-five patients were available in this prospective study with a 5 year follow-up. Thirty-six were alive and free of melanoma, 36 had died of the disease, 9 had died of intercurrent disease, and 4 were alive with recurrent malignant melanoma.

Some of the features which were found to be of high value in predicting a poor outcome, in addition to lymph node involvement or disseminated disease, were occurrence in a male, a nonexposed site of the lesion, an occult or mucosal lesion, and occurrence on the trunk or in the anogenital region.

Use of this score sheet method permitted differentiation of the 36 tumor free survivors and the 36 who died of melanoma with an accuracy of greater than 80 per cent, at the time they were first observed. Nineteen per cent of the 85 patients had scores which fell into the middle range, and their outcome could not be reliably predicted by this technique.

Successful use of this or a modified schedule was carried out in Switzerland, France and Scotland. Attempts to use this scoring technique in Queensland, Australia, were unsuccessful and this suggests that melanoma in Australia behaves differently from the same tumor in other climates.—*E. Frederick Lang, M.D.*

RADIOISOTOPES

VAN DYKE, DONALD, ANGER, HAL O., SULLIVAN, ROBERT W., VETTER, WILLIAM R., YANO, YUKIO, and PARKER, HOWARD G. Cardiac evaluation from radioisotope dynamics. *J. Nuclear Med.*, Aug., 1972, *13*, 585–592. (Address: Donald Van Dyke, Donner Laboratory, University of California, Berkeley, Calif. 94720.)

An improved method for evaluation of left ventricular ejection fraction using radioisotopic angiocardiography is discussed.

The major components of the system are a scintillation camera, a quantitative television data system for rapid data recording and read out, and a bolus injection of Tc^{99m} albumin through the central circulation.

The improvement of the method consists in the subtraction of overlapping scintillations from the right ventricle, aorta, etc., when the area of interest is the left ventricle. When the ejection fractions from contrast angiography are compared to the isotopic data, the correlation is very good.

Other usable data obtained from this method include ventricular end diastole volume, and rates of change of ventricular volume.

The examination is noninvasive and can be repeated at frequent intervals.

The disadvantages include lack of anatomic detail as compared to contrast angiography and lack of hemodynamic pressure data as in a full heart catheterization.

Extensive clinical evaluation is necessary before including the method as a routine clinical procedure. —*Donald P. Harrington, M.D.*

MEINDOK, HARRY. Visualization of arterial and arterial graft patency by intravenous radionuclide angiography. *Canad. M. A. J.*, June, 1972, *106*, 1180–1182. (Address: 25 Leonard Avenue, Suite-410, Toronto 2B, Ontario, Canada.)

Fifty-four radionuclide arteriographies were performed on 35 patients.

Tc^{99m} pertechnetate was used in conjunction with a Nuclear Chicago Pho/Gamma H.P. Camera.

Example cases of normal anatomy in patients having brain scans are shown.

In pathologic cases, comparison of radionuclide and contrast angiography is demonstrated.

Occlusions are defined by the persistence of activity in the large vessels after the bolus phase. Correlation with angiograms to define patency and level of blockage was good, but anatomic detail was lacking; the field size is also quite small.

The study is recommended to determine arterial and/or arterial graft patency in patients who are immediately postoperative or are severely debilitated or uncooperative.—*Donald P. Harrington, M.D.*

GERSON, BARRY D., DORFMAN, HOWARD D., NORMAN, ALEX, and MANKIN, HENRY J.

Patterns of localization of ^{85}strontium in osteosarcoma: a correlative study using gross autoradiography. *J. Bone & Joint Surg.*, June, 1972, *54-A*, 817–827. (Address: B. D. Gerson, M.D., 1919 Madison Avenue, New York, N. Y. 10035).

The clinical aspects of 4 cases are reviewed.

The methods of autoradiography are given in detail. Histologic areas of (1) marked cellularity of tumor; (2) primitive tumor bone and tumor osteoid formation; and (3) endochrondal ossification and calcification of neoplastic cartilage are correlated with areas of high isotope uptake.

Areas of decreased to absent uptake were noted in histologic sections of: (1) nonviable tumor tissue; (2) excessively anaplastic or totally undifferentiated tumor; and (3) more mature tumor bone.

These findings supplement the theories of Charkes and associates of isotope uptake as a reactive process.

Variability within the tumor and timing of the isotope study are responsible for the tumor, and timing of the isotope study is responsible for the multiple responses noted.—*Donald P. Harrington, M.D.*

MISCELLANEOUS

Dhar, Phani, Moore, Terrence, Zamcheck, Norman, and Kupchik, Herbert Z. Carcinoembryonic antigen (CEA) in colonic cancer: use in preoperative and postoperative diagnosis and prognosis. *J.A.M.A.*, July, 1972, *221*, 31–35. (Address: Norman Zamcheck, M.D., 818 Harrison Avenue, Boston, Mass. 02118.)

This study of colonic cancer patients was based on the fact that there is a detectable circulating carcinoembryonic antigen (CEA) in patients with colonic carcinoma.

The study was intended to determine: (1) the usefulness of the serum CEA assay in the diagnosis of colonic carcinoma; (2) the prognostic value of preoperative and postoperative CEA tests in patients undergoing resections; and (3) the usefulness of interval CEA determinations in follow-up studies.

A negative serum CEA assay in patients with colonic carcinoma indicates localized, probably curative, tumor, while a strongly positive serum assay may indicate the opposite prognosis. Postoperatively, a positive CEA test indicates residual tumor, but a negative test does not rule out the presence of residual tumor. Interval CEA determinations may detect recurrence that is early enough to be treatable.

Before this test is widely used, further clinical study and standardization of the assay technique are necessary. More data concerning specificity of CEA seem necessary.—*Richard E. Kinzer, M.D*

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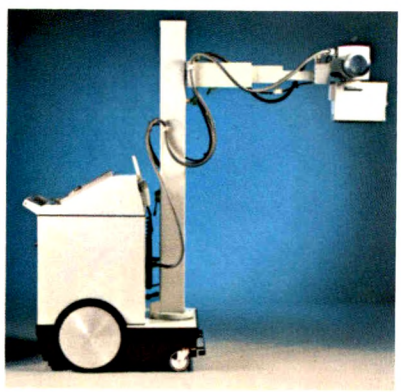
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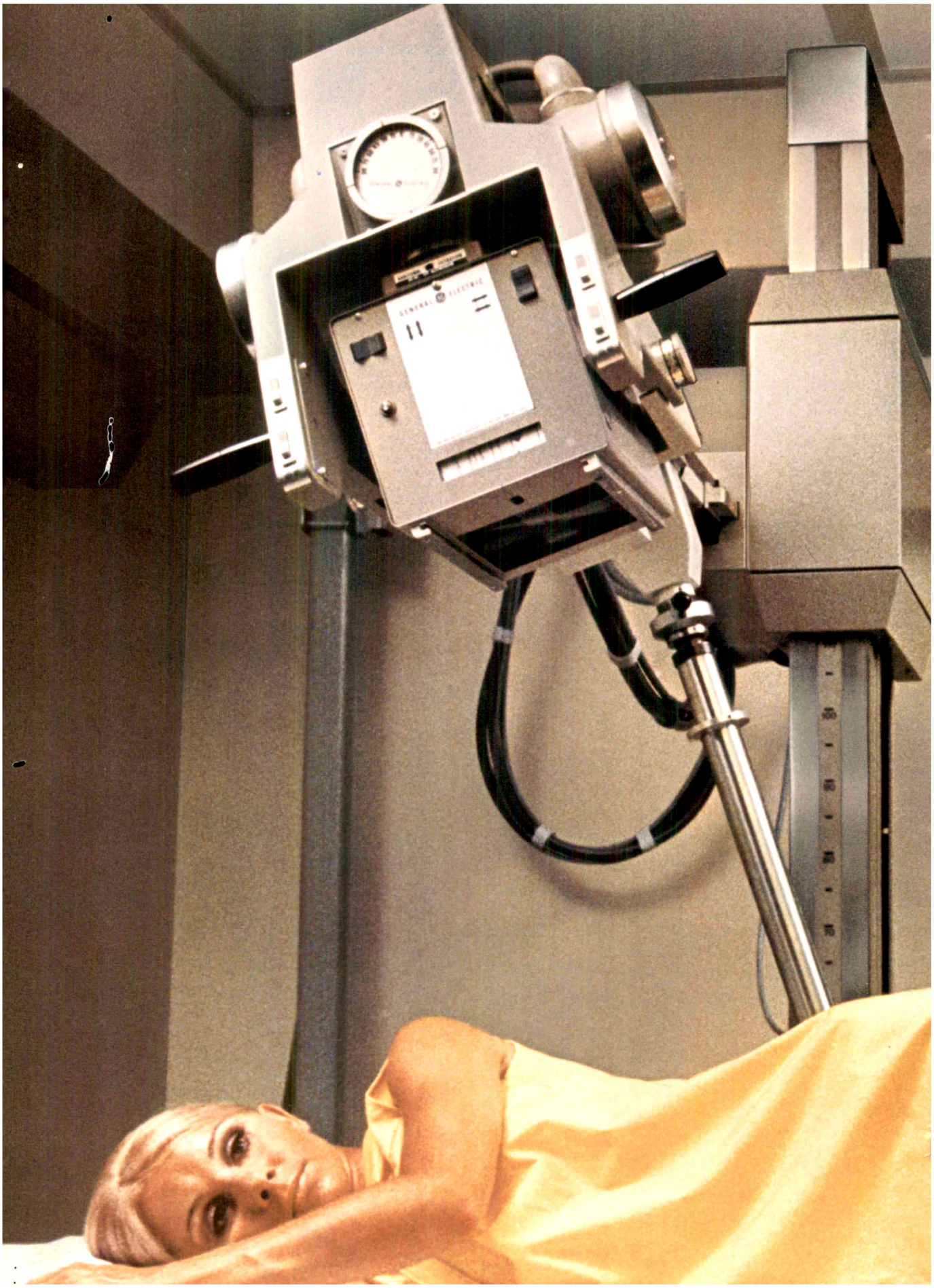
Move it to bedside. To emergency. Surgery. Cast rooms. Orthopedics. ICU/CCU wards. For all procedures, the unit's crisp video presentation makes available instant information for on-the-spot decisions.

Mobile Fluoricon smoothly tracks straight ahead or to either side. The C-arm rotates up to 180° side to side, 90° front to back, to pinpoint the desired viewing area. Powered 18 inch vertical travel, and reversal of tube unit and intensifier positions, add positioning versatility.



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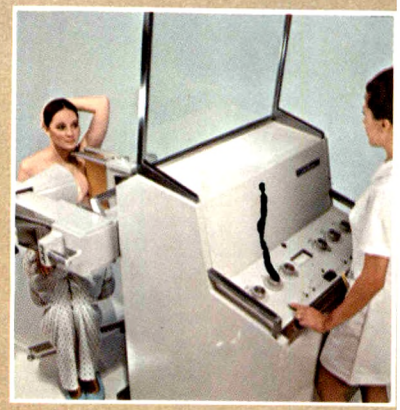
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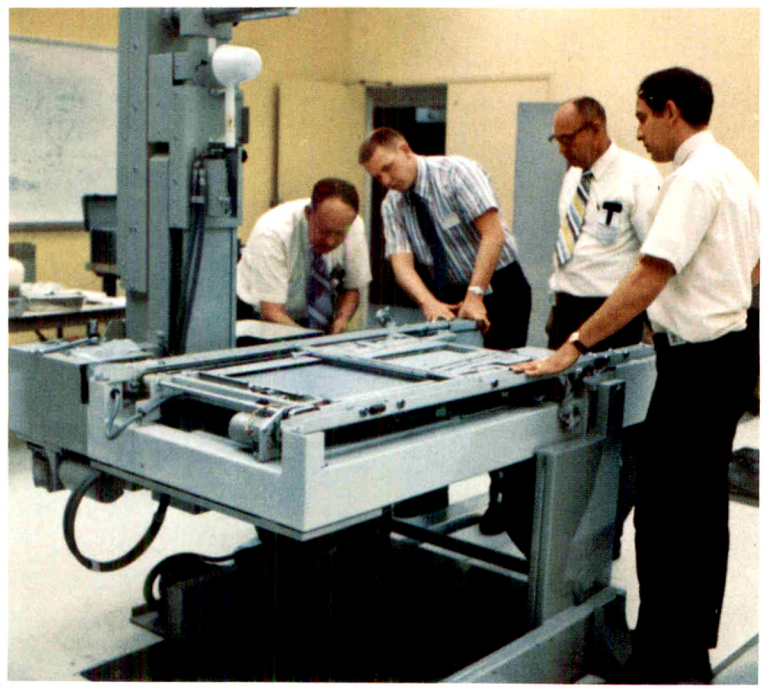
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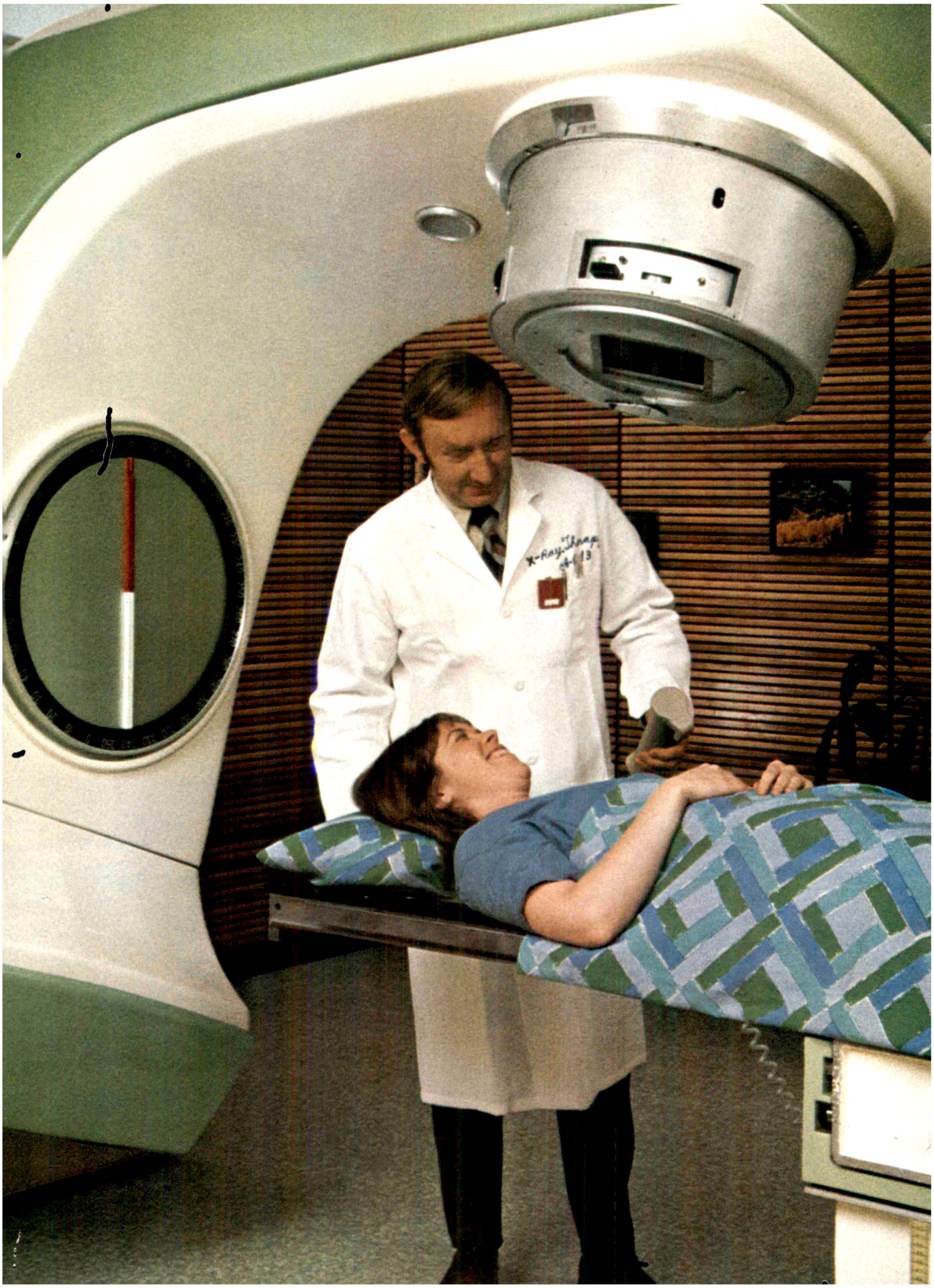
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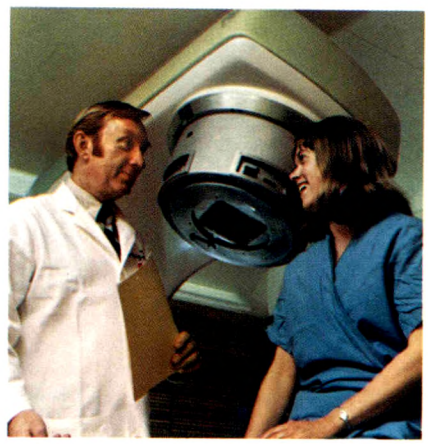
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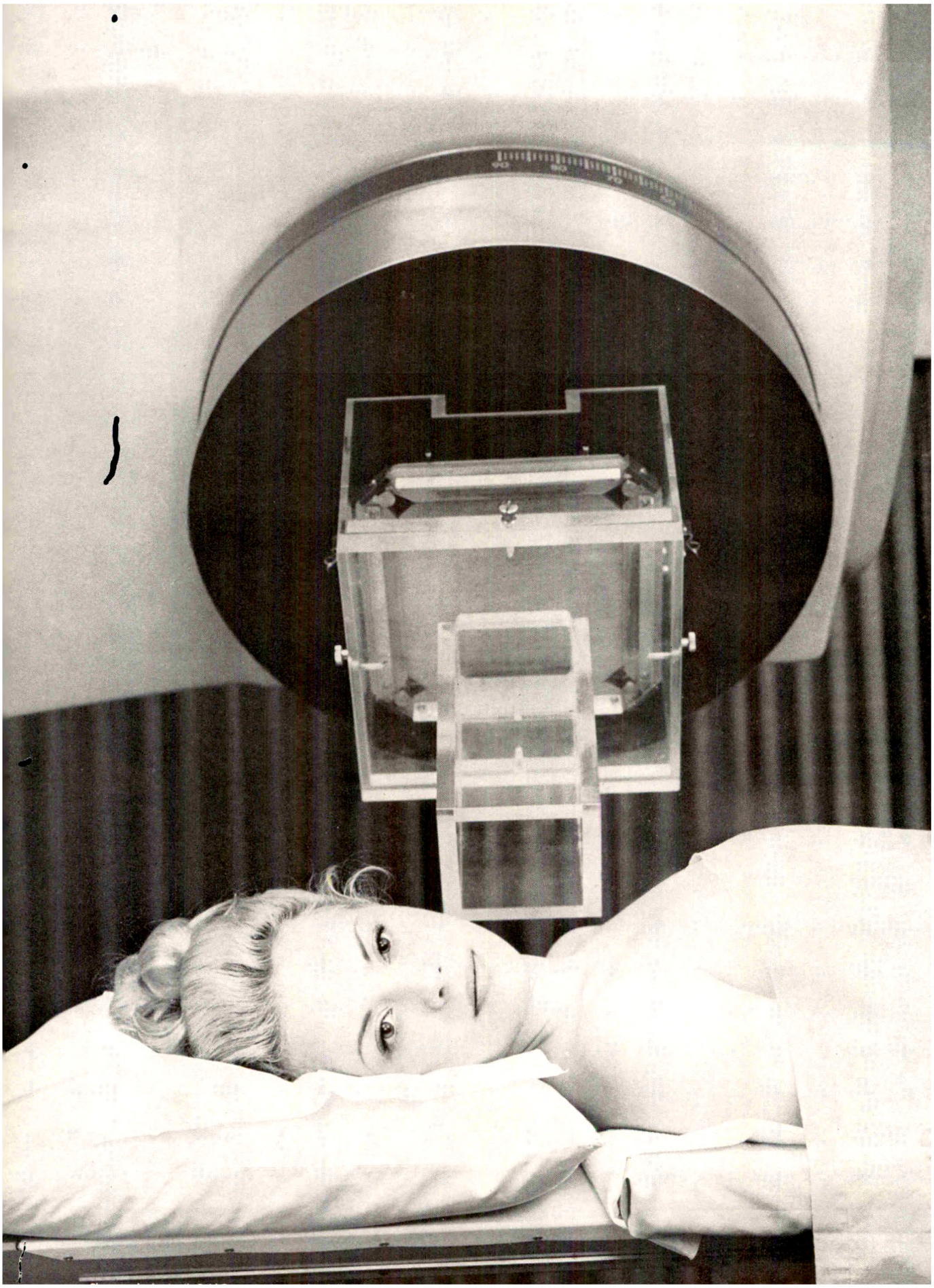
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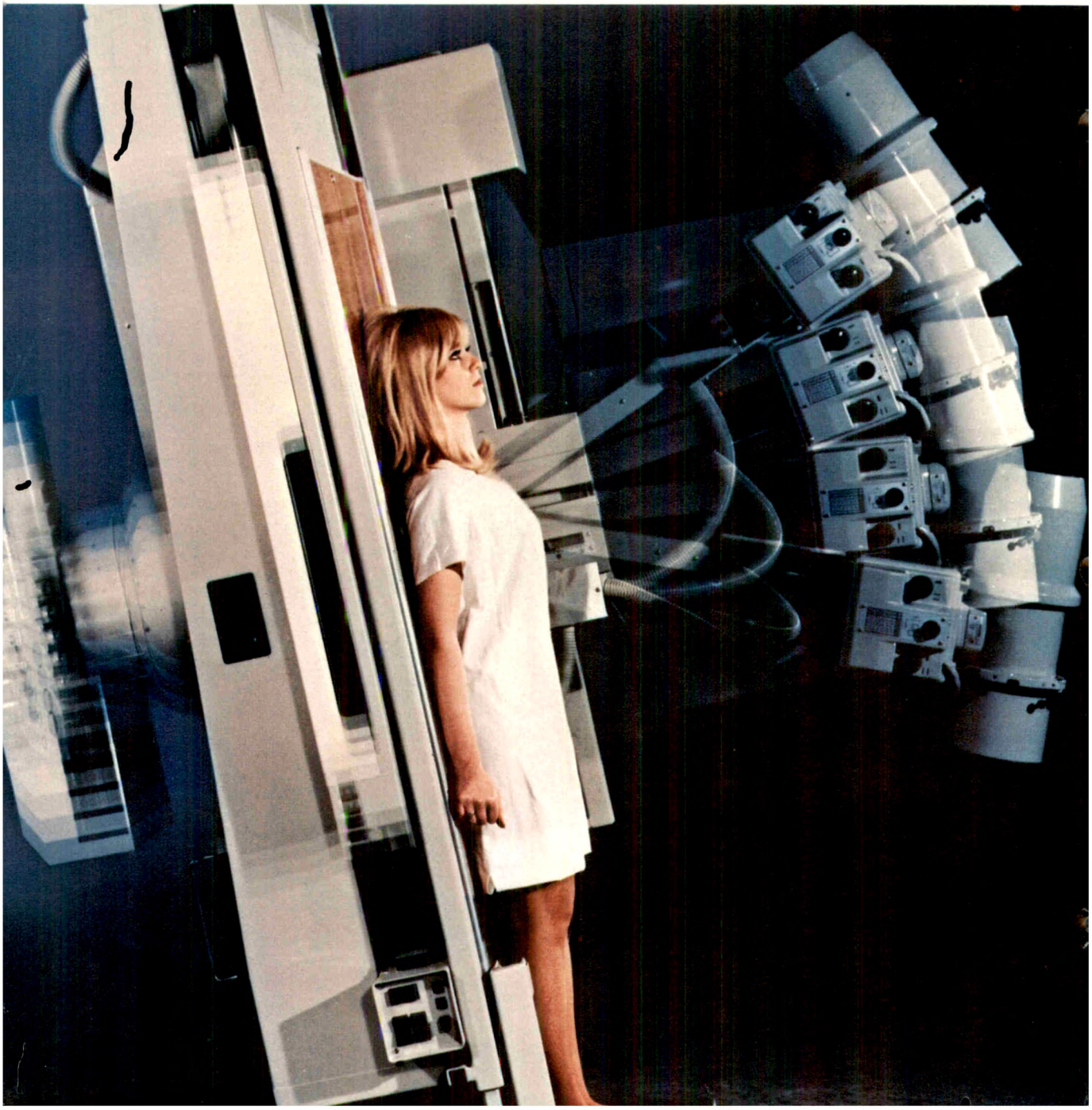
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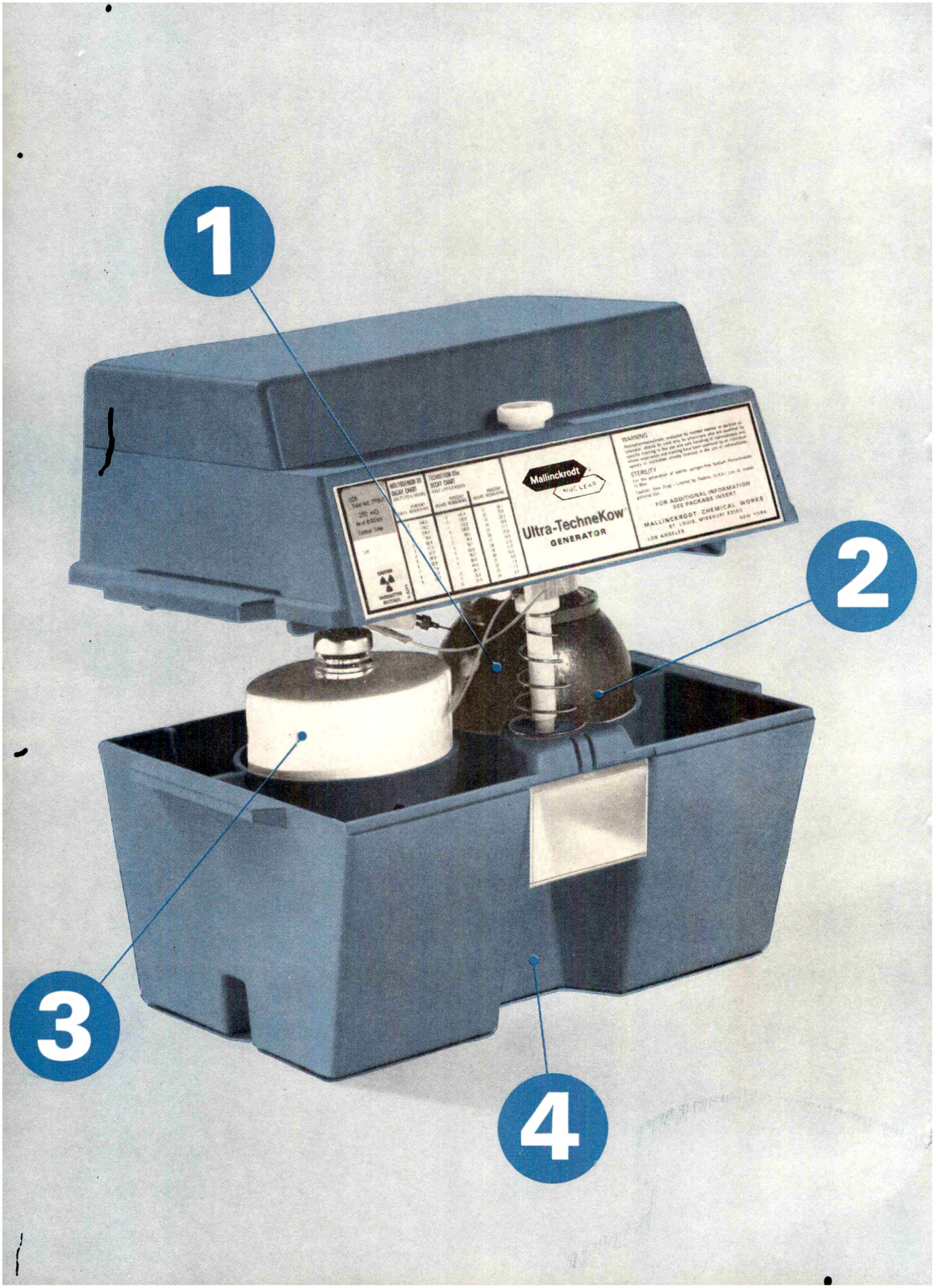
The Siregraph system permits oblique fluoroscopy and radiography at angles to ±30° along the table axis. As the table is tilted, the radiographic object remains centered between the x-ray beam and image intensifier or film carrier, keeping a fixed focus distance to the film at all times.

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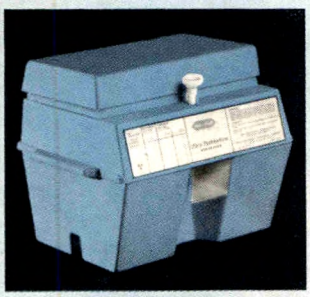
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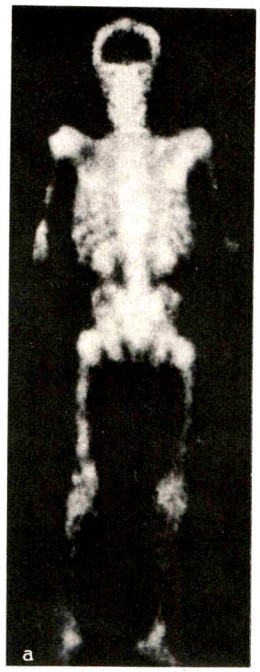
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(a) Technetium 99^{m} Polyphosphate Whole-Body Image.

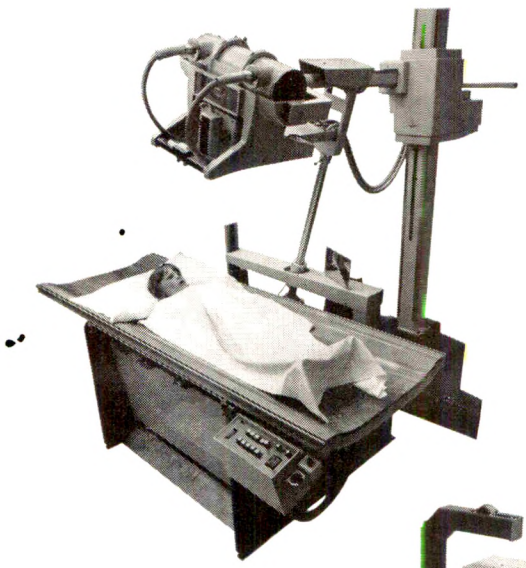
(b) Left Lateral Brain Image from Dyna Camera 2C (50% reduction).

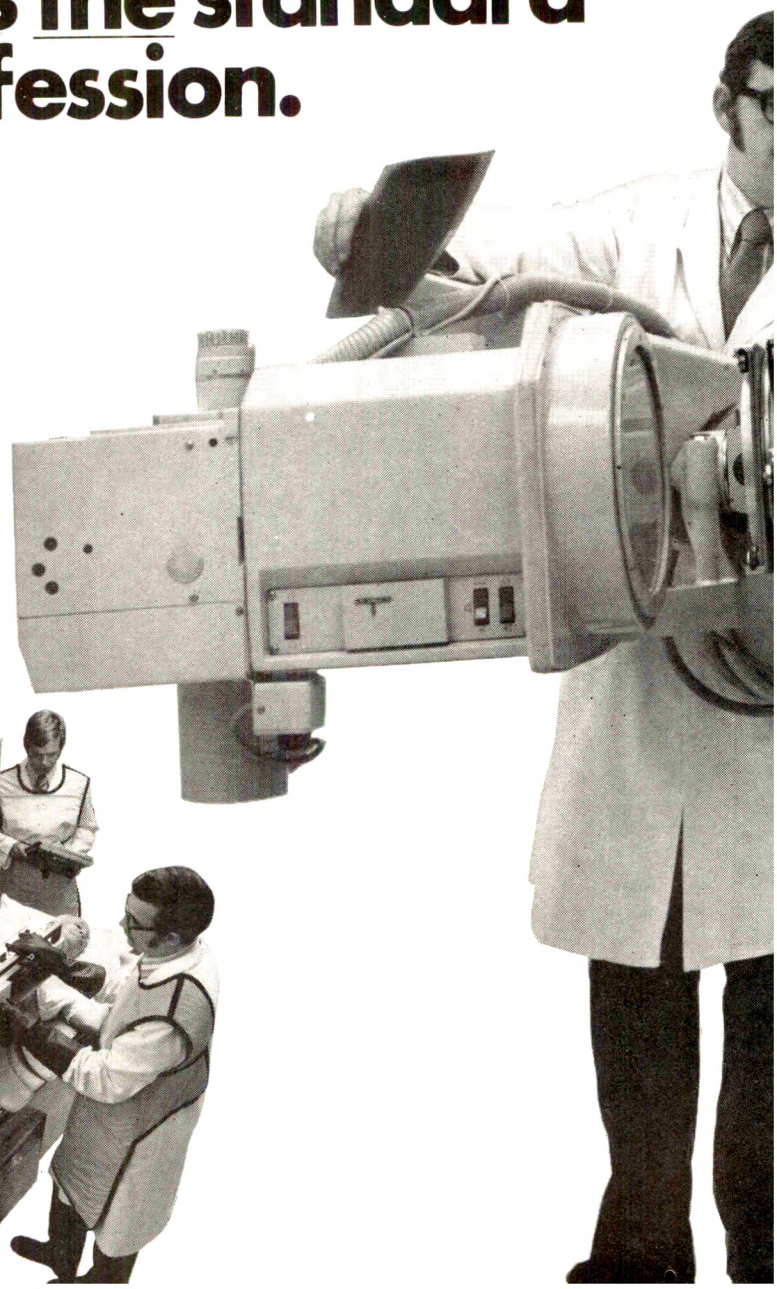
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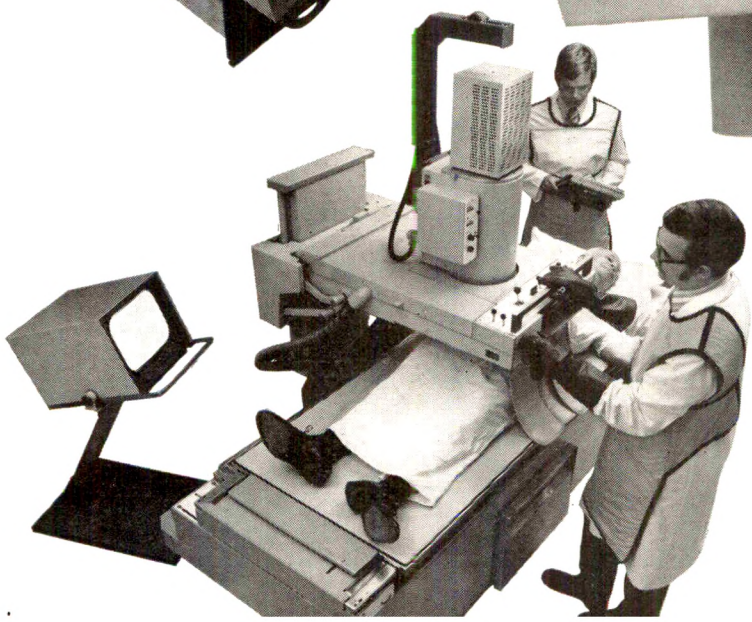
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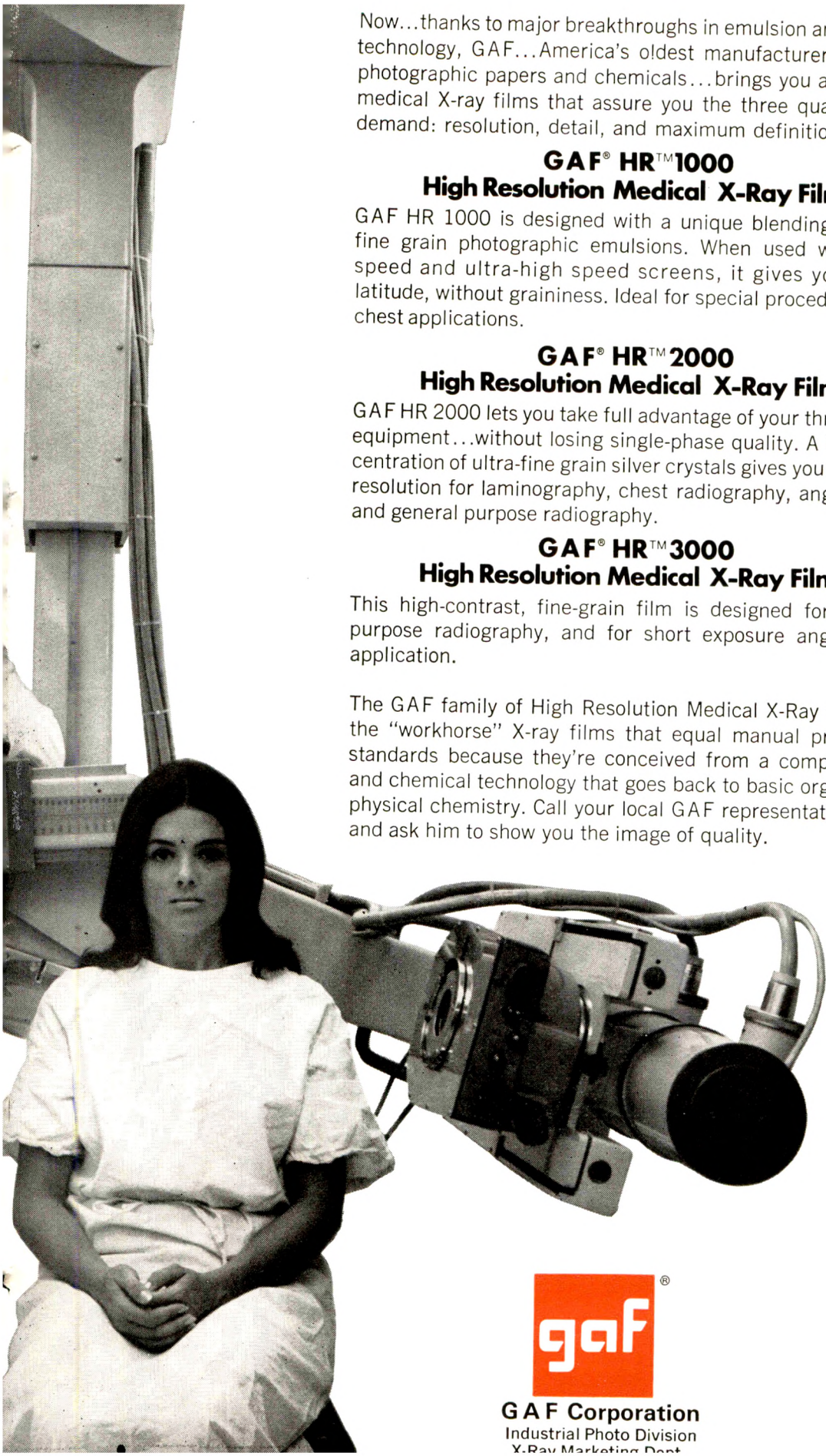
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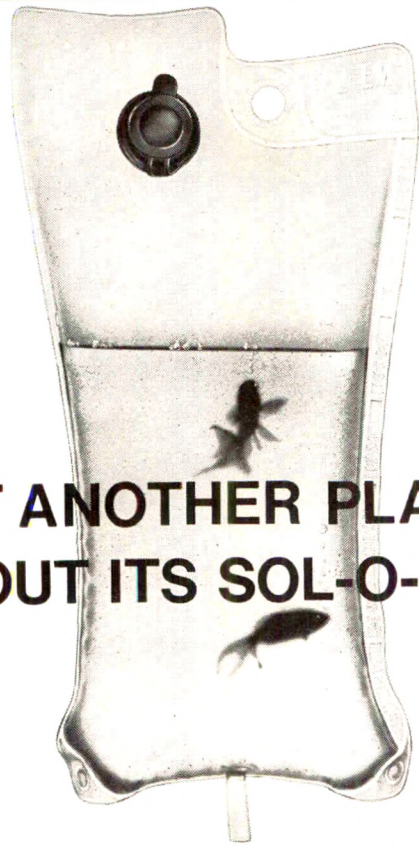
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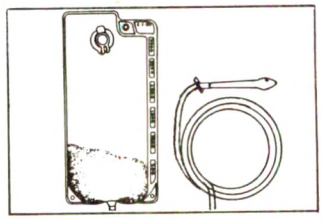
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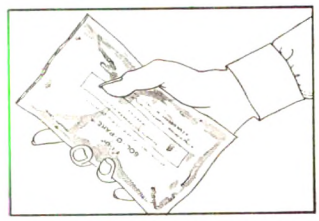
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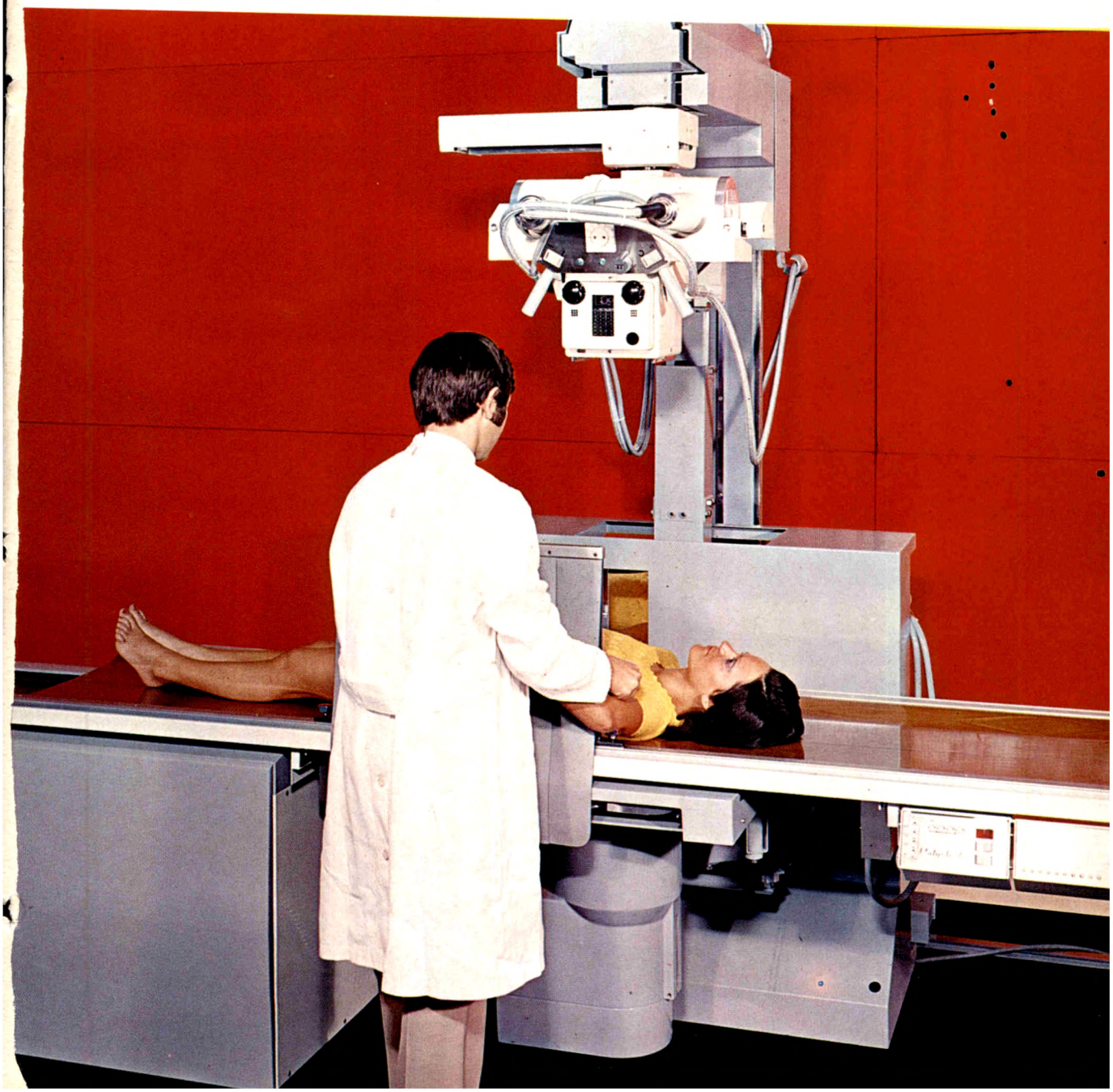
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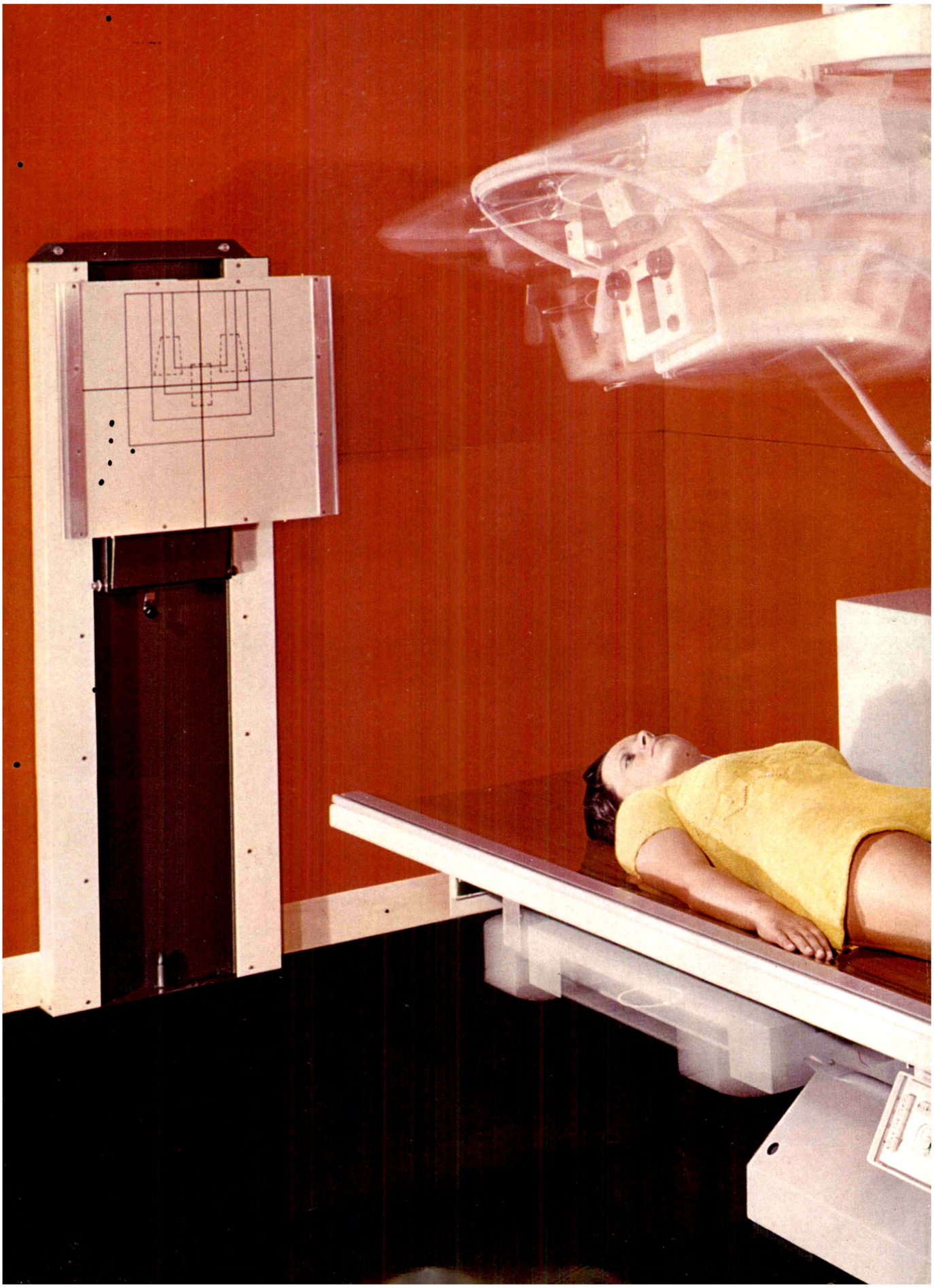
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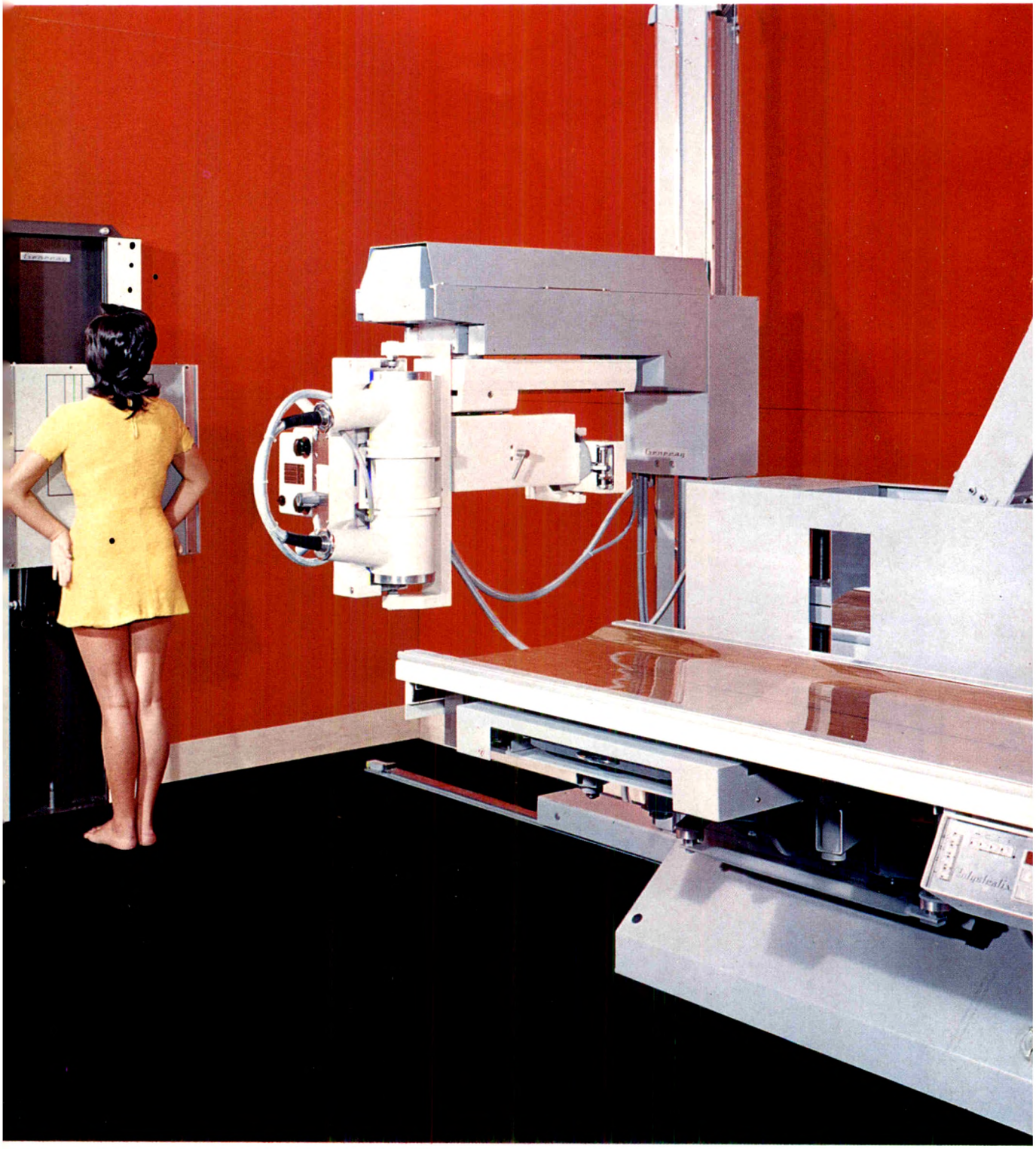




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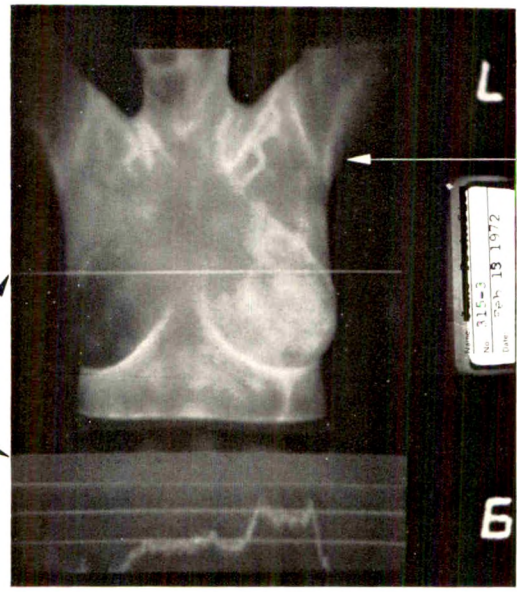
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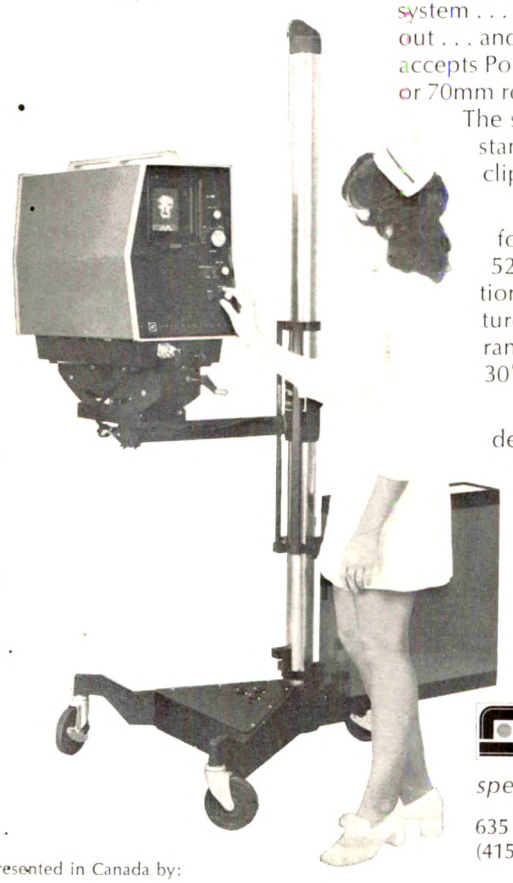
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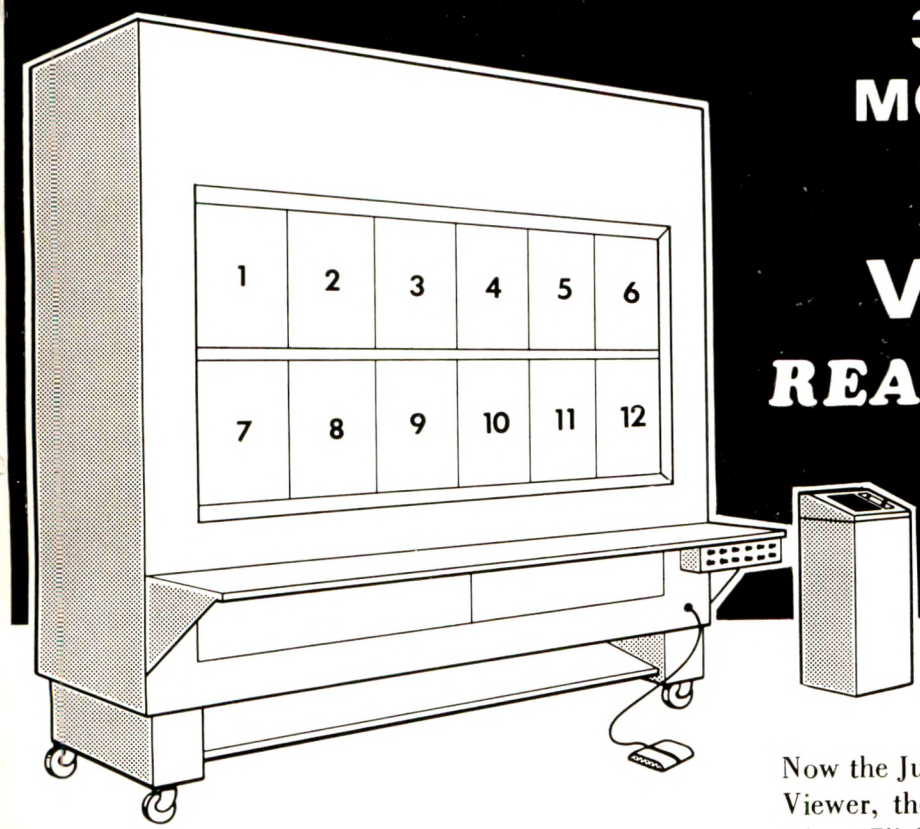
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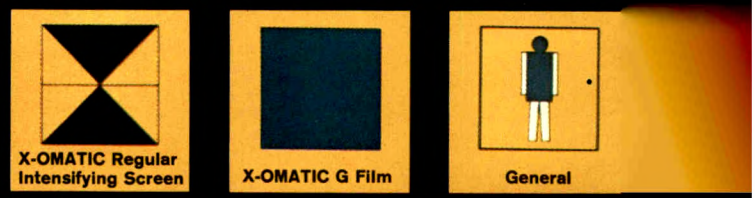
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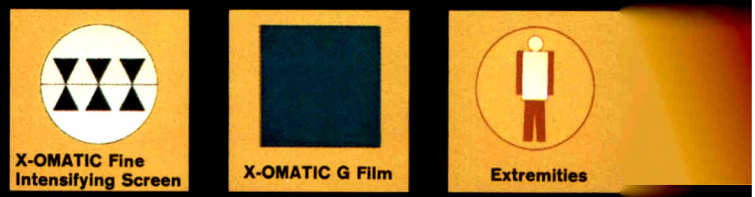
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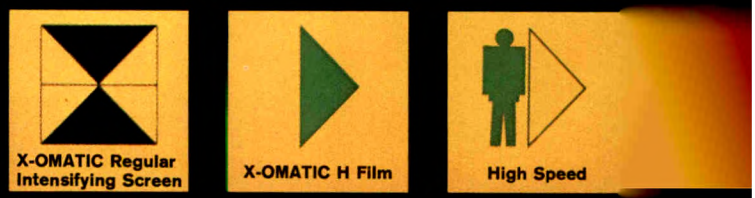
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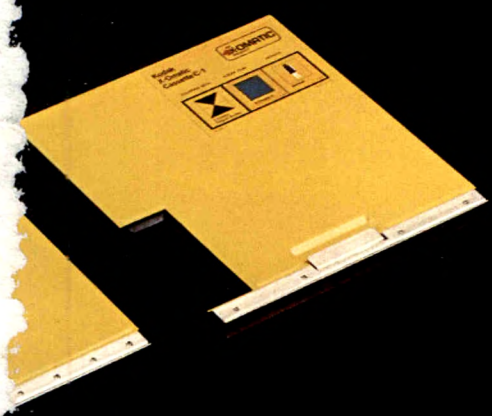
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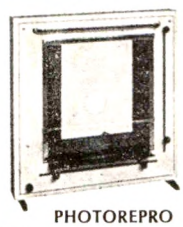


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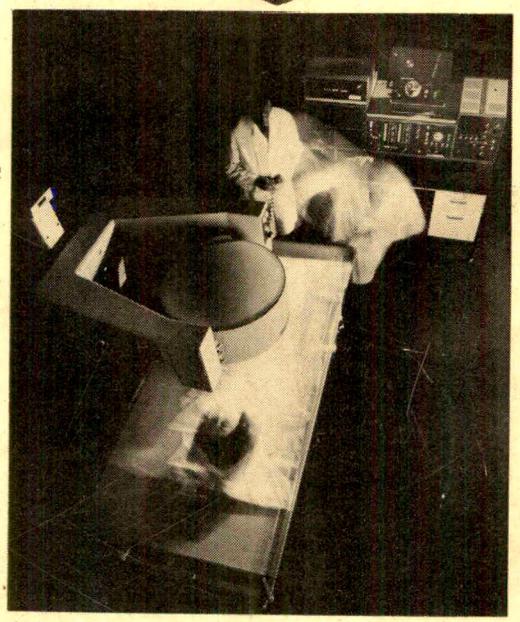
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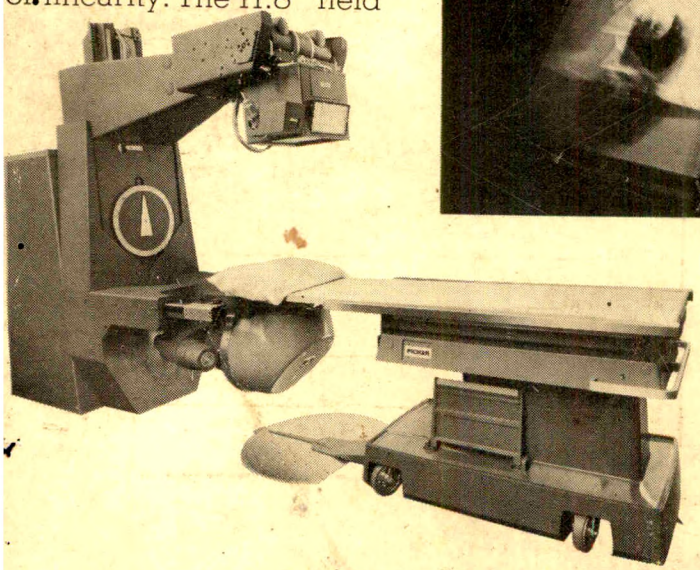
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